APPENDIX D

Transcript of American Osteopathic Association
Audio Educational Service Tape Sponsored by Abbott Laboratories

The element of management which is probably the most readily available and most effective particularly for the physician who is not able to spend the amount of time for personal catharsis and for the patient who is unwilling or unable to avail himself of this type of help is the utilization of a chemotherapeutic regimen. Let me say at the outset that you should use the least potent drug that will control the problem and, where possible, drugs that can be taken as infrequently as possible. Obviously, a once-a-day regimen would be the best for patient compliance.

We'll consider briefly three general categories of psychotherapeutic drugs which may be effective in anxiety. The first general category and the one that the majority of anxiety cases can be treated with is the group known as the minor tranquilizers. These include such medications as Tranxene, meprobamate, Valium and Libruim.

It is important to remember that even though these drugs are classified as minor tranquilizers, they are not without serious problems if abused. However, the advantage of their use is that they produce less side effects than the major tranquilizers or antipsychotic drugs and that there is less discomfort and, consequently, better compliance. And, I repeat, most anxiety problems can be adequately treated with this class of drug.

The second group that I will touch on are those defined as the major tranquilizers. The most important being the phenothiazine group such as Thorozine and Stelazine. Then there are the butyrophenones such as Haldol, and thioxanthines such as Taractan. For many years, since these drugs have been available, they have been primarily used on psychotic individuals. However, even though they may not be specifically indicated in anxiety, if a patient is resistant to the minor tranquilizers, they may be of some benefit if the patient is known not to be psychotic and is having an exceptional amount of anxiety.

If you use these drugs and do not see any results in a period of two to three weeks, then, perhaps, the extremely anxious patient should be referred to a psychiatrist. However, even though I recommend referral if you do not see results, you should not be afraid with any of the psychotherapeutic drugs to use dosages that would be effective.

Many times a general practitioner will correctly diagnose a problem of anxiety or, perhaps, a more serious psychiatric problem, but fail to give an adequate dose of medication. The guidelines on the package insert or in the Physicians' Desk Reference are wise to consult, but you should not be afraid to increase the dosage of tnese medications within reasonable limits and see how your patient responds. Many times his reaction to the varying doses of medication will be an indication of the extent of his illness and drug administration can thus be a diagnostic tool in this sense.

APPENDIX E

TRANSCRIPT OF VISTARIL TAPE

So whatever you do, you are under some stress, but that may be excessive and, in that case, some measure must be taken against it. There are lots of physiological or psychophysiological means through techniques of relaxation which we won't discuss today. We have to try to use psychological means and self-control in order to avoid anxiety, but its useless to tell a patient, "Don't be anxious." If he is the type, he is anxious and if you can't control it that way you have to help him by giving him some minor tranquilizer at least.

Now, there, I think one should select one that is not particularly dangerous and has not too many side effects. One should take amounts which are not too soporific and do not derange the normal function to an excessive degree. Especially in the evening if you can't go to sleep before because of anxiety. Another aspect of the use of tranquilizers is that you should not only give it once, when the patient is in a case of acute anxiety, but that very often you may have to prolong treatment.

For example, a patient who had a cardiac infarct will remain anxious after that too for months afterwards. In fact, if he is so disposed, even following complete recovery, he will spend the rest of his life in a state of anxiety worrying about whether he won't get another cardiac infarct. So, I think a mild condition of tranquilization, as long as it does not interfere with your normal functioning, for example, driving your car properly and so on, should in some cases, be a constant procedure.

APPENDIX F

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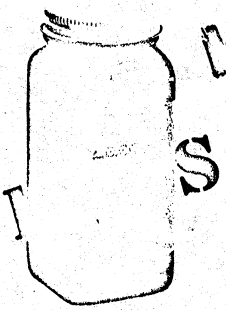
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APPENDIX G

THE SCIENTIFIC EXHIBITS

AMERICAN UROLOGICAL ASSOCIATION FILM LIBRARY S-112

DAVID A. CULP, M.D., *University of Iowa Hospitals, Iowa City.*
This exhibit displays a series of basic color, sound motion pictures designed for graduate and postgraduate medical education in the field of urology. Films are distributed in the 8mm cartridge-loaded format for use in the Fairchild Mark IV projector, in super 8 cartridges for the Fairchild Seventy 07 projector and in 16 mm as well.

ROLE OF PROSTAGLANDINS IN RHEUMATOID ARTHRITIS S-113

DAVID H. BAEDER, PH.D., *University of Missouri-Kansas City.*
A presentation of the effect of prostaglandins on the initiation of the inflammatory reaction resulting in rheumatoid arthritis, and the pharmacologic basis for drugs in the treatment of rheumatoid arthritis.

RELATIVE EFFICACY OF CEPHRADINE AND CEPHALEXIN CAPSULES IN THE TREATMENT OF URINARY TRACT INFECTIONS S-114

DAVID J. ALBERT, M.D., GERALD J. HARDNER, M.D., DATTA G. WAGLE, M.D., AND JOSEPH B. GAMBACORTA, M.D., *Buffalo, N.Y.*
A number of synthetic cephalosporins have appeared in recent years. This exhibit explores the relative efficacy of one of the newer cephalosporins, cephradine (generic name), compared with a commonly used cephalosporin, cephalexin (generic name), in the treatment of acute and chronic urinary tract infections. The clinical evaluation of each agent was derived from clinical observations correlated with bacteriological response. In addition the exhibit will contain an educational self-assessment quiz whereby the viewer can test himself on his knowledge of urinary tract infections.

TOLMETIN VERSUS ASPIRIN IN THE TREATMENT OF RHEUMATOID ARTHRITIS S-115

JEROME J. SCHNEYER, M.D., S. WONG, J. F. GARDOCKI AND T. P. PRUSS, *Southfield, Mich.*
In a six-month, double-blind study, tolmetin, a nonsteroidal anti-inflammatory agent, was significantly superior to aspirin in the treatment of 50 patients with active rheumatoid arthritis: 29 percent of the aspirin patients and 92 percent of the tolmetin patients showed marked or moderate response to medication. Mean daily dosage: 1193 mg. tolmetin; 4533 mg. aspirin.

NONURINARY FINDINGS ON THE EXCRETORY PROGRAM: A SELF-ASSESSMENT PROGRAM S-116

PAUL B. HANDEL, M.D. AND MICHAEL M. WARREN, M.D., *University of Texas Medical Branch, Galveston.*
Excretory urography is often performed for urinary tract symptoms. Careful scrutiny of the entire x-ray film, however, may reveal significant disease in other organs—in the abdomen, skeleton and pelvis—without additional films. This exhibit gives examples of some of these diseases in a self-assessment format.

TOLMETIN: LONG TERM THERAPY IN PATIENTS WITH RHEUMATOID ARTHRITIS S-117

GEORGE E. EHRLICH, M.D., *Albert E. Einstein Medical Center, Philadelphia,* THOMAS R. HOBBS, *Camp Hill, Pa.,* LEE J. CORDREY, M.D., *Tampa, Fla.,* AND DANIEL HAMATY, M.D., *New Haven, Conn.*
In a long-term (30 month) study with 430 patients, tolmetin, a nonsteroidal anti-inflammatory agent, was found to produce a significant reduction of symptomatology in patients with active rheumatoid arthritis. Daily dosage of tolmetin ranged from 200-2000 mg (mean: 1250 mg). A high degree of safety attended long-term administration.

THE SCIENTIFIC EXHIBITS

TOBRAMYCIN IN THE TREATMENT OF ACUTE URINARY TRACT INFECTIONS — S-118

LAYNE O. GENTRY, M.D. AND BRIAN B. WALKER, M.D., *Baylor College of Medicine, Houston* AND THEODORE MCNITT, M.D., *Brooke Army Medical Center, Fort Sam Houston.*

Tobramycin is an effective, safe, new aminoglycoside in the treatment of acute urinary tract infections caused by susceptible pathogens. Data displayed suggest that concurrent bacteremia is also effectively eradicated with this antibiotic. Resistant pathogens have been encountered. Re-infection commonly is related to underlying urinary tract pathology rather than ineffective antibiotic therapy.

OSTEOPOROSIS: DETECTION AND CLINICAL STUDIES — S-119

ANTHONY A. ALBANESE, PH.D., AND EVELYN H. WEIN, *Burke Rehabilitation Center, White Plains, N.Y.* AND EDWARD J. LORENZE, III, M.D., *Veterans Administration Hospital, New York City.*

Application of a quantitative radiographic technique for the detection of bone loss in some 3000 female and 1000 male "normal healthy" volunteers indicated that bone loss is an age-sex related phenomenon. The early detection of subclinical osteoporosis has been found to afford a useful criterion for study of etiological factors and efficacy of various therapeutic modalities for overcoming or minimizing bone loss. Quantitative data on the effects of a calcium supplement on the bone density of patients who have suffered hip fractures will be presented.

Confirmation of airline reservations may be made in American Airlines booth on the +20 (lobby) Level near the outbound taxi stand at McCormick Place.

DIAGNOSIS OF CHRONIC PANCREATIC DISEASE — S-120

F. WARREN NUGENT, M.D., ROBERT E. WISE, M.D., M. GARABEDIAN, M.D. AND CARL R. LARSEN, M.D., *Lahey Clinic Foundation, Boston.*

Current techniques in the diagnosis of chronic pancreatic disease will be described and compared. The diagnostic accuracy of conventional laboratory and barium examinations will be compared with hypotonic duodenography, selective angiography and pancreatograms obtained by transduodenal endoscopic cannulation of the pancreatic duct. The addition of selective angiography has improved diagnostic accuracy and, more recently, the availability of pancreatograms by noninvasive technique has added another important tool for diagnosis.

THE DIAGNOSIS AND TREATMENT OF OSTEOARTHRITIS—IBUPROFEN: A NEW TREATMENT MODALITY — S-121

GARY E. RUOFF, M.D., AND JAMES W. MELLUISH, M.D., *Kalamazoo, Mich.*

This exhibit demonstrates the clinical and radiological diagnosis and treatment modalities of osteoarthritis. Ibuprofen, a new drug for the treatment of osteoarthritis, is highlighted. The efficacy of treatment and its lack of serious side effects, including ophthalmologic problems, will be presented.

COMMUNITY-BASED COMPREHENSIVE GERIATRIC SERVICES: A MULTIDISCIPLINARY MENTAL HEALTH MODEL — S-200

CHARLES M. GAITZ, M.D. AND ROY V. VARNER, M.D., *Texas Research Institute of Mental Sciences, Houston.*

This exhibit demonstrates the practical operation of a comprehensive geriatric service providing psychological and physical care; it explains how a community-based mental health facility effectively mediates the various modalities required to meet the diverse needs of elderly persons; and it underlines the major importance of preventive and therapeutic intervention.

THE SCIENTIFIC EXHIBITS

AND THOMAS CHEN, *School of Basic Medical Sciences, University of Illinois, Urbana.*
Try your hand at managing a 35 year-old woman (simulated by a computer) who comes to you for help because of an increasing incidence of early morning occipital headaches, or a 14 year-old girl with an elevated temperature (103.5°F) accompanied by an overly-anxious mother.

PRECEPTORSHIPS IN PRIMARY CARE S-515

HAROLD MOESSNER, M.D., RICHARD CAPLAN, M.D. AND ROBERT RAKEL, M.D., *University of Iowa College of Medicine, Iowa City.*
This exhibit will demonstrate the results of 20 years experience with preceptorships in primary care at the University of Iowa College of Medicine. The repeated exposure of medical students to the primary care setting through a preceptorship program does influence the medical student in his selection of primary care as a specialty and in his selection of his practice location.

FRACTURES OF THE HAND AND FINGERS S-517

SIGURD C. SANDZÉN, JR., M.D., *Abington, Pa.*
Most hand and finger fractures and epiphyseal injuries are managed conventionally. However, surgical intervention is indicated with open fractures, displaced articular fractures, unstable fracture dislocations, unstable shaft fractures and certain ligamental avulsions. Malalignment, malrotation, angulation and infection are prevented by proper fracture reduction and position maintainence with indicated surgical intervention.

HALCINONIDE: A NEW AGENT FOR THE TREATMENT OF PSORIASIS AND OTHER STEROID-RESPONSIVE DERMATOSES S-519

PILLMORE K. BAGATELL, M.D., PH.D., EUGENE LEIBSOHN, M.D., DANIEL SHANAHAN, M.D. AND PETER CASPER, M.D., *Phoenix,* AND H. BRIAN LANDES, M.D. AND C. STUART BUCHANAN, M.D., *Winchester, Va.*
Halcinonide cream was compared with betamethasone valerate cream in double-blind paired comparison fashion in the treatment of psoriasis and other steroid-responsive dermatoses. There was a statistically significant superiority of halcinonide in the treatment of psoriasis. No differences were noted in the treatment of atopic dermatitis (including pediatric patients). Halcinonide cream was also statistically superior to its placebo cream, both in psoriasis and in atopic dermatitis.

ANAEROBIC INFECTIONS S-521

RONALD LEE NICHOLS, M.D. AND WILLIAM SCHUMER, M.D., *University of Health Sciences, Chicago Medical School, Chicago,* SHERWOOD GORBACH, M.D., JOHN BARTLETT, M.D., *UCLA School of Medicine, Los Angeles* AND LLOYD NYHERS, M.D., *University of Illinois College of Medicine, Abraham Lincoln School of Medicine, Chicago.*
This exhibit will acquaint physicians with the types of anaerobic infections they may encounter in their practice. Clinical clues and laboratory techniques used to determine the diagnosis are offered. Typical case histories of pulmonary and abdominal sepsis are included. A review of preoperative colon preparation is included as well as a treatment plan.

SALVAGE OF THE ISCHEMIC LOWER EXTREMITY IN PATIENTS WITH POOR RUNOFF S-600

RICHARD T. PURDY, M.D., AND PRAFULL BOLE, M.D., *New York Medical College, New York City.*
Twenty-three patients with threatened loss of the lower extremity in the presence of poor runoff are presented. All had a femoropopliteal bypass despite the presence of trifurcation obstruction distally. There was long-term preservation of functional extremities in 21 of 22 surviving patients.

THE SCIENTIFIC EXHIBITS

COMPARISON OF GUANETHIDINE AND METHYLDOPA IN MODERATE HYPERTENSION — S-601

NORMAN GLAZER, M.D., *Louisville.*
This exhibit describes a study which compared the efficacy and safety of guanethidine and methyldopa given in combination with a single daily dose of 50 mg hydrochlorothiazide to 33 patients with a standing diastolic blood pressure of 100 to 125 mm Hg. Blood pressure reduction after 16 weeks of treatment is compared with that obtained after two weeks of treatment with 100 mg hydrochlorothiazide. Results obtained with the two drugs are also compared. Graphic illustrations are used to depict all study results.

XERORADIOGRAPHY OF THE BREAST — S-602

RALPH B. BERGERON, M.D., *Ochsner Clinic New Orleans.*
This exhibit demonstrates the xerographic process of radiography of the breast. It shows illustrative examples of benign breast problems as well as malignant tumors. Differentation between benign and malignant conditions is stressed. Indications for the examination and clinical usefulness are listed. Accuracy of diagnosis is mentioned in the general summary.

DISCOVERY AND CLINICAL EVALUATION—A UNIQUE ANTIHYPERTENSIVE AGENT — S-603

NORMAN E. PITTS, M.D. AND MARY S. D. GHALY, M.D., *Groton, Conn.*
This exhibit features an audiovisual presentation tracing the development of a new therapeutic agent. The discussion provides insight into the basic pharmacologic hypotheses, animal testing and, finally, extensive clinical evaluation leading up to a new drug application.

INTERDISCIPLINARY EVALUATION AND MANAGEMENT OF CRANIOFACIAL DISORDERS — S-604

MICHAEL LEWIN, M.D., *Montefiore Hospital and Medical Center, Bronx, N.Y.*
The exhibit deals with congenital disorders resulting in deformities of the craniofacial skeleton. The exhibit also emphasizes the role of the extended team, which includes the interdisciplinary evaluation and treatment provided by plastic surgery, neurosurgery, anesthesiology, neurology, speech pathology, otorhinolaryngology, dentistry, psychiatry, psychology, audiology, radiology, pediatrics, internal medicine, ophthalmology, social services and others in dealing with deformities of craniofacial anatomy and physiology.

NATIONAL HIGH BLOOD PRESSURE EDUCATION PROGRAM — S-605

GRAHAM W. WARD, *National Institutes of Health, Bethesda, Md.*
The National High Blood Pressure Education Program is part of a nationwide campaign to develop an awareness in the medical profession and general public of the importance of diagnosing and controlling high blood pressure. The exhibit contains information and materials for the professional concerning the Program's recommended guidelines for screening, evaluation, drug therapy and patient education.

DOPLER ULTRASOUND IN PERIPHERAL VASCULAR DISEASE — S-606

STEVEN M. DOSICK, M.D., WILLIAM S. BLAKEMORE AND ROBERT J. NAVARRE, M.D. *Medical College of Ohio, Toledo.*

This exhibit on peripheral vascular disease discusses the principles of arterial and venous examination with Doppler ultrasound and how these apply to the evaluation of patients by primary physicians and by vascular surgeons.

APPENDIX H

138—Scientific Exhibits

tobramycin in 248 patients acutely ill with serious urinary tract infections. Approximately 50 percent had complicating features, i.e., obstructive lesions, tumors, stones, etc. Remission of signs and symptoms of infection occurred in 96 percent of all patients. The presence of an underlying disease did not unduly delay clinical improvement. Mild and transitory adverse effects occurred in 7 patients. Bacteriological response was satisfactory in 80 percent of the patients. Thirty-four (14 percent) of the patients became bacteriuric within 3 weeks following therapy. Of these, 21 were reinfected with new organisms, most commonly Pseudomonas aeruginosa.

It is hoped that the rational use of this new aminoglycoside antibiotic will delay appearance of significant numbers of strains which are resistant to it.

BOOTH 707
ROLES OF CLINICAL PHARMACOLOGY

Teaching Consultations Surveillance Research
Duncan Hutcheon, M.D., F.A.C.P., Benjamin Calesnick, M.D.,
Walter W. Baker, Ph.D., Gerald Balakin, M.D.
American College of Clinical Pharmacology
New York, New York

The exhibit consists of five panels summarizing the roles of clinical pharmacology in continuing medical education, research and patient care. The central panel consists of an audiovisual unit in which selected topics in clinical pharmacology are presented to illustrate principles of rational drug therapy. The goals, organization and drug information sources provided by the American College of Clinical Pharmacology are also displayed.

BOOTHS 713 and 714
PSEUDOMONAS INFECTION

Edward L. Quinn, M.D.
Henry Ford Hospital
Detroit, Michigan

In recent years, such pseudomonas infections have become increasingly important. Age, use of immunosuppressive agents, chronic diseases and heroin addiction have all been implicated in this change as well as use of respirators, urinary catheters and intravenous indwelling lines. Although new and unusual clinical syndromes due to Pseudomonas aeruginosa have been recognized, the course and prognosis of these infections have been greatly influenced by 1) new antibiotic agents, 2) new surgical techniques, and 3) new preventive measures.

Check for Messages at Message Center

BOOTHS 715 and 716
GASTRO-ESOPHAGEAL (G-E) SCINTISCANNING TO EVALUATE G-E REFLUX

Robert S. Fisher, M.D., Leon Malmud, M.D., Ira Lobis, M.D., Eugene Mayer, M.D., Richard Tolin, M.D., Marc Feldman, M.D., John Capuzzi
Temple University Health Sciences Center
Philadelphia, Pennsylvania

Many techniques have been employed to detect symptomatic reflux. Recently, we have adopted scintiscanning techniques to the detection and quantitation of G-E reflux. This exhibit will be under four major headings: I. Symptoms of and diagnostic tests for G-E reflux: II. Technique of G-E scintiscanning. The sensitivity of G-E scintiscanning will be compared to that of other diagnostic tests; III. Quantitation of G-E reflux. A group of normal subjects will be compared to patients with reflux; IV. Effects of therapeutic modalities used for reflux. Bethanechol, antacids, a combination of alginic acid with antacids and changing body position will be compared.

BOOTH 717
DOES MAN HAVE TUMOR ANTIBODIES?

National Naval Medical Center
Bethesda, Maryland

Radiographs will show unusual multiple small pulmonary nodules from metastatic squamous cell carcinoma of the cervix. Tumor immunology, concepts of isoimmune response, photomicrographs and the gross tissue specimens are shown.

BOOTH 718
DISCOVERY AND CLINICAL EVALUATION

Norman E. Pitts, M.D., Allen P. Borger, M.D.
Dept. of Clinical Research—Central Research Div.
Groton, Connecticut

This exhibit gives an overview of the process involved in the development of a new drug in the United States. It features an audiovisual presentation of the sequential discovery and development of a new antihypertensive agent, prazosin HCl. The research objective was to develop an agent which exerted its antihypertensive efficacy by arteriolar vasodilation. The search was conducted in a novel chemical series, the quinazolines. Significant steps will be highlighted from discovery through clinical evaluation including biochemical/pharmacological goal of the research project, animal pharmacological profiling of the new agent, and the subsequent data which emerged from the NDA clinical program.

Visit the Exhibit

Charges of overprescribing are all the rage. But what about the equally dangerous tendency to underprescribe?

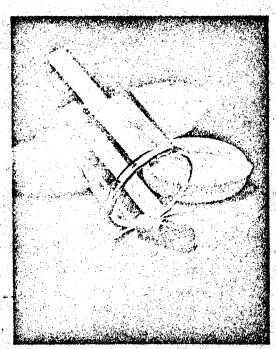
The undermedicated society

Richard L. Peck
Senior editor

Who is the "irrational prescriber"? It's usually said that he's the physician who prescribes too much—whether it's oral hypoglycemics, minor tranquilizers, or antibiotics; whether for the wrong indication, with no indication, or in place of a safer, equally effective drug. The family physician's alleged propensity to overprescribe has provoked well-publicized investigations, both Congressional and clinical.

But what of the other side of the problem—underprescribing? Do doctors sometimes prescribe too little? In interviews with experts in several specialties, CURRENT PRESCRIBING found that the answer is yes. For many conditions, and with a number of drugs, MDs may be giving their patients therapeutic short change.

Take pain, for example. Says Dr. John Bonica, professor and chairman



of anesthesiology at the University of Washington and an internationally known authority on pain treatment: "Many physicians undermedicate with narcotics, especially in patients with chronic pain due to cancer and in those with acute postoperative pain.

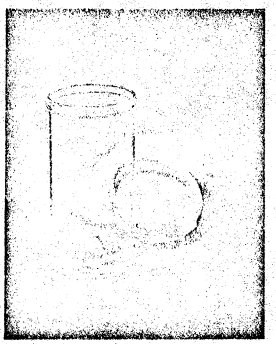
"This probably happens because our medical schools don't adequately teach the use of narcotics for clinical pain, either acute or chronic. For instance, they don't teach IV administration of narcotics, and physicians are afraid to use this route. But with severe pain from a kidney stone, a gallstone, or serious lacerations, giving a narcotic any other way is an error. A subcutaneous or intramuscular dose is absorbed too slowly to reach a sufficiently high peak level.

"It's true that serious reactions may occur with an IV dose—severe nausea, hypotension, hypertension, impaired cerebral function—but these are due to improper technique, usually too rapid injection. It's worth re-emphasizing that in severe acute pain, this is the best and most effective route for the initial dose."

Another mistake physicians tend to make, says Dr. Bonica, is prescribing an analgesic on demand for chronic pain. "We've been taught to do it this way for years, but it actually fosters chronic pain behavior. In effect,

The undermedicated society

one rewards the patient for his complaints of pain. As for narcotics, they should be avoided in patients with chronic pain—except those with malignant disease. In such cases the narcotic ought to be given at fixed



intervals and in sufficient doses to provide a steady level of *adequate* analgesia."

Physicians are often overcautious in treating some childhood disorders, says Dr. Sydney Gellis, chairman of pediatrics at New England Medical Center and author of a widely used pediatric textbook.

"Take salicylates for rheumatoid arthritis," he says. "Doctors are afraid of salicylate toxicity, but treatment may require doses as high as 1 gr/lb of body weight daily to be effective. For assurance, blood levels should be taken, aiming at a serum level between 25 and 30 mg/100 ml.

"Meningitis is another example. Antibiotic doses ought to be calculated according to the child's body weight, but often aren't. Instead, suboptimal doses are given."

Another frequently underused drug in children, says Dr. Gellis, is prednisone (Delta-Dome, Deltasone, Meticorten, Orasone, Sterapred, et al). "The full dose is 2 mg/kg daily, and it should be given for severe asthmatic attacks or severe cases of poison ivy or hives. But we often see family practitioners using doses of 0.25 mg or 0.5 mg/kg for these conditions, and that's not adequate. If the condition is severe enough to justify corticosteroids, they should be given adequately. Otherwise, they shouldn't be given at all.

"Phenobarbital (Eskabarb, Hypnette, Luminal, Solfoton) is underdosed, too. Perhaps an eighth of a grain is used for an infant instead of the more appropriate quarter grain. Rather than quiet the child down, this only makes him more excitable."

Finally, notes Dr. Gellis, there's the much discussed problem of patient noncompliance with antibiotic regimens. "Physicians tend to blame the parents if a child doesn't complete the full 10 days of treatment in strep throat or otitis media, for example. But I suspect it's sometimes the doctor's fault for insufficiently impressing parents with the importance of compliance. He should do so not only verbally, but in written instructions. And he should make a point of telling parents that there's a way to get around the problem if their child refuses to take the oral preparation: They must bring him in for an injection of long-acting antibiotic."

Another physician who finds family practitioners sometimes erring on the side of caution is Harry M. Robinson Jr., chief of dermatology at the University of Maryland Hospital.

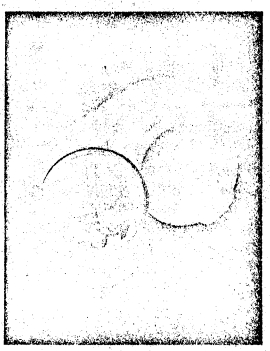
"Many times we've seen cases of one of the reactive erythemas, such as erythema multiforme, in which we've really had to slug the patient with a huge dose of prednisone to overcome the basic problem. Their doctors had been using 10 to 15 mg a day, and we've had to triple or quadruple the dose—in fact, we've used as much as 100 to 150 mg a day.

"The same thing happens with severe cases of bullous pemphigoid, pemphigus foliaceus, pemphigus vulgaris, and systemic lupus erythmatosus. We've had patients come in completely denuded from some of these disorders, because they didn't receive a sufficiently high dosage of prednisone.

"Let's face it," continues Dr. Robinson, "when it comes to cases like these, some of the FDA-approved dosage recommendations are almost homeopathic. You can't send a boy to do a man's work."

Depression

No help at all is what too many physicians give for depression, says psychiatrist Nathan S. Kline. In fact, Dr. Kline went so far as to call depression "the most undertreated of all major diseases" in a 1974 JAMA article (227:1158, 1974). "The percentage of nonpsychiatrists attempting to treat depression is discouragingly small," Dr. Kline wrote, "and frequently



treatment is not done well." An example he cited was underdosing—using less than 25 mg of a tricyclic tid or qid [or less than 5 mg of nortriptyline HCl (Aventyl) tid or qid], or using whatever dose of a tricyclic for

less than three weeks.

Not everyone agrees completely with Dr. Kline. "I, for one, am not convinced that family physicians are equipped to treat depression as readily as Dr. Kline says they can," remarks psychiatrist Daniel X. Freedman of the Pritzker School of Medicine, University of Chicago.

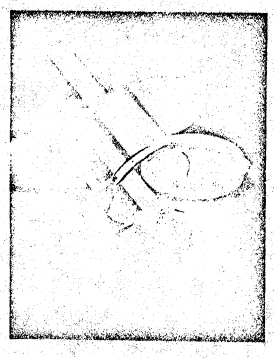
"It's a complicated matter, and most nonpsychiatrists need more training and readily available psychiatric consultation to cope with these cases." But Dr. Freedman agrees that antidepressants are sometimes misused by family physicians.

"Hospital studies have shown that physicians tend not to monitor treatment closely enough, and that they don't tailor a drug regimen to the patient's needs. Drug therapy seems to be aimed at the average patient, rather than the individual. The drugs are usually used when they're indicated, these studies show, but they're not used with enough careful thought. And some patients are undermedicated."

Hypertension, of course, is the classic undermedicated condition, but physicians seem to be improving their performance with it, according to Dr. Richard S. Ross, president of the American Heart Association.

He thinks physicians increasingly are treating mild and moderate cases, rather than waiting until the diastolic hits 115. Treatment ought to start at diastolic 105, according to the National Heart and Lung Institute, but Dr. Ross says that he and other physicians regularly initiate treatment at 90 to 100 for some patients, depending on such factors as age, family history, target organ damage, and cholesterol level.

"In the last five years we've learned that there's no such thing as 'benign' essential hypertension," says Dr. Ross. "We know that treatment of mild cases can reduce the incidence of stroke and cardiovascular-related mortality in later life."



It's possible, though, says Dr. Ross, that physicians using information eight or ten years old are still treating only severe cases. It may also be that some physicians are discouraged from taking on the extra burden because treatment, as Dr. Ross puts it, "is a nuisance. You've got to do a lot of talking to convince a patient to take a drug that may make him feel worse to prevent something hypothetical from happening. That's a difficult idea for some people to grasp."

Of course, much hypertension goes untreated simply because people don't know they have it. But of the known hypertensives, according to the National Heart and Lung Institute, only one-fourth are getting adequate treatment. Doctors may be changing that, as Dr. Ross says, but clearly there is plenty of room for improvement in this area.

Antibiotics

Even with that class of drugs that physicians are accused so often of *over*prescribing—antibiotics—there are occasions when they undermedicate. "It's rare," says Dr. James J. Rahal Jr., chief of the infectious disease division at the New York (Manhattan) VA Hospital, "but it does happen—particularly with Group A streptococcal infections. Everyone knows that streptococcal pharyngitis will usually respond to 125 to 250 mg of penicillin four times a day. So many physicians, when they see a strep infection like cellulitis, for example, think it will respond to the same small amount of penicillin. When it doesn't, they ask, 'What does this patient have? It can't be strep.' I used to do the same thing myself until I got burned a few times.

"Now, with really severe cases of streptococcus cellulitis, I sometimes have to give penicillin intravenously or intramuscularly, perhaps a half million to a million units every four hours, and sometimes more," Dr. Rahal observes.

These experts agree, then, that our supposedly overmedicated society is sometimes undermedicated. They're also in agreement about the remedy: Be confident of your diagnoses, know what the available medications can do (and package inserts aren't *always* a reliable guide), and tailor each prescription to the individual patient's needs. In so doing, you'll avoid the ranks of those irrational prescribers who are not cavalier, but too cautious. □

Undermedicated society: Additional prescribing information

For more information on therapeutic agents discussed in this article, see Physicians' Desk Reference, 1975 edition.

	PDR page
Nortriptyline	
Aventyl (Lilly)	912
Phenobarbital	
Eskabarb (Smith Kline & French)	1383
Luminal (Winthrop)	1604
Solfoton (Poythress)	1193
Prednisone	
Delta-Dome (Dome)	741
Deltasone (Upjohn)	1515
Meticorten (Schering)	1331
Orasone (Rowell)	1291

Serotonin inhibitors, antihistamines, antidepressants, beta blockers —they're all in the new wave of 'headache remedies.'

text&test

Migraine...and more

Treatment and prevention

Headache sufferers are a desperate breed. Worn and confounded by their pain, they will ask for "something, anything" to relieve it. Happily, these days there are more remedies to offer them. Even for migraine, where the venerable ergot derivatives have stood almost alone, there are new possibilities.

CUTLER: The most important thing is to identify the source of the headache. For some reason, both patients and physicians tend to think first of organic disease when they're looking for the cause of recurring headaches. As the sinuses, teeth, and eyes are commonly implicated, many patients wander in vain from one specialist to another seeking a cure. Headaches with an organic basis generally have easily identifiable signs and symptoms, and physicians can usually rule out hypertension, brain tumor, aneurysms, and meningitis by clinical observation, plus tests and x-ray films, when indicated.

In fact, the vast majority of headaches are functional and not associated with organic brain disease. (The use of the term "functional," however, doesn't negate the possibility of some neurochemical basis.) These headaches fall into four categories: migraine, cluster, tension, and toxic vascular. Dr. Fuller, would you agree?

FULLER: I don't think we should try to make a hard and fast clinical distinction between patients with organic headaches and those with functional ones. Most patients are affected both by somatic factors—tension in the muscles, vascular problems—and emotional factors. But regardless of the degree to which the psychological factors outweigh the somatic, all headache patients suffer real discomfort; their problems are not imaginary.

Migraine

CUTLER: Let's take the case of a 42-year-old woman who's had recurrent, severe, throbbing headaches for many years. They occur on one side of her head or the other, most often in times of stress. They're associated with nausea and vomiting, and preceded by flashing lights and diminished vision on the side opposite the headache. They last for 6 to 12 hours. As a child, she was often carsick. And her mother used to have similar headaches. What's the diagnosis, and what therapy would you prescribe.

ALDREDGE: That's a classic history of migraine vascular headache ("Migraine's three phases," p 49). For

Round table at the University of Texas Health Science Center at San Antonio

Paul Cutler, MD, moderator
Associate dean for continuing medical education, professor and head, division of general internal medicine, department of medicine

Horatio R. Aldredge, MD
Clinical assistant professor of neurology, division of neuroscience, department of medicine

David S. Fuller, MD
Associate professor of psychiatry, department of psychiatry

Everard J. Siller, MD
Assistant professor of neurology, division of neuroscience, department of medicine

Migraine...and more

acute treatment of vascular headaches, the most valuable drugs are generally the vasoconstrictors. They're most helpful early in the vasoconstrictive phase during the aura, but less helpful in the vasodilatation phase, when the full-blown headache has set in.

The most useful vasoconstrictor is ergotamine tartrate, which is available in parenteral form and oral tablets (Gynergen), in sublingual tablets (Ergomar), and in aerosol form (Medihaler-Ergotamine). The intramuscular or subcutaneous dose is 0.5 to 1 cc at onset of attack, repeated in 40 minutes, if necessary; maximum weekly dose is 2 cc. The average oral dose is two to six 1-mg tablets per attack, while the sublingual schedule is one 2-mg tablet at the first sign of attack or as soon as possible after full onset, then one tablet at half-hour intervals, if necessary, not to exceed three tablets in 24 hours nor 10 mg a week. The injectable form is used most often and seems preferable to me—oral therapy may be ineffective because of the nausea and vomiting that generally accompany the headache.

When combined with caffeine, ergotamine is marketed as Cafergot, in oral tablet and suppository forms. Dosage for oral Cafergot is two tablets at onset of the syndrome, followed by one tablet every half hour thereafter up to a total of six per attack. No more than 10 tablets should be taken weekly. If oral medication is impractical, the patient can take one Cafergot suppository (2 mg ergotamine, 100 mg caffeine) at the start of the attack and a second an hour later, if needed. Maximum is four to five per week.

Still another ergotamine preparation is Migral. Migral tablets contain 1 mg ergotamine, 25 mg cyclizine HCl, and 50 mg caffeine. Cyclizine is an antihistamine of the piperazine class and is noted for its antinauseant and antiemetic effects. It's most effective when given in the prodromal phase; the usual dose is one tablet.

For patients who have some intolerance for ergotamine, there's a closely related preparation, dihydroergotamine mesylate (D.H.E. 45), which is usually given IM in doses up to 2 or 3 cc per attack.

Another useful agent for treating acute headache when ergotamine is inadvisable is the synthetic antispasmodic isometheptene.* Single-drug products include Octin, available as isometheptene mucate oral tablets and isometheptene HCl for IM administration. Dosage is one tablet three or four times a day; or 1 ml (100 mg) IM, repeated once in four to six hours. Midrin, a product containing isometheptene mucate, dichloralphenazone, and acetaminophen, is supplied in oral capsules; two capsules are taken at the onset of an attack, followed by one every hour up to an additional three capsules, with a weekly maximum of 20 capsules.†

In frequent, severe migraine, prophylactic treatment has to be considered. The patient's tolerance for the attacks should guide you in deciding on preventive therapy. The most valuable preparation is methysergide maleate (Sansert); dosage is one 2-mg tablet with each meal, up to four daily. It may require two or three weeks for methysergide to take effect. It works by blocking the effect of serotonin, a substance which may be involved in the mechanism of vascular headache. Cyproheptadine HCl (Periactin), an antihistamine with mild to moderate antiserotonin activity, has also been used prophylactically, but may be only slightly more effective than a placebo.‡

In addition, Bellergal, a combination of ergotamine, belladonna alkaloids, and phenobarbital sodium, has been used for prophylactic administration, but I've not found it very effective for long-term use (preparations containing ergotamine are probably ill-advised for preventive or long-term use). To be fair, I must mention that many physicians have used it for years in the prevention of both migraine and cluster headaches. Pizotyline, which is closely related to cyproheptadine and is also known as pizotifen or BC-105, is a relatively new preparation sold as Sandomigran. It has had widespread trials in other countries. Propranolol HCl (Inderal) has been reported effective pro-

Learning objectives

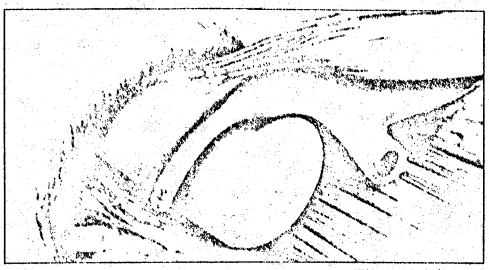
- How to recognize the four basic types of headache
- Causes of each headache type
- How to treat and prevent migraine headache
- How to treat cluster headaches
- Use and limitations of psychotherapeutic aids
- Role of food, drugs, and mood

*Use for this indication has not been approved by the FDA.

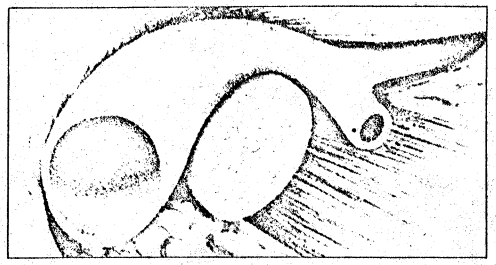
†The manufacturer's suggested dosage limit is five capsules within a 12-hour period.

‡Use for this medication has not been approved by the FDA.

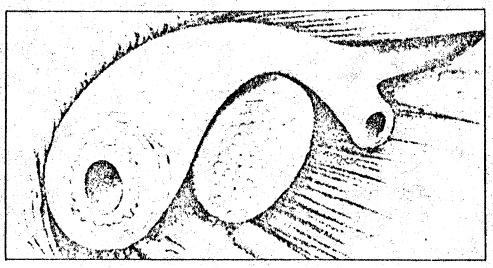
Migraine's three phases



The initial phase is characterized by constriction of the ophthalmic artery, branching from the internal carotid artery and transversing the optic nerve. Resulting ischemia causes central scotoma.



In the second phase, the same artery is dilatated, and the patient experiences a throbbing, pounding headache.



The third phase begins when edema of the opthalmic artery results in a pounding headache with duller, steadier, more constant pain that's more diffuse in distribution. This phase may not always occur.

phylactically in oral doses of 20 mg four times a day.§ In my experience, however, it has been effective in only a small percentage of cases.

Since depression may cause frequent, severe migraine headaches, prophylactic antidepressant therapy should also be considered to treat the underlying depression. We use the tricyclics, such as imipramine HCl (Imavate, Janimine, Presamine, SK-Pramine, Tofranil) and amitriptyline HCl (Elavil, Endep) in preference to the monoamine oxidase inhibitors (MAOIs).

CUTLER: Dr. Siller, you've had experience with propranolol in migraine. Does it work well?

SILLER: It's too early to tell whether it's effective. I'd use it only if standard treatments don't bring improvement.

CUTLER: Dr. Aldredge, you said that when a patient can't tolerate ergotamine, you try one of the other agents. What do you mean by "can't tolerate?"

ALDREDGE: Ergotamine is a potent, long-acting vasoconstrictor. Many patients taking it experience not only gastric side effects, such as nausea and vomiting, but also paresthesias, muscle pain, or precordial pain, along with alterations of pulse and blood pressure. These are immediate contraindications to continued use. Another is pregnancy, because the drug's a uterine stimulant. (Isometheptene, however, can be used safely in pregnancy.)

Side effects have also been reported with methysergide. It's been associated with retroperitoneal, pleuropulmonary, and heart-valve fibrosis. In several clinics with large series, it's been found safe when given up to six months, followed by periods of four to six weeks off the medication. Contraindications include peripheral vas-

§Use for this indication has not been approved by the FDA.

Migraine...and more

cular disease, coronary heart disease, severe hypertension, and liver and renal disease.

CUTLER: Some patients insist on morphine sulfate or meperidine HCl (Demerol) for prompt relief, claiming that nothing else helps. They want to be "knocked out" and do seem to get good results. Why not use these agents?

ALDREDGE: Occasional use of such agents for acute headache is acceptable. But I prefer vasoconstrictors, especially if attacks occur monthly or more often, and there's no contraindication to their use.

CUTLER: What kinds of people get migraine headaches?

FULLER: Compulsive, perfectionistic, ambitious, intelligent people who have a lot of drive. They often have attacks as they let up after achieving one of their goals. Onset commonly occurs on weekends or holidays—early or late in the day—when patients are relaxing from their chronic tense state.

CUTLER: How would you treat a patient who gets migraine headaches for psychological reasons?

FULLER: It's usually not feasible to try to change the personality of compulsive, hard-driving, tense individuals. It is helpful, however, to respect such patients for their usually superior intelligence and to fill them in on what's going on. Explaining things often pays off with the obsessive-compulsive.

CUTLER: Foods cause migraine, too, don't they?

SILLER: There's evidence to support this premise. Most patients for whom it's true tell you spontaneously that they have to avoid certain foods. If they don't, run through the list of foods on page 51 with them and see if they can connect any with their headaches. Then restrict the implicated foods.

Many of the foods listed contain tyramine, which is dangerous not only to patients prone to migraine but also to those taking MAOIs. Migraine patients are also very sensitive to the flavor enhancer monosodium glutamate; some may even be prostrated after eating a Chinese dinner that's been "enhanced." Another rule is to avoid cured meats and hot dogs. Sodium nitrite, a food preservative, is the offender and causes "hot dog headache."

ALDREDGE: Going through this list is more diagnostic than therapeutic. Very few headaches are clearly linked to specific substances. If patients can associate one or two foods with their headaches, this strengthens a diagnosis of vascular headache.

SILLER: One other point concerning food: Patients who tend to become hypoglycemic should eat three well-balanced meals daily, avoiding large quantities of carbohydrates. Low sugar threatens the integrity of brain neurons and may cause dilatation of the intracranial vasculature.

CUTLER: Do oral contraceptives play a role in vascular headache?

ALDREDGE: Probably. If your patient develops headaches when she's on the Pill, or if her premenstrual headaches

Migraine drugs: Your options

- Vasoconstrictors

Ergotamine preparations

Isometheptene preparations*

- Drugs that simulate action of serotonin on receptor sites, acting as competitive serotonin inhibitors

Cyproheptadine HCl (Periactin)*

Methysergide maleate (Sansert)

Pizotylene or pizotifen (Sandomigran)*

- Beta-adrenergic blocking agents, which prevent vasodilatation by acting on blood-vessel receptors

Propranolol HCl (Inderal)*

- Drugs preventing depletion of vasoactive amines (catecholamines and histamine) by interfering with the action of their deactivating enzymes:†

Monoamine oxidase inhibitors

- Isocarboxazid (Marplan)
- Phenelzine sulfate (Nardil)
- Tranylcypromine sulfate (Parnate)

Tricyclic antidepressants

- Imipramine HCl (Imavate, Janimine, Presamine, SK-Pramine, Tofranil)
- Amitriptyline HCl (Elavil, Endep)

*The FDA has not approved use in migraine.
†These drugs are used to treat the underlying depression of migraine, not the migraine itself.

worsen, the drug should be stopped. She may be one of the patients in whom OCs allegedly cause stroke.

Cluster headache

CUTLER: A 52-year-old man complains of a severe, unilateral, burning headache. It lasts about an hour, occurs frequently for a few weeks, and goes away for a year or more. It's associated with marked tearing and considerable pain in one eye, and runny nose on the same side. There are no preceding neurologic or eye symptoms. What does he have, and what do you do about it?

SILLER: This is a cluster headache. Typically, an adult man is awakened every night for two or three weeks by the most severe headaches he's ever experienced. Some patients are jolted out of bed before they fully wake up. Then the headaches go away for perhaps a year or a year and a half.

Cluster headache is a type of migraine; it's also called Horton's headache, after the physician who described it, or histamine cephalalgia, after the substance thought to cause it. Many physicians still carry out histamine desensitization with patients suffering from this syndrome, though it's probably not indicated. Remember, though, that the man with a cluster headache is probably sensitive to many vasodilative substances. Histamine desensitization may remove at least one from the list but you can't desensitize him to all vasodilators.

The treatment of choice is ergotamine, the same as for ordinary migraine headache. This drug is particularly suitable because a patient is usually awakened at nearly the same hour each night. Tell the patient to set his alarm clock so he awakes an hour before he customarily is aroused by the headache, and to take two 1-mg tablets. He should do this for several consecutive nights. This regimen usually averts the cycle and wins you a friend for life.

CUTLER: Building up the dosage and tolerance of histamine has been the treatment of choice for many physicians, and I've used it until today. Now you're telling me that this course really doesn't do much good and that we should treat cluster headache the same way we treat migraine.

ALDREDGE: That's correct.

CUTLER: What about breathing 100% oxygen for three to five minutes to relieve cluster headaches?

ALDREDGE: You can often—though not always—get beneficial results with oxygen. Breathing pure oxygen may decrease CO_2, producing central cerebral vasoconstriction.

CUTLER: What about interval treatment to prevent cluster headaches?

SILLER: It's unnecessary and ineffective. Treat the acute situation only.

Food can be at fault

If your patients can associate their headaches with any specific foods, restricting their diet may help. Here's a checklist of foods frequently implicated:

Food	Causative agent
Alcohol, particularly red wines and champagne	Histamine, tyramine
Strong or aged cheese, especially cheddar	Tyramine
Pickled herring	
Chicken livers	
Canned figs	
Pods of broad beans	
Chocolate	Unknown
Fish, especially smoked fish	
Dairy products	
Eggs	
Wheat	
Nuts	
Tomatoes	
Cured meats—such as hot dogs, bacon, ham, and salami	Sodium nitrite
Certain snack foods	Monosodium glutamate
Chinese food	

Tension headache

CUTLER: Now let's take up the case of a 56-year-old woman who has an al-

Migraine...and more

most constant headache occurring all over her head. There are no prodromals. The pain is dull and feels like a constricting band. She can sleep, but when she wakes up, the headache is still there. Occasionally she has nausea and even vomits. What are we dealing with, and how can we help?

FULLER: You've cited a classic example of tension headache, probably the commonest type of headache. It's mediated most commonly through increased tension of the posterior neck muscles, especially in association with psychological stress and unexpressed resentments. Many patients with tension headache don't go to a doctor—they take over-the-counter analgesics.

It's important to evaluate the headache patient for serious underlying psychiatric problems. Is he anxious? Depressed? Schizoid? Schizophrenic? Treatment varies accordingly. A checklist designed to elicit pertinent psychiatric information is helpful:

○ While taking a history, try to determine your patients' personality configuration by observing their habitual patterns of behavior and the coping mechanisms they repeatedly use.

The hysterical patient uses repression, suppression, and denial—often unconsciously and in a childlike manner playing a role such as that of a sick person or even a sexy doll—to avoid unpleasant realities and responsibilities.

Obsessive-compulsive patients have great difficulty in expressing and dealing with hostility and affection. Some may be distrustful and angry, maintaining a constant vigil for others who may do them harm.

Finally, among other identifiable personality configurations, there's the long-suffering, self-sacrificing martyr who unconsciously enjoys suffering.

○ Evaluate the patient's overall coping abilities. Does he adapt well under a variety of circumstances or fall apart under minor stress?

○ Determine how well your patient relates to the people in his life. Does the person have meaningful relationships with relatives, friends, and with you?

○ It's especially important to determine how well patients deal with the anger that accompanies frustration.

○ Look for the areas in which the patient is most likely to be experiencing life stress. In working men and women, suspect a job situation; in homemakers, it's likely to be interpersonal conflict with family members.

○ Finally, do a quick evaluation of mental status. First determine your patient's mood. Of course, it isn't always obvious—depressed patients don't always come in crying and saying they're sad. Instead, a patient may say, "I'm tired. I don't have any energy. I wake up in the middle of the night." Indeed, the depressed patient often has multiple somatic concerns.

Take note of your patient's sensorium. Is he confused or disoriented? Does he have difficulty remembering recent events? Such a patient may have a major neurological problem. Actually, many people who complain that they can't remember are depressed, and if you check, you may find that their memories aren't impaired.

Evaluate your patient's intelligence. People with low-normal intelligence—IQs of 85 or 90—may find it difficult to carry heavy responsibilities. This is especially true of patients who have large families.

Finally, in evaluating mental status, attend to your patient's thought processes. Consider the possibility of schizophrenia. Studies of the most refractory headache patients have revealed people with very poor coping abilities—even psychoses.

CUTLER: How do you select the prop-

The many causes of toxic vascular headache

- **Febrile illnesses**
  - Infectious mononucleosis
  - Influenza
  - Malaria
  - Measles
  - Mumps
  - Pneumonia
  - Poliomyelitis
  - Septicemia
  - Tonsillitis
  - Trichinosis
  - Tularemia
  - Typhoid fever
  - Viral hepatitis
- **Other conditions**
  - Alcohol hangover
  - Hypoglycemia
  - Hypoxia (especially with rising P_{CO_2})
  - Withdrawal from drugs: ergot (especially after prolonged treatment), caffeine, amphetamines, phenothiazines
- **Poisons**
  - Benzene
  - Carbon monoxide
  - Carbon tetrachloride
  - Insecticides
  - Lead
- **Other substances**
  - Alcohol
  - Barbiturates
  - Hydralazine HCl (Apresoline, Lopress)
  - Indomethacin (Indocin)
  - Monoamine oxidase inhibitors
  - Monosodium glutamate
  - Nitrates and nitrites
  - Oral progestational agents
  - Oral vasodilators
  - Tyramine

Second opinion

In my experience, ergotamines have no role in the treatment of cluster headache. In advocating the use of these drugs, Dr. Siller says that "a patient is usually awakened [by a cluster headache] at nearly the same hour each night." Actually it's extremely unusual for cluster headaches to occur at such specific time intervals. What's more, most cluster headaches last only 30 minutes, and it would take longer than that for ergotamine medications to be helpful.

As a rule, it's the cluster headache rather than the classic or common migraine that's most amenable to treatment with prophylactic medications. I believe that most physicians who treat headaches prefer to use methysergide maleate (Sansert) in cluster headache. However, it would seem that propranolol HCl (Inderal) may someday prove to be equally effective while having fewer potential side effects.

I agree that breathing 100% oxygen is extremely beneficial therapy in cluster headache patients. A number of my patients keep an oxygen cylinder at their bedside to abort a middle-of-the-night attack.

Also unmentioned is a precipitating cause for cluster headache. In my experience, alcohol and nitroglycerin preparations are classic examples. As a matter of fact, sublingual nitroglycerin (Nitroprn, Nitrostat) has been used diagnostically to precipitate a typical cluster headache—in about 45 minutes.

Finally, while I agree that narcotics should usually be avoided in severe migraine headaches, I disagree that ergotamines are helpful. In my experience, the ergotamines have no role in the treatment of severe headache accompanied by nausea and vomiting. Indeed, they may only make the patient sicker. Instead, I've found that chlorpromazine (Chlor-PZ, Promachel, Promapar, Sonazine, Thorazine) is the most helpful therapeutic agent: 75 mg IM usually stops the nausea and vomiting, aborts the headache, induces sleep, and avoids the problem of potential drug abuse. In some patients, I have equally good results using 100-mg rectal suppositories of chlorpromazine (Thorazine).

Charles G. Reul, MD
Ochsner Foundation Hospital
New Orleans

er therapy for the patient with a tension headache?

FULLER: First, use your knowledge of the patient's personality to establish a cooperative and supportive relationship with him—this helps the patient's usual coping methods work better. For instance, bright obsessive-compulsive patients should be given informative explanations.

With the hysteric, on the other hand, the doctor should respond to the patient's need for security and attention.

The martyr should be given recognition for his or her suffering, but should be challenged to work toward recovery rather than coddled. And with the distrustful, angry patient, try not to argue, but don't ignore the patient's complaints.

After establishing a therapeutic relationship, you can apply specific therapy to patients with particular clinical findings. You can usually help basically mature but acutely anxious headache patients by doing a good evaluation to rule out organic disease and offering reassurance that there's no serious medical problem that requires attention. Anxious patients are most likely to benefit from minor tranquilizers, such as diazepam (Valium), 2 to 5 mg or up to 10 mg four times a day, or chlordiazepoxide HCl (Librium), 5 to 10 mg four times a day. Aspirin, alone or in combination with phenacetin and caffeine, or with a barbiturate—or all four in a Fiorinal-type product—is also useful in tension headache.

Many depressed patients can benefit from tricyclic antidepressants. Amitriptyline, because of its greater sedative effect, is especially helpful for patients who are bothered by tension and agitation. For a depressed patient who's anergic and lacks motivation, a better choice is imipramine.

You can give supportive psychotherapy to some depressed patients, helping them to express their resentment and hostility and to feel comfortable with these emotions, or to accept giving up a cherished goal. However, some severely depressed patients should be referred to a psychiatrist, especially if you suspect suicidal tendencies.

Patients with long-standing, severe hypochondriasis are difficult to deal with. Most of them have problems besides headaches. Support these patients, accepting them as people who feel deeply inadequate and who need their headaches and other complaints to express their feelings of inadequacy and hostility. Don't try to talk with them too much about what's going on psychologically in their lives. Hypochondriacs often aren't psychologically oriented, and they adapt best

Migraine...and more

by being physically sick and seeking medical assistance. You can be of great help, especially if you don't try to rid them of their symptoms.

Finally, headache patients who are schizophrenic are likely to benefit from an antipsychotic agent such as haloperidol (Haldol) or a phenothiazine such as chlorpromazine (Chlor-PZ, Promachel, Promapar, Sonazine, Thorazine). Consider hospitalization only for those who are likely to benefit from such treatment or who can't adjust outside such a setting.

SILLER: Underlying depression is by far the most common cause of frequent, recurring headaches of all sorts. For this reason, the tricyclics are among the most valuable treatments.

Toxic vascular headache

CUTLER: Now, there's still another kind of headache besides the migraine, cluster, and tension types—the toxic vascular headache. I lump it with functional headaches, as it's not usually associated with organic brain disease.

SILLER: The common denominator is intracranial vasodilatation. Anything that dilates the large vessels at the base of the brain—for example, exercise, febrile illnesses, or drinking too much alcohol—can cause vascular headache, sometimes as severe as migraine headache. (Not all morning-after headaches are the result of intracranial vasodilatation, however; some may be due to depression about having made a fool of oneself the night before.) Hypoglycemia and hypoxia also threaten the integrity of the brain and cause vasodilatation. The challenge lies in finding out why the patient became hypoglycemic or hypoxic—not simply in determining that headache results from one of these conditions. Of course, headaches due to encephalitis or menin-

Beware of drug abuse

Headache patients—perhaps more than any others—abuse analgesics. When they have frequent recurrences, they tend to take larger quantities of drugs that have serious side effects.

For instance, methysergide maleate (Sansert) may cause fibrosis, while certain analgesics in large doses over a long period may cause renal damage. Aspirin causes GI bleeding. Ergot compounds provoke ergotism and may precipitate or worsen angina and peripheral vascular disease.

Of course, narcotics are to be avoided because it's so easy for a patient with chronic headache to get hooked on meperidine HCl (Demerol) or morphine sulfate. A physician should be especially cautious with patients who say their headaches are never relieved by anything but a shot of morphine.

Cutler

Headaches: Additional prescribing information

For more information on therapeutic agents discussed in this article, see Physicians' Desk Reference, 1976 edition.

	PDR page
Amitriptyline HCl	
Elavil (Merck Sharp & Dohme)	1070
Belladonna alkaloids, phenobarbital, and ergotamine tartrate	
Bellergal (Dorsey)	764
Chlordiazepoxide HCl	
Librium (Roche)	1290
Chlorpromazine	
Chlor-PZ (USV Pharmaceutical)	1548
Thorazine (Smith Kline & French)	1457
Cyproheptadine HCl	
Periactin (Merck Sharp & Dohme)	1080
Diazepam	
Valium (Roche)	1307
Dihydroergotamine mesylate	
D.H.E. 45 (Sandoz)	1344
Ergotamine tartrate	
Ergomar (Fisons)	805
Gynergen (Sandoz)	1345
Ergotamine tartrate and caffeine	
Cafergot (Sandoz)	1342
Ergotamine tartrate, cyclizine HCl, and caffeine	
Migral (Burroughs Wellcome)	679
Haloperidol	
Haldol (McNeil)	1011
Hydralazine HCl	
Apresoline (Ciba)	700
Imipramine HCl	
Imavate (Robins)	1263
Janimine (Abbott)	523
Presamine (USV Pharmaceutical)	1567
SK-Pramine (Smith Kline & French)	1448
Tofranil (Geigy)	827
Indomethacin	
Indocin (Merck Sharp & Dohme)	1074
Isocarboxazid	
Marplan (Roche)	1292
Isometheptene HCl	
Octin (Knoll)	858
Isometheptene mucate	
Octin (Knoll)	858
Isometheptene mucate, dichloralphenazone, and acetaminophen	
Midrin (Carnrick)	693
Meperidine HCl	
Demerol (Winthrop)	1654
Methysergide maleate	
Sansert (Sandoz)	1349
Nitroglycerin, sublingual	
Nitrostat (Parke-Davis)	1168
Phenelzine sulfate	
Nardil (Warner-Chilcott)	1628
Propranolol HCl	
Inderal (Ayerst)	591
Tranylcypromine sulfate	
Parnate (Smith Kline & French)	1438

gitis are actually organic in origin.

CUTLER: Do you ever have difficulty with rebound headache phenomena following vasoconstriction?

SILLER: Rebound usually occurs in patients taking ergot every day to avoid a migraine headache, or taking more ergot than they should. After the vasoconstricting drug is removed, rebound occurs, and patients may have the most devastating headache of their lives. □

SUGGESTED READING • Diamond S, Baltes BJ, Levine HW: A review of the pharmacology of drugs used in therapy of migraine. Headache 12:37, 1972 • Dalessio DJ: Dietary migraine. Am Fam Physician 6:61, 1972 • Reul CG: Diagnosis and management of common headache problems. Mod Treat 8:231, 1971.

CME Exam

This program is acceptable by the American Medical Association for one hour in Category 1 toward a Physician's Recognition Award. Familiarize yourself with the text, then take this test. Fill out the answer sheet (p 60) and mail it in with $5 to cover administrative cost, computer time, and the expense of mailing back the answers. You'll get a complete set of answers, notification of your grade and credit, and a printout of questions missed. Test answers will not be printed in CURRENT PRESCRIBING, *so please save the entire Text & Test section for reference.*

ONE: A 48-year-old executive suffers typical migraine headaches every two to three months. Your first treatment should be:

A Tranquilizers
B Antidepressants
C Ergotamine tartrate or dihydroergotamine by injection when he gets the first indication of an attack
D Meperidine HCl injection prn and instructions to go to bed
E Ergotamine tartrate and caffeine tablets, four at once, then one tablet every half hour until ten are taken

TWO: The same patient, who got good symptomatic relief from your treatment, returns two years later because his headaches have increased in frequency. They now come once or twice a month. A careful history rules out underlying depression, organic disease, and relation to specific foods. You would now:

A Continue the same management because it's effective
B Start methysergide, 2 mg tid
C Start propranolol, 20 mg qid
D Give a tricyclic antidepressant even though there's no clear-cut evidence of depression
E Prescribe Bellergal tid

THREE: Your patient is now 56 and president of his company. He's on preventive therapy, but still gets two or three attacks per year. During a recent physical exam, he complained of chest pain and intermittent leg pains and appropriate studies confirmed angina and claudication. For his headache attacks, you

A Continue the same management
B Cut the dose in half
C Change to oral isometheptene
D Give meperidine by injection and send him to bed
E Start psychotherapy

FOUR: You see a 42-year-old man with typical cluster headaches: During each two- to three-week cycle he awakes nightly with an excruciating headache. Last night he had his first headache this year. Your advice is:

A Take a sleeping capsule each night
B Start histamine desensitization
C Take dihydroergotamine at the onset of each attack
D Take two ergotamine tartrate and caffeine tablets an hour before the predicted onset of the headache
E Drink two ounces of alcohol at bedtime

FIVE: A 65-year-old woman complains of almost constant headaches. They feel like a tight band, are partly relieved by aspirin, but seem unrelated to anything she does or eats. Appropriate examinations rule out an intracranial organic disease. She doesn't seem to be anxious, but she's depressed and is tired. If she has no organic disease, the treatment of choice is probably:

A Diazepam, 2 to 5 mg tid
B Amitriptyline, 25 mg tid
C Aspirin 0.6 gm qid
D Imipramine, 25 mg tid
E Either B or D

SIX: Concerning birth control pills and their relation to headache, which of the following statements is false?

A Patients with headaches shouldn't take the Pill
B Patients who develop headaches after starting the Pill should stop oral contraceptive therapy
C Patients with headaches that seem to be made worse by the Pill should stop oral contraceptives
D The etiologic relationship between birth control pills and headaches hasn't been definitely established

SEVEN: A 48-year-old man suffers from typical cluster headaches once or twice yearly. The last episode ended a week ago. He would like very much to avoid another series. You advise:

A A series of histamine injections
B Methysergide for prevention
C Nothing
D Avoid coffee, tea, cola drinks

Continued on page 58

LIDEX® (fluocinonide)
0.05% Cream and Ointment
TOPSYN® (fluocinonide)
0.05% Gel

Summary of prescribing information

DESCRIPTION
LIDEX cream 0.05%, LIDEX ointment 0.05% and TOPSYN gel 0.05% contain the active compound fluocinonide. Fluocinonide, which is the 21-acetate ester of fluocinolone acetonide, has the chemical formula $6\alpha, 9\alpha$,-difluoro-11β, 16α, 17α, 21-tetrahydroxypregna-1,4-diene-3, 20-dione, cyclic 16, 17-acetal with acetone 21-acetate.
LIDEX cream contains fluocinonide in FAPG® cream, a specially formulated cream base consisting of stearyl alcohol, polyethylene glycol 6000, propylene glycol, 1, 2, 6-hexanetriol and citric acid. This white cream vehicle is greaseless, non-staining, anhydrous and completely water miscible. The base provides emollient and hydrophilic properties. In this formulation, the active ingredient is totally in solution.
LIDEX ointment contains fluocinonide in a specially formulated ointment base consisting of Amerchol CAB (mixture of sterols and higher alcohols), white petrolatum, propylene carbonate and propylene glycol. It provides the occlusive and emollient effects desirable in an ointment. In this formulation the active ingredient is totally in solution.
TOPSYN gel contains fluocinonide in a specially formulated gel base consisting of propylene glycol, propyl gallate, disodium edetate, and Carbopol 940 (carboxypolymethylene) with NaOH and/or HCL added to adjust the pH. This clear, colorless, thixotropic vehicle is greaseless, non-staining and completely water miscible. In this formulation, the active ingredient is totally in solution.

INDICATIONS
LIDEX cream and ointment and TOPSYN gel are intended for the relief of inflammatory manifestations of corticosteroid responsive dermatoses.

CONTRAINDICATIONS
Topical steroids are contraindicated in vaccinia and varicella. Topical steroids are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparation.

PRECAUTIONS
If irritation develops, the cream, ointment or gel should be discontinued and appropriate therapy instituted. In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted.
If a favorable response does not occur promptly, the corticosteroid cream, ointment or gel should be discontinued until the infection has been adequately controlled.
If extensive areas are treated, the possibility exists of increased systemic absorption and suitable precautions should be taken.
Although topical steroids have not been reported to have an adverse effect on pregnancy, the safety of their use in pregnant females has not absolutely been established. Therefore, they should not be used extensively on pregnant patients, in large amounts or for prolonged periods of time.
LIDEX® (fluocinonide) cream and ointment and TOPSYN® (fluocinonide) gel are not for ophthalmic use.

ADVERSE REACTIONS
The following local adverse reactions have been reported with topical corticosteroids:

burning	folliculitis
itching	acneform eruptions
irritation	hypopigmentation
dryness	striae
secondary infection	skin atrophy

DOSAGE AND ADMINISTRATION
A small amount should be gently massaged into the affected area three or four times daily, as needed.

HOW SUPPLIED
LIDEX 0.05% Cream—15 g 30 g and 60 g tubes.
LIDEX 0.05% Ointment—15 g 30 g and 60 g tubes.
TOPSYN Gel 0.05% 15 g and 60 g tubes.

SYNTEX
SYNTEX LABORATORIES, INC.
PALO ALTO, CALIFORNIA 94304

Migraine...and more

E Diazepam, 5 mg tid

EIGHT: The role of certain foods in the precipitation of migraine or migraine-like headaches in selected patients is well recognized. Which of the following statements is true?
A Diet and specific foods play some role in every patient with migraine headaches
B Aged cheeses may cause headaches because of the tyrosine content
C Cured meats and hot dogs should be avoided in selected migraine patients because of the sodium nitrite additive
D The patient needs a high-carbohydrate diet to avoid hypoglycemia
E Italian food should be avoided, as it's high in monosodium glutamate, a frequent offender

NINE: The use of analgesics for headache pain and the use of vasoconstrictors for treatment and prevention of the underlying pathophysiology is fraught with risks and complications. Which of the following drugs is not properly matched with a serious complication?
A Methysergide—retroperitoneal fibrosis with hydronephroses
B Oral isometheptene—termination of pregnancy
C Phenacetin—renal failure
D Ergotamine—peripheral vascular disease
E Aspirin—gastrointestinal bleeding

TEN: Toxic vascular headaches can result from many diverse factors, but cerebral vasodilatation is the common denominator. Which of the following won't cause this type of headache?
A Influenza
B Excessive alcohol intake
C Insulin overdose
D Hyperventilation (with low P_{CO_2})
E Vigorous exercise

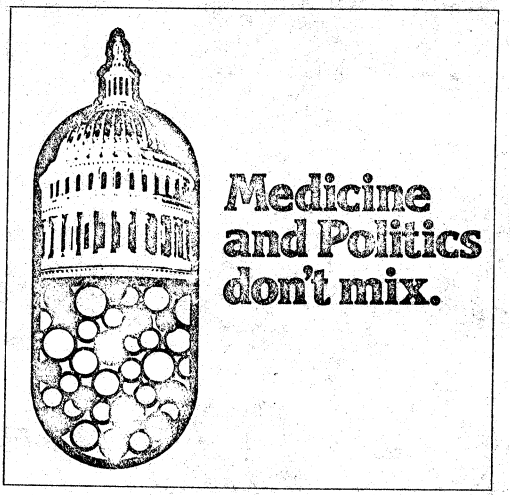
DARVOCET-N® 100
propoxyphene napsylate with acetaminophen
Indication: For the relief of mild to moderate pain, either alone or accompanied by fever. **Contraindications:** Hypersensitivity to propoxyphene or to acetaminophen. **Warnings:** *Drug Dependence*—Propoxyphene can produce drug dependence characterized by psychic dependence and, less frequently, physical dependence and tolerance. Propoxyphene will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to that of codeine although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine. *Usage in Ambulatory Patients*—Propoxyphene may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as driving a car or operating machinery. The patient should be cautioned accordingly. *Usage in Pregnancy*—Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, propoxyphene should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards. *Usage in Children*—Propoxyphene is not recommended for use in children, because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric age group. **Precautions:** Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The central-nervous-system depressant effect of propoxyphene may be additive with that of other C.N.S. depressants. **Adverse Reactions:** The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients, and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include constipation, abdominal pain, skin rashes, lightheadedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses exceeding 800 mg. per day has caused toxic psychoses and convulsions. [011976]

DARVON® COMPOUND-65
propoxyphene hydrochloride, aspirin, phenacetin, and caffeine
Indication: For the relief of mild to moderate pain. **Contraindication:** Hypersensitivity to propoxyphene, aspirin, phenacetin, or caffeine. **Warnings:** *Drug Dependence*—Propoxyphene can produce drug dependence characterized by psychic dependence and, less frequently, physical dependence and tolerance. Propoxyphene will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to that of codeine although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine. *Usage in Ambulatory Patients*—Propoxyphene may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as driving a car or operating machinery. The patient should be cautioned accordingly. *Usage in Pregnancy*—Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, propoxyphene should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards. *Usage in Children*—Propoxyphene is not recommended for use in children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric age group. Salicylates should be used with extreme caution in the presence of peptic ulcer or coagulation abnormalities. **Precautions:** Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The central-nervous-system depressant effect of propoxyphene may be additive with that of other C.N.S. depressants. Phenacetin has been reported to damage kidneys when taken in large amounts for a long time. Salicylates may enhance the effect of anticoagulants and inhibit the uricosuric effect of uricosuric agents. **Adverse Reactions:** The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients, and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include constipation, abdominal pain, skin rashes, lightheadedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses exceeding 800 mg. per day has caused toxic psychoses and convulsions. [011975]

Lilly
Additional information available to the profession on request.
Eli Lilly and Company, Inc.
Indianapolis, Indiana 46206
500J93

County Medical Societies Campaign to Beat Drug Substitution

by the editors of PRIVATE PRACTICE



Increasing pressure on the Oklahoma State Legislature to repeal Oklahoma's strong anti-substitution law had doctors worried. "When I write a prescription for one of my patients," Dr. Orange Welborn of Ada, Oklahoma, told PRIVATE PRACTICE, "I want him to get the exact drug I prescribe — not a cheap generic some bureaucrat in Washington claims is equivalent to the brandname I have used and trust."

But lobbying by labor unions, consumerists, and retired people's groups — using the chimera of reduced prescription drug costs — was getting close to success. Soon, Oklahoma doctors might have their prescriptions changed and generic drugs substituted without their or their patients' knowledge.

So instead of wringing their hands, the Pottawatomie and Pontotoc County Medical Societies and Pharmacists Associations decided to do something about it. With the help of the Congress of County Medical Societies, they launched a massive media campaign in their Oklahoma counties — using TV, radio, and newspapers — to stop the repeal of the anti-substitution law. So far as PRIVATE PRACTICE has been able to discover, this campaign is unique in American medicine. "We know constituent pressure *does* influence legislators as nothing else does," Dr. Welborn said, "and we are mobilizing the voters — our patients — through advertising explaining that drug substitution can hurt their health. Believe me, the results so far show that the politicians listen, and listen very carefully."

THE RISKS OF SUBSTITUTION

Dr. Francis A. Davis, chairman of the Pottawatomie County Medical Society's legislative committee, noted: "If all the chemically identical medicines available for substitution were really the same, there would be no danger to our patients at all. But just as a diamond and a chunk of coal start with the same chemical composition, and wind up completely different, so do medicines.

"Those differences are enough to kill our patients or to cure them or to make them sicker or to make no difference at all. Substitution plays Russian roulette with our patients' health, and we just aren't willing to sit back and let it happen."

"One of the most serious instances," added Dr. Welborn, head of the campaign for the Pontotoc County Society, "concerned digoxin. In tests, equal dosages of different manufacturers' digoxin were absorbed at startlingly different rates. In some cases, myocardial infarctions and deaths resulted. Lanoxin avoids these troubles. And if I prescribe Lanoxin, I don't want a generic digoxin substituted. If drug substitution becomes law, I'll have no way of knowing what my patients eventually get."

"The quality of medical care is certain to deteriorate," says Dr. Davis, "if the physician's choice of therapy is subject to economic considerations over purely medical ones. In fact, the Judicial Council of the AMA has held that 'the physician has an ethical responsibility to assure that high-quality products will be dispensed to his patients. Obviously, the benefits of the physician's skill are diminished if the patient receives drugs . . . of inferior quality.' If a doctor cares about his patients and cares about the quality of medicine he practices, he *must* work to stop drug substitution."

DOES SUBSTITUTION SAVE MONEY?

Proponents of drug substitution say it would save substantial sums of money for patients, but Roy Kelly, MD, of Shawnee, Oklahoma, disagrees: "Studies have shown that generic substitution would save patients 1.7¢ on the dollar at most. And, in fact, in Saskatchewan, Canada, prescription prices rose an average 19% after substitution was enacted, probably because pharmacists' malpractice premiums went up so much. In Kane County, Illinois, doctors and pharmacists tried substitution. The experiment was ended after fifteen months, because patients didn't save any money."

The anti-substitution campaign ended with a two-hour movie, and the commercial breaks were used to answer viewers' questions on substitution. During a segment of the movie, moderator Orange Welborn, MD (third from left), confers with Francis A. Davis, MD, president of the Congress of County Medical Societies (fourth from left). The other panel members are J. B. Wallace, MD (far left), William Bryan, RPh (second from left), and Gordon Richards, RPh (far right). In the background, members of Dr. Welborn's office staff have telephoned questions from viewers. 135 were received.



Gordon Richards, RPh, of Shawnee, told PRIVATE PRACTICE: "I tell my customers that they've got to remember that today's medications are more powerful than they used to be. So while individual tablets or capsules may cost more, it takes fewer to treat them. Because of this, the average dosage price has gone down, not up, since 1960."

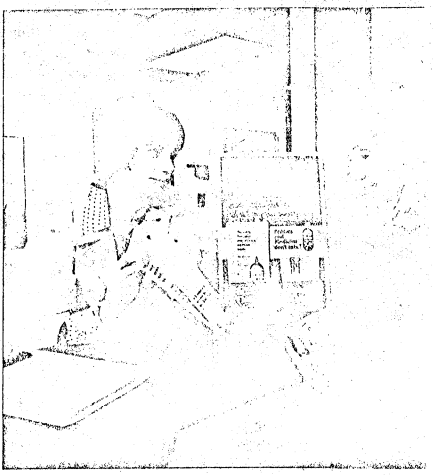
PHARMACISTS ALSO OPPOSED TO SUBSTITUTION

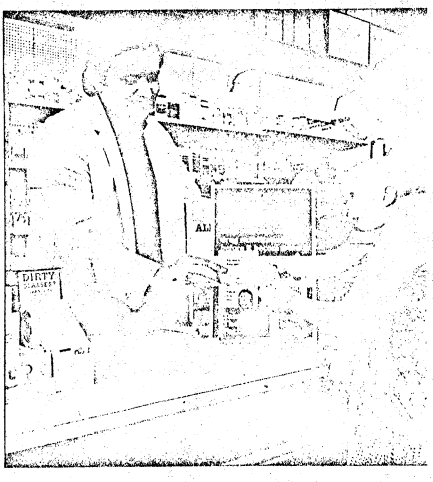
The American Pharmaceutical Association has been pushing hard for substitution since 1970, even though it was instrumental in getting anti-substitution laws passed in the early 1950s. Because of the APhA, many think that all pharmacists want substitution. But ethical pharmacists do not, and the Pottawatomie and Pontotoc County Pharmacists Associations joined their medical colleagues in fighting it. Adds pharmacist Richards: "Drug substitution is bad for the prescriber, for the pharmacist, and, most of all, for the patient. I am completely opposed to it." The pharmacist's scientific training makes him realize all the factors that can make chemically equal drugs act differently in the body. "In my day-to-day dispensing, I have seen the unsatisfactory results of one drug, as compared to a similar drug, in the experience of my customers," said another pharmacist.

"Drug substitution severs the lines of communication between the pharmacist and physician," said Mr. Richards. "It eliminates the 'teamwork' from the health-care team by taking the physician out of the final choice of the drug to be used. When the pharmacist and physician work independently rather than together it is the patient who suffers most. It is in the best interest of the patient for the pharmacist to consult the physician before making any changes in the prescription. If drug substitution were legalized, neither the physician nor the patient could be certain that a prescription was dispensed exactly as written. It is important that the doctor and patient have confidence that the medicine dispensed was what the doctor intended."

ADVERTISING

In the Autumn of 1975, representatives of the two county medical societies met in Oklahoma City with the Congress of County *(continued on page S-4)*





A doctor's office (above) and a pharmacy (below) during the anti-substitution campaign.

(continued from page S-3) Medical Societies and a wellknown local advertising agency, Adsociates. Out of the meeting grew the indepth TV, radio, and newspaper effort to stop substitution in its tracks.

Working closely with physicians in private practice, Berry Wheeler, president of Adsociates, and his staff, crafted six full-page newspaper advertisements, three TV spots, and two radio spots (see Appendix). During the five weeks, the full-page newspaper ads appeared regularly in the daily newspapers of the two counties, accompanied by good editorial support. The 60-second radio and 30-second TV spots were used sparingly at first, and built to a crescendo during the last week. All told, the advertisements appeared on radio 700 times, on TV 99 times, and in newspapers, 28 times.

Each ad was designed to create interest and action. People were given a local phone number in each county, manned 24-hours a day, seven days a week, to call for information. Everyone who wanted more information was sent a pamphlet, "Why Prescription Drug Substitution is Bad Medicine for Oklahoma," written for lay people by Francis A. Davis, MD, and Gordon Richards, RPh (see Appendix), which contained a card to fill in and send to local representatives. Some people wanted to protest immediately to their legislators, and so cards were sent in their names by the two county medical societies.

As a backup, every doctor's office and pharmacy in the two counties had a large counter card on prominent display, headlined: "Oklahomans, Help Yourselves! Stop Prescription Drug Substitution Before It Becomes Law!" In two pockets at the bottom of the card were the Davis-Richards pamphlets and pre-stamped protest cards.

The last night of the campaign, Saturday, February 21st, two hours of TV time were purchased for a movie — *The Bravos*, starring George Peppard — and the 20 minutes of commercial breaks were used to take phone calls from patients about the issue; a panel of doctors and pharmacists answered their questions, telethon-style.

POLITICS AND MEDICINE DON'T MIX

Since the March issue of PRIVATE PRACTICE had to be at the printer before the final results were in, they will be reported in our April issue. But there is enough evidence to support Shawnee's Dr. Leon Combs, who called the effort a "real success." Other county societies in Oklahoma are gearing up to repeat the campaign in their areas, if necessary. But there is no doubt that in the population of Pottawatomie and Pontotoc Counties, some 70,000, thousands of people have become militant substitution-fighters. Many protest cards and letters hit the State Capitol, causing quite a stir, and probably killing drug substitution in Oklahoma for the foreseeable future. The people agreed with the campaign's slogan, "Politics and Medicine Don't Mix." Interestingly, older people led in number of protests, giving the lie to lobbyists who claim that retired people support substitution.

One letter from a woman in Shawnee to her state legislator read: "I am gravely concerned about the bill before the legislature which will permit drug substitution for the person preceding a prescription from his or her physician to a drug store.

"I feel this is a very dangerous bill and could have far reaching effects on the individual patient. I have a chronic ear condition and the particular drug prescribed by my physician is quite effective in giving relief. If I should have this drug substituted it could increase the infection or even might cause loss of hearing. In fact, this particular bill seems to be the most dangerous legislation which has been presented to the legislature.

"Please exert every effort to defeat this particular bill and you will certainly have contributed a great service to all the people in your district."

THE FUTURE

"Now that we know this kind of approach works — and works well," notes Dr. Combs, "we're ready to share it with all the other county medical societies in America. Any county society wanting to run a similar campaign will have to pay only for the media time — the Pottawatomie and Pontotoc County Societies will supply video tapes, audio tapes, and newspaper mats for free. Any county society that wants more information can write to the Congress of County Medical Societies [3037 Northwest 63rd Street, Oklahoma City, Oklahoma 73116] or Adsociates [5929 North May Avenue, Oklahoma City, Oklahoma 73112].

"Right now our societies and the CCMS are working to design a media campaign against the disaster of National Health Insurance for use in selected Congressional districts. The socialists haven't won this battle yet." PRIVATE PRACTICE will be reporting on this soon. □

Appendix

Here follow the texts of the anti-substitution pamphlet, and the radio and TV spots, along with actual reproductions of the full-page newspaper advertisements, that were used so successfully in Oklahoma. Newspaper mats, video and audio tapes, and the rights to the pamphlet, are available to any county medical society wishing to sponsor a similar campaign, free of charge. Write to the Congress of County Medical Societies, Inc., 3037 N.W. 63rd St., Oklahoma City, Oklahoma 73116.



Why Prescription Drug Substitution is Bad Medicine for Oklahoma

by
Francis A. Davis, MD, and
Gordon Richards, Jr., RPh
Shawnee, Oklahoma

You may never have heard of Oklahoma's anti-substitution law, yet it's one of the most important pieces of consumer-protection legislation ever put on the books. Why? Because it helps protect your health — and the health of every patient in Oklahoma. There's nothing more important than that.

But the Oklahoma anti-substitution law, and similar laws in most other states, are under attack. Right now, legislation is pending in the State House of Representatives to repeal this extraordinarily valuable law.

What The Law Says

Briefly, the law says that a pharmacist must fill the prescription your doctor writes for you *exactly as written.* Proposed legislation would encourage the pharmacist to substitute a different drug for the one your doctor prescribes, *without his prior knowledge or consent.*

Anti-substitution laws were first passed in the 1950s to curb substitution abuses. Pharmacists, doctors, and legislators recognized that prescription drug substitution was unethical and definitely not in the best interests of patients or professionals.

(continued on page S-6)

Three Myths of Prescription Drug Substitution

Twenty years later, a minority of pharmacists, some politicians, and groups claiming to speak for consumers and older Americans, are pushing for the repeal of anti-substitution laws.

The repeal effort is based on three myths:

ONE, that pharmacists are more knowledgeable than physicians about prescription drug products;

TWO, that patients would reap large savings if "generically equivalent" drugs were substituted for brandname products;

THREE, that prescription drugs with the same chemical ingredients will have the same therapeutic effect.

Myth One

Pharmacists are very important, indeed indispensable, members of the patient-care team. But when it comes to knowing how drugs work in *people*, rather than in the abstract, physicians have more training and experience.

Your doctor has listened to you, questioned you, examined you, and made a diagnosis of your condition. The drug he prescribes is based on the therapeutic results he has witnessed in previous cases. Only the physician knows the particular diagnosis for each case, and only the physician should have the final choice of drug to be used for it.

The pharmacist is qualified to give you valuable advice concerning the usage, side effects, contraindications, and usual dosage of your prescription; however, deciding what drug would work best for you is the right and responsibility of the physician. Without the final authority to choose the drug, the physician cannot effectively control the patient's therapy.

Myth Two

Then there is the claim that substitution will save you money. An independent research firm did an extensive survey to determine what the savings would be if all drugs were prescribed generically — essentially the same as permitting pharmacists to substitute. The savings of prescribing by chemical rather than brandname (using more cheaply made drugs) would average only 1.7% (or 1.7¢ on the dollar). Companies that cut corners in manufacturing and quality control can sell more cheaply than the national firms that do not, but not by a wide margin. In fact, America's pharmaceutical industry has an enviable record in holding down costs. On the whole, the average tablet or dose costs less today than in 1960. There aren't many other products you can say that about.

But the study above is only a projection. What about the places where substitution has actually been implemented?

It's legal in Saskatchewan, Canada, and instead of prices going down, they went *up* — 19% on the average. Some think the culprit was the increased cost of malpractice insurance for pharmacists. When they started substituting, their liability increased.

In this country, fifteen months of substitution in Kane County, Illinois, ended when no savings to patients could be shown. In Massachusetts, Maryland, and Kentucky — where substitution has been implemented — no savings has resulted.

Myth Three: The Critical Issue

Myths Numbers One and Two are not the critical issues, however. Myth Three, and the risk it can pose to your health, is.

Drug expert William H. Havener, MD, gives seven reasons — and he says there are many more — why chemically equivalent drugs may not have the same effect.

1) *Purity.* Purity can vary greatly. Generic (nonbrandname) penicillins can contain up to 15% impurities, says the Food and Drug Administration, and still be sold. Granted, penicillin impurities are very hard to remove, but reputable brandname penicillin is 98% pure. In fact, people with "penicillin allergies" are often allergic to the impurities rather than the penicillin;
2) *Stability.* A product's package doesn't seem very important, but ill-packaged drugs can quickly deteriorate and become unusable... but by then, you've already paid for them;
3) *Taste, smell, color, consistency.* If you have children, you know how important these are in medicines. They're also vital in longterm adult therapy;
4) *pH.* What is the degree of acidity or alkalinity?
5) *Coating.* The right kind of coating protects sensitive medicines against destruction by stomach acids. The wrong kind can permit a pill

or capsule to pass through the body undissolved, with no medical effect whatsoever;

6) *Deterioration.* Some drugs, if improperly produced, can deteriorate to ineffective or toxic substances. The widely used antibiotic tetracycline, if dispensed in relatively acidic capsules, slowly transforms into a deadly kidney poison. Without appropriate — and costly — safeguards, this kind of problem can occur;
7) *Absorption.* How well a medicine is absorbed into the body depends on many factors, including how rapidly it dissolves, the nonactive ingredients used, stability in digestive juices, and how it reacts with food in the stomach.

The Dangerous Effects of These Differences

Here are some specific examples from medical journals that also received some attention in newspapers.

A few years ago, it was discovered that while Chloromycetin (brandname) is a very powerful and effective antibiotic for certain infections, all the generic equivalents of chloramphenicol (chemical name) would not do the job, no matter *how* much was given to patients.

Digoxin (generic name) is used by millions of Americans to help their hearts beat more forcefully. In 1974 the Food and Drug Administration discovered that some manufacturers' digoxin varied so much in absorption rate from batch to batch, that patients could get dangerously high or low amounts from the *same* dosage. The FDA also noted that Lanoxin (brandname) had no such problem. It was a little more expensive, but it worked, unlike its cheaper counterparts.

Alan Tasoff, MD, writes of his experiences as an Air Force doctor in Thailand in 1972: "Struggling to overcome a penicillin-resistant gonorrhea epidemic among airmen — of the magnitude of twenty new cases per day — we were armed with an Italian-manufactured tetracycline, purchased in massive quantities by Congress. The drug was chemically equivalent — in the judgment of consumer groups — to brandname drugs. The failure of this drug to dissolve in the alimentary tract was known to all physicians prescribing it, but supplies had to be consumed before a replacement could be made available. The ultimate cost to the airmen involved was chronic, intractable urethritis and prostatitis."

Two Branches of the Government Say All Drugs Aren't Equal

With all the controversy surrounding this question, the United States Senate Health Subcommittee asked the Office of Technology Assessment (OTA), an agency of Congress, to study the whole problem of prescription drug bioequivalence (whether chemically equal drugs will be equally available in the body, therefore allowing them to have an equal effect).

The OTA set up a Drug Bioequivalence Study Panel, and asked Dr. Robert M. Berliner, dean of the Yale University Medical School, to be its chairman. After months of intensive study, the panel released its report in July 1974. Among its findings were:

"Current standards and regulatory practices do not assure drug bioequivalence for drug products."

"Present... guidelines do not insure quality and uniform bioavailability for drug products. Not only may the products of different manufacturers vary, but the product of a single manufacturer may vary from batch to batch or may change during storage."

"The problem of bioinequivalency in chemically equivalent products is a real one."

The Food and Drug Administration has so far identified 193 categories of drugs, including thousands of different products, as having known or strongly suspected equivalence problems.

The People Say They Don't Want Prescription Drug Substitution

In a democracy, what the people think counts. So it's important to know the findings of the national and two state surveys on the relative importance of drug cost, quality, effectiveness, safety, and bioequivalence. 2,532 people were surveyed all across America, and 1,149 people were questioned in California and Wisconsin.

Seventy-one percent of the people questioned nationally believe the physician, not the pharmacist, should determine which drug product a patient takes. (In the state surveys, the percentage was 75.)

In the state surveys, people placed far greater importance on effectiveness and safety of prescription drugs than on their cost or speed of relief. In fact, cost was last by a decisive margin.

Over 70% in the national poll said they didn't want substitution of generic drugs for those prescribed,

(continued on page S-8)

even if the substitution resulted in a cost savings. In California, people aged 55 and over oppose pharmacist drug-selection by a whopping margin of 83%.

What it all boils down to is this: people don't want to take chances with their health to save a little money.

If Substitution Were Allowed

Imagine, for a moment, that the Oklahoma anti-substitution law has been repealed. You're sick, and you go to your doctor. He examines you and prescribes a medication. But without his knowledge, the pharmacist substitutes a cheaper product. It doesn't work, but your doctor doesn't know why, since he doesn't know about the substitution. He knows how the drug he prescribed works, but you're not taking it. Did the substitute drug deliver too little medication into your bloodstream? There's no telling. In the meantime, you're incurring additional expenses because of prolonged illness.

Generic drugs may cost a little less in dollars and cents, but they can exact a high price in health.

If your doctor is to do his best for you, he must be free to use his best judgment, in drug therapy and every other area. He must be free to choose the drug he knows will help you the most.

If Oklahoma's anti-substitution law is repealed, you may be getting drugs from low-quality-control, no-research, minimum-distribution, fly-by-night companies.

All for the sake of savings that will never materialize.

How to Save Money on Medicine Right Now

Carefully choose a reputable pharmacy, conveniently located, that will serve you with quality drugs at reasonable prices. Make sure it has the services you want. Can you get quick service on filling prescriptions? Is there free home delivery? These, of course, can affect prices.

Talk to your doctor about the quality and price of your medication. Tell him you want the least expensive product consistent with your medical needs. Make sure you get it by asking your doctor to specify that particular company's product on your prescription.

If your condition requires longterm therapy, ask your doctor to consider prescribing a larger supply. This will usually cut the cost per dosage.

Keep a record of all your prescription drug purchases — or ask your pharmacist to do so. Around income tax time, this will be invaluable in computing your medical deduction.

Oklahoma's anti-substitution law protects you and your family, whenever sickness threatens.

What to Do . . . Right Now

Use the attached card to write to your State Representative or Senator. Tell him you object to the repeal of Oklahoma's strong anti-substitution law, one of the best consumer protection laws we've got.

Do it now. The alternative is enough to make you sick.

If you are convinced Prescription Drug Substitution (Generic Medicine) is bad for Oklahoma, please tell your Representative or Senator . . . just fill in the spaces on the card at right. Address it, sign it, stamp it. And send it.

"I oppose Prescription Drug Substitution and as a concerned Oklahoman, I urge you to fight any proposed legislation that would bring such bad laws into our state."

Name _____

Address _____

City _____

Signature _____

Address the card to your appropriate Senator or Representative.

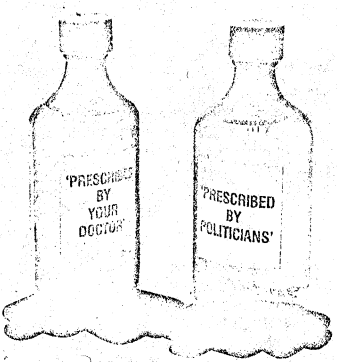
POTTAWATOMIE COUNTY:
Representative Charles Henry
Representative James Townsend
Senator Ralph Graves

PONTOTOC COUNTY:
Representative Lonnie Abbott
Senator Wes Watkins

Newspaper

Published in the *News-Star*, Shawnee, Oklahoma, and the *Evening News*, Ada, Oklahoma.

Who do you trust with your life?



Lately there's been a lot of palaver about the excessive profits big drug companies are making.

And now the politicians have come up with a sure-fire solution to knock the props out from under them: Permit prescriptions calling for the brand name drugs of these firms to be filled with substitutes that may not be therapeutically equal. In fact, they not only may not cure you . . . they may even make you sicker.

They say prescriptions filled using generic (or chemical) names for the drugs instead of the brand names will save you money.

They say since all drugs with the same chemical compound — no matter what they're named — are the same thing.

In the midst of all this rhetoric that amounts to playing politics with your money and well-being, we say, "Those big drug companies didn't get big by making crummy medicine."

On-going research. Stringent quality control. Professional integrity. Years of experience. That's what made those big drug companies big. That's what made your doctor trust them. That's what made their brand name mean something.

Who stands behind generic medicine? Who knows? It could be a reputable manufacturer. It could be a small or marginal company producing to the lowest quality control standards permitted under the law. You can't be sure if drug substitution is allowed. It could be an unknown firm who does little if *any* research to improve the effectiveness of the medicine you get and to find new drugs to treat you.



Make you think? Try this: No one really knows if generic drug substitution will save you money or not. In one area where it was legalized the price of medication rose 19¢ on the dollar.

Now try this: Those "cheaper" chemically equal drugs aren't equal either. Despite the identical chemical ingredients, the slight differences in fillers, coatings, acidity and absorption rate in drugs can make them totally ineffective — or worse than that — potentially harmful and even fatal.

Okay, we'll ask you again. Who do you trust with your life? Those big profitable drug companies that put their name on the line every time you take a pill? Or those who don't?

Prescription drug substitution legislation is now pending right here in Oklahoma, so it's imperative that you answer the question for yourself, and then act.

Call 273-8838 Now!

If you're already convinced, tell us when you call. We'll send a protest in your name to your State Senator and State Representative, or write your own letter. But don't wait. *Now* is the time to take action.

The alternative is enough to make you sick.

Medicine and Politics don't mix.

This campaign to Stop Prescription Drug Substitution Legislation sponsored by the Pottawatomie County Medical Society and Pharmaceutical Association

Published in the *News-Star*, Shawnee, Oklahoma, and the *Evening News*, Ada, Oklahoma.

Soon, the law could say your doctor can't prescribe the medicine he thinks is best for you.

For instance. You get pneumonia. Your doctor wants you to be treated with a drug trademarked "Terramycin." But he can't be sure you will get it because of proposed drug substitution legislation. He knows it is manufactured by a reputable pharmaceutical firm to the highest quality standards, but in Oklahoma, under proposed law this may not be enough to insure that your prescription is filled as written unless extraordinary steps are taken by the doctor.





Granted. Oxytetracycline is chemically equal to Terramycin, but this does not mean it is therapeutically equal. It is well known that it does not act the same way in your body to produce desired results. Coatings, fillers and quality control can make the difference ... and can make you sick.

Doctors know for a fact that no Oxytetracycline works so well as Terramycin. But that seems to make no difference to supporters of prescription drug substitution ... no matter how much difference it could make to you, the pneumonia patient.

This is just one vital drug. There are literally *hundreds* more that have proved to be less effective when manufactured to careless quality standards. Or worse. Some drugs are KNOWN to be totally ineffective in all forms except those marketed by reputable pharmaceutical firms.

How could anyone dream of sponsoring such an idiotic law? Easy. It's a law that can be made to sound terrific. It's a law that'll let politicians say, "You're paying too much for drugs. The big pharmaceutical companies are ripping you off. This law will mean you'll get the same drugs for a lot less money."

Well, you've seen they're not the same drugs — though they may be made with the same chemical substances. Now to examine that "less money" bill of goods.

One, do you really want to pay less money when your health is at stake?

Two, not a single advocate of these so-called prescription drug substitution laws has proved that such laws will cut your drug bills any more than about 2¢ on the dollar. A very small premium to pay for your health.



Three, your overall medical bills may GO UP. You may have to go back to the doctor several times when lesser quality drugs don't work. Your doctor will probably have to pay even higher malpractice insurance premiums. So will your pharmacist.

You think no one in his right mind could endorse such a law? You're wrong. It's already on the books in 11 states. A similar law has already passed the House in Oklahoma and is pending in the State Senate. It could become law here unless you act fast.

Call this number for more information. If you're already convinced, tell us when you call. We'll send a protest in your name to the people in Oklahoma City who can prevent this disaster — your state legislators. Or write your own letters. But don't wait. *Now* is the time to take action. Write your letters today or

Call 332-4116 Now!

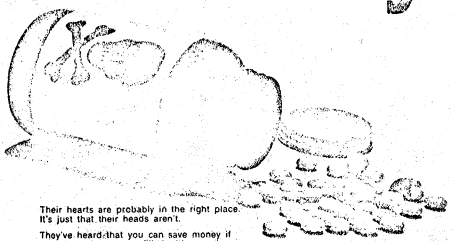
The alternative is enough to make you sick.

Medicine and Politics don't mix.

This campaign to Stop Prescription Drug Substitution Legislation is sponsored by the Pontotoc Medical Society and the Ada Pharmacists Association.

Published in the *News-Star*, Shawnee, Oklahoma, and the *Evening News*, Ada, Oklahoma.

They're out to save you money on medicine... even if it kills you.



Their hearts are probably in the right place. It's just that their heads aren't.

They've heard that you can save money if your prescriptions are filled with generic or chemical name drugs with the same chemical compound as brand name medication.

And they've jumped on the legislative bandwagon. Possibly without realizing that the law is flying in the face of everything your doctor has learned.

Should that bill become law, your doctor will have to write prescriptions specifically including a statement that "bioequivalence" is considered critical in order to prevent the substitution of another so called "equal" drug. "Bioequivalence" or "therapeutic" equivalence means that the drug will produce the same effect in your body as the brand-named drug prescribed by your doctor. Most generic or chemically equivalent drugs do not, in fact, produce the same results because of differences in coating, fillers, quality control, acidity and absorption rates!

If all generic drugs having the same chemical compound could do the same job, that would be one thing. But *all* generic drugs aren't the same.

And that difference could be a killer.

Take plain ol' Oxytetracycline. It's an antibiotic, a common one, and chances are someone in your family has taken it.

But if that harmless little drug is manufactured under slipshod quality control conditions, if the chemical compound is encased in slightly acidic capsules, it can slowly transform into a deadly kidney poison.

Yet, quality control is one place corners can be cut to bring you cheaper generic name drugs.



And that's the kind of risk they want you to run. Even though nobody's really proved that prescription drug substitution can save you money.

The fact is, in one area with such a law, a recent study found that prescription prices went up an average of 19¢ on the dollar after the law was passed.

It's high time you bypassed these do-gooders and did a little good for yourself.

Call this number for more information on the proposed prescription drug substitution legislation. If you're already convinced, tell us so when you call. We'll send a protest in your name to the people in Oklahoma City who can stop this disaster before it starts — your state legislators. Or write them yourself. But don't wait. Write your letters today or

Call 273-8838 Now!

The alternative is enough to make you sick.

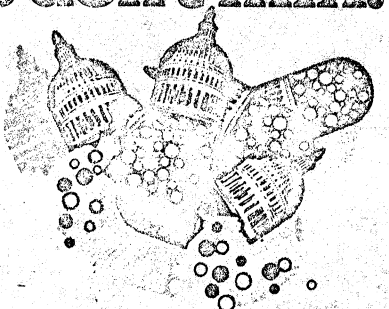
Medicine and Politics don't mix.

This campaign to Stop Prescription Drug Substitution Legislation is sponsored by the Pottawatomie County Medical Society and Pharmaceutical Association. *Gordon Richardt, Jr. RPh., Francis A. Davis, M.D., Co Chairmen*

Published in the *News-Star*, Shawnee, Oklahoma, and the *Evening News*, Ada, Oklahoma.

WARNING:

Medicine and Politics don't mix.



The citizens of eleven states now have doctors who are playing Russian roulette with their patients.

It's not the fault of the doctors. It's the law.

In eleven states, the politicians have passed laws that sound good, but they're bad medicine if you look beyond the promises.

They're called generic drug laws, or prescription drug substitution laws. And they *promise* lower prices for your prescription drugs.

In practice, what they do is to prevent you from getting what your doctor wants you to have.

And no one has yet proved that they can save you a nickel; in one area with such a law, a recent study shows that prescription prices went *up* an average of 19¢ on the dollar after the law was passed.

Generally, the laws say that a doctor must write the generic (chemical) name of any drug he wants you to have. (*Not* the brand name, in spite of the fact that the doctor wants you to have a specific brand because he knows and trusts it.)

So you may wind up with medicine made by some fly-by-night company that appears to be chemically equal but isn't.

And that's the catch that can kill you:

The same politicians who say — with a law — that all generic drugs are "equal" do not require competing drug companies to *make* them equal.

These so-called generic drugs are not compounded identically, and they're not manufactured identically.

And every doctor can tell you horror stories about patients who found that out the hard way:



Heart medicine that was supposed to be equal to the original but *wasn't* has *caused* heart attacks and killed the patients who took it.

And in an incredible case in California, a patient had to have 7 inches of his intestine removed because 150 "equal" antacid tablets failed to dissolve in his stomach and obstructed it.

Ask any doctor. Ask *your* doctor if all generic drugs are the same.

Then call this number for more information. If you're already convinced, tell us. We'll send a protest in your name to the people in Oklahoma City who can stop this disaster — your state legislators, or you can write them yourself . . . but don't wait, now is the time to take action. Write your own letter or

Call 273-8838 Now!

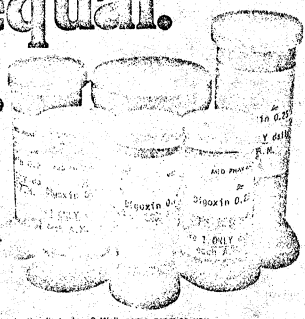
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Published in the *News-Star*, Shawnee, Oklahoma, and the *Evening News*, Ada, Oklahoma.

New York heart patients proved that all these generic drugs were not equal. They died.



Soon, Oklahoma law may give you the chance to test the equality of generic medicines for yourself.

The heart medicine was all seemingly the same. It had the same chemical compound as the effective prototype drug.

But in spite of that, patients reacted violently to the slight differences. They suffered heart attacks. They died.

And now everyone's willing to admit that, yes, this particular medicine does differ radically from manufacturer to manufacturer even though it's chemically the same.

But for those patients in New York, the admission came too late.

It's *not* too late for us here in Oklahoma . . . yet. A preposterous law has been proposed that would mean your doctor may not be certain that the drug he knows and trusts and which he has prescribed for you will in fact be the drug you *get* when your prescription is filled.

But it's not law yet. And it shouldn't be.

We've already seen one tragic instance where all generic drugs were not equal. Ask your doctor. There are others.

Like the man in California who found that all generically equal antacid tablets weren't the same . . . after 150 of them didn't dissolve in his stomach and he had to have them surgically removed, along with 7 inches of his intestine!

The fact is, the government doesn't even require that all generic drugs be equal. They need only meet minimum standards to be on the market.

How could anyone even propose prescription drug substitution in the first place? Well, some promise you that you'll save money if your prescriptions are filled by generic, rather than brand, name.

Well, a promise is all it is.

No supporter of prescription drug substitution has yet proved that this law will save you more than about 2¢ on the dollar. And we must ask, in all sincerity, what good is saving money on medicine if the medicine doesn't save your life?

As we said, there's no law that says we have to have prescription drug substitution . . . yet. But unless you act now . . . and urge others to act, too . . . we're really gonna get it. Really.

Call this number for more information. If you're already convinced, tell us when you call. We'll send a protest in your name to the people in Oklahoma City who can prevent this disaster — your state legislators, or write them yourself . . . but don't wait. *Now* is the time to take action. Write your own letter or

Call 332-4116 Now!

The alternative is enough to make you sick.

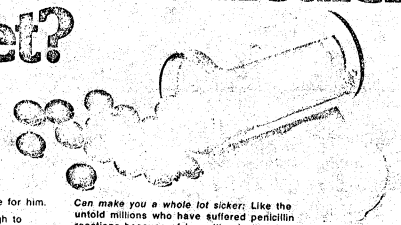


Medicine and Politics don't mix.

This campaign to Stop Prescription Drug Substitution Legislation is sponsored by the Pontotoc Medical Society and the Ada Pharmacists Association.

Published in the *News-Star*, Shawnee, Oklahoma, and the *Evening News*, Ada, Oklahoma.

When you're critically ill do you want a politician or your doctor to decide what medicine you get?



You trust a politician enough to vote for him.

Does that mean you trust him enough to dictate the medicine you and your family should take?

When he decided that's what it means, it's time to get worried.

Like now.

Some of them have decided they know enough to severely limit your doctor's options as to what medicine he gives you.

No kidding.

The proposed law involves prescription drug substitution. The selling point is that generic (or chemical) name drugs are usually cheaper than brand name medication.

And since all drugs with the same chemical name are *not* the same therapeutically, why shouldn't your *doctor* be the one to prescribe the medicine he wants you to get and have the assurance that you'll get it? Generic drugs are not required to meet equal quality control standards for purity or processing, but only minimum government levels.

We're here to tell you. And if you value your health and your money, we suggest you read on.

"Chemically equal" *does not*, REPEAT *NOT*, mean "therapeutically equal". Therapeutic action refers to the way the drug acts in your body. Differences in quality control, fillers, acidity and absorption rates will cause the so called chemically equal drugs to act differently in your body.

And the inequalities that exist

Can kill you: **Like the heart patients in New York who died because different batches of the same heart medicine *caused* heart attacks.**

Can maim you: **Like the man in California who had 7 inches of his intestines and 150 antacid tablets removed surgically because they *wouldn't dissolve* in his stomach.**

Can make you a whole lot sicker: **Like the untold millions who have suffered penicillin reactions because of impurities in the drug.**

Second, the drugs *won't* be cheaper. Despite grandiose claims nobody's proved that prescription drug substitution can save you more than 2¢ on the dollar on your medicine.

But if the cheaper drugs don't work, and you have to pay for repeated visits to the doctor, plus additional medicine, have you really saved any money at all?

You sent some of those people to Oklahoma City to represent you. Now it's time to give them a hand.

Call this number for more information on the prescription drug substitution legislation now pending. If you're already convinced, tell us when you call. We'll send a protest in your name to your State Senator and State Representative, or, you can write them yourself. But don't wait. *Now* is the time to take action. Write your letters or

Call 332-4116 Now!



The alternative is enough to make you sick.

Medicine and Politics don't mix.

This campaign to Stop Prescription Drug Substitution Legislation is sponsored by the Pontotoc Medical Society and the Ada Pharmacists Association.

Radio & Television

Radio Spot #1

Announcer:
You trust a politician enough to vote for him. Does that mean you trust him enough to dictate the conditions your pharmacist must follow in filling prescriptions? When he decides that's what his legislative position means, it's time to get worried. Like now. In eleven states they've already passed drug substitution laws that mean patients can't be sure they'll get the exact medicine their doctors prescribe. They may wind up with a similar but cheaper drug. That'd be fine if all the medicine was equal. But it's not. And the differences can make you a whole lot sicker than you already are . . . it has happened elsewhere, and it *can* happen here. If you don't act now, it's going to *happen*. Right here in Oklahoma. You sent people to Oklahoma City to represent you in state government. On *this* issue you had better let your legislators know how you feel. Call (Shawnee: 273-8838) (Ada: 332-4116) and voice a protest against prescription drug substitution. Do your part . . . medicine and politics don't mix.

Radio Spot #2

Announcer:
Oklahoma, it's time to get worried. In eleven of our sister states, the politicians have decided they're as qualified as your doctor is to prescribe your medicine. So they've passed laws — drug substitution laws — that fly in the face of everything a doctor has spent years learning. If their law passes, all the doctor would be able to do would be to indicate a *general type* of medicine . . . not the specific brand that he knows and trusts. That'd be fine if all the medicine was equal. But it's not. And the differences can kill you, can maim you, can make you a whole lot sicker than you already are. In documented instances it already has. Oklahoma, it's time to get worried 'cause the first step in such a program has passed the House and is pending in the Senate. Stop pending drug substitution laws now. Call (Shawnee: 273-8838) (Ada: 332-4116), get the facts and voice a protest, because medicine and politics don't mix.

Broadcast over radio stations KGFF, Shawnee, Oklahoma, and KTEN, KEOR, and KADA, Ada, Oklahoma.

Television Spot #1

Video: Three tablets of Digoxin displayed on counter, hands and shells come down to cover pills and shell game begins.

Audio: Some politicians say these drugs are generically alike.

Video: Shell game continues.

Audio: Actually, because of the quality of their processing — or lack of it — one of these tablets may kill you, one may cure you and one may do nothing at all.

Video: Shell game stops and shells are raised to look at tablets.

Audio: Can you tell the difference? Your doctor can, if you let him. But he can't if drug substitution becomes law like it already has in some states.

Video: Fade to black.

Video: Superimpose in white, upper third of screen:

Video: Don't let Drug Substitution become Oklahoma Law.

Video: Superimpose phone number, center screen.

Audio: Don't let it happen here. Call us now, (Ada 332-4116) (Shawnee 273-8838) we'll give you the facts.

Video: Fade out upper third copy, leaving phone number. Fade in logo upper third. Superimpose: Medicine and politics don't mix.

Audio: Medicine and politics don't mix.

Video: Fade to black.

Television Spot #2

Video: Extreme close-up of drug boxes lined up. First one has been tipped and they fall domino style, in slow motion.

Audio: Some politicians said these drugs were generically the same. Some New York heart patients proved otherwise.

Video: Last box falls.

Audio: They died.

Video: Dissolve to announcer seated on corner of desk where boxes lay. Picks up last box as he speaks.

Audio: Prescription drug substitution supposedly gives you the same drugs for less money. Sounds good. Chemically the same yes, but therapeutically the way the body reacts to it
. . . *some* difference!

Video: Superimpose this copy in white, upper third of screen.

Audio: Don't let Drug Substitution become law here.

Video: Superimpose phone number, center screen.

Audio: Call (Ada #332-4116) (Shawnee #273-8838) now. We'll give you the facts.

Video: Fade out upper third copy, leaving phone number. Fade in logotype upper third, "Medicine and politics don't mix." Lower third.

Audio: Medicine and politics don't mix.

Video: Fade to black.

Television Spot #3

Video: Extreme closeup of large unmounted, cut diamond and piece of coal same size.

Audio: A diamond. A chunk of coal. Both pure carbon . . . chemically equal . . . but therapeutically different . . . and *some difference!* — WOW!

Video: Dissolve to announcer seated on corner of desk where diamond and coal are.

Audio: Chemically equal medicines may be just as dramatically different therapeutically . . . some *difference!* But you won't see it . . . you'll just *feel* it . . . *if* prescription drug substitution becomes law in this state.

Video: Fade to black.

Video: Superimpose in upper third of screen: Don't Let Drug Substitution Become Law Here. Superimpose phone number center screen.

Audio: Don't let it happen. Call this number now, get the facts and then oppose any form of drug substitution legislation — LOUDLY —

Video: Fade out upper third copy leaving phone number. Fade in logotype upper third.

Audio: because medicine and politics don't mix.

Video: Superimpose: Medicine and politics don't mix. Lower third.

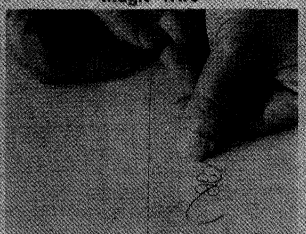
Video: Fade to black.

Broadcast over television station KTEN, Ada, Oklahoma.

VOL. [illegible] JANUARY-FEBRUARY 1976

Clinical Trends in CARDIOLOGY

'Magic' Wire



Straight wire (left) made of new space alloy turns itself into a clover-leaf blood clot filter when introduced into vena cava through angiographic catheter. For story, see page 6.

Outpatient Therapy Seen Possible

Self-Injected Heparin Reported Effective Against Thrombophlebitis

SELF-ADMINISTERED heparin provides effective outpatient therapy for persons with acute or subacute thrombophlebitis, Dr. Richard M. Stillman told the American Heart Association meeting in Anaheim, Calif.

Dr. Stillman reported on 407 patients treated over the past 10 years at the State University of New York Downstate Medical Center in Brooklyn. "Our protocol induced symptomatic resolution in under two months in half of those patients with acute or subacute conditions, and in under six months in 78 percent," he said.

Within those two categories, Dr. Stillman added, patients are excluded who have pulmonary involvement or whose condition is so severe as to confine them to bed; hospitalization and intravenous heparin would be better in their case.

The first step in applying the Downstate regimen, which was developed by Dr. Philip Sawyer, chief of vascular surgical services there, is to classify patients according to the status of their disease: acute, subacute or chronic. Those who present with markedly swollen calf and a positive Homan's sign are considered to have acute thrombophlebitis and, in the absence of fever and leukocytosis, are put on the outpatient protocol immediately.

Subacute thrombophlebitis is indicated by aching, tender, somewhat swollen calves and a negative Homan's

Continued on Page 4

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Arrhythmia Prevention Called Critical

Deceptive Signs, Altered Drug Responses Held Common Pitfalls In Managing MI in Elderly Patient

The detection and treatment of myocardial infarction in elderly patients present the physician with first-rate clinical challenges.

In an exclusive interview, Dr. Raymond Harris, chief of cardiology at St. Peter's Hospital, Albany, N.Y., clinical associate professor of medicine at Albany Medical College, and president of the Center for the Study of Aging, drawing upon his extensive experience with elderly patients, suggests approaches to differential diagnosis and effective management.



Dr. Harris

Q — Dr. Harris, in your experience, what are the main differences between the geriatric patient and younger persons so far as myocardial infarction is concerned?

A — Statistically, cardiovascular disease is more than twice as prevalent in people over 65 as it is in younger persons. Of course, there are some people under 65 who are physiologically much older and therefore subject to the same risks as the geriatric patient.

The aging process usually means decreased cardiac function, increased collagen in valves and endocardium, apparent shrinkage and loss of muscle fibers, focal hypertrophy of individual fibers, calcification of the media, and the likely presence of additional chronic disease including brain changes. Because of this, symptoms in the elderly person may be misleading, attenuated or simply absent, making the diagnosis of MI more difficult than in younger patients and causing many cases to go undetected in the older population.

Complications from MI may be more serious if the elderly patient survives the attack, because his vascular system may already be compromised and his body defenses cannot fight back as effectively.

Q — What, specifically, makes the diagnosis more difficult?

A — Often the symptomatology differs from the classic clinical presentation. Instead of an alarming chest pain radiating into the arm or the back, there may be little if any pain. Sometimes the older patient may report a sensation typical of gastrointestinal discomfort. So-called "silent infarction" is quite common — occurring in anywhere from one third to almost two thirds of these patients. Also, the ECG may be ambiguous when not downright misleading.

Q — How, then, do you approach the diagnosis?

A — When taking the history or examining the patient, the physician should look

Continued on Page 7

Rx for Acute Type III

Aortic Dissection Found Arrested by Drug Regimen

EDINBURGH, SCOTLAND — Depression of myocardial contractility, combined with a reduction in systolic blood pressure, is the most effective way to treat and control Type III acute aortic dissection, according to Dr. Richard J. Cleveland, chairman of surgery at Tufts University School of Medicine in Boston.

Reporting on the results of animal experiments, Dr. Cleveland recommended the use of trimethaphan to produce the necessary hypotension, noting that the effect of this drug "may be the critical factor in limiting dissection."

Implications Assessed

The implications of the study, he explained, are that once initiated, aortic dissection will progress in the face of a significant depression of myocardial contractility alone, but can be halted by a combination of depressed myocardial contractility and moderate hypotension.

In addition, Dr. Cleveland observed in his report to the recent meeting here of the International Cardio-Vascular Society, the fact that trimethaphan has been found to slow myocardial contractility to the same levels obtained with very large doses of propranolol indicates that trimethaphan may be the more effective drug for control of acute aortic dissection.

Experimental Evidence Given

In the Boston experiments, he said, 30 mongrel dogs underwent left thoracotomy and construction of a standardized intimal tear in the proximal descending aorta. They were divided into three groups – 10 untreated controls and 10 each treated with propranolol or with trimethaphan before development of the dissecting aneurysms.

When the control animals were sacrificed after an hour, nine showed a progression of aortic dissection by an average of 81.5 percent of the distance from incision to celiac axis. One animal showed no progression, and in two cases the dissection covered only 50 and 65 percent of the distance. No statistically significant change had occurred in myocardial contractility, Dr. Cleveland noted, but ascending aortic systolic pressure in these animals had decreased from a mean of 174 mmHg to 140 mm Hg during the observation period.

In the propranolol-treated group, he continued, nine animals showed aortic dissection over a mean of 65 percent of the distance to the celiac axis. In four dogs, dissection had

Continued on Page 6

* Important updated dosage information: LANOXIN® (digoxin) The recommended dosage has now been reduced.

Lanoxin is the *original* digoxin . . . the result of a research breakthrough by Burroughs Wellcome Co. over 40 years ago. It has been setting the standards ever since.

Through the years, Lanoxin has been distinguished by a reputation for consistent effect. This is clinically significant because digoxin preparations which have the same digoxin content have differed greatly in bioavailability[1] and consequent pharmacological effect.

As a result of recent pharmacokinetic studies, often supported by Burroughs Wellcome Co., new lower dosage recommendations have been adopted. They are summarized in our revised prescribing information which appears on the following page. We urge you to read this important updated information.

1. Peetbisz JJ, Butler VP, Lindenbaum J: Digoxin tablet bioavailability: single-dose and steady-state assessment. *Ann Intern Med* 81: 469, 1974.

LANOXIN® TABLETS
0.125 mg (yellow)
0.25 mg, scored (white)
0.5 mg, scored (green)

73-617 O - 76 - 16

Important updated prescribing information LANOXIN®(digoxin) Tablets

OUR LABELING HAS CHANGED, BUT NOT THE PRODUCT
Recent pharmacokinetic studies of digoxin using radioimmunoassay suggest that serum levels within the therapeutic range (1 to 2 ng/ml) can be achieved with doses lower than those previously recommended.
As the discoverer of digoxin and the leading manufacturer of this product, B.W. Co.® has accordingly modified the dosage recommendation for Lanoxin brand Digoxin, and has expanded the information concerning precautions, adverse reactions, warnings, and contraindications. Please note: *the product has not changed.* Below you will find brief prescribing information. You may obtain complete prescribing literature from your Burroughs Wellcome Representative or by writing to Department PML.

Brief Summary of Prescribing Information: Lanoxin® (digoxin)
Contraindications: The presence of toxic effects induced by any digitalis preparation is a contraindication to all of the cardiac glycosides.
Allergy, though rare, does occur. It may not extend to all preparations and another may be tried.
Vehtricular fibrillation is generally a contraindication.
Warning: Dosage must be carefully titrated. Monitoring of ECG and serum digoxin levels may be necessary to avoid intoxication.
Many arrhythmias for which digitalis is advised are identical with those reflecting digitalis intoxication. If the possibility of digitalis intoxication cannot be excluded, cardiac glycosides should be temporarily withheld if permitted by the clinical situation.
Patients with congestive heart failure may complain of nausea and vomiting which may also be indications of digitalis intoxication. Clinical determination of the cause of these symptoms must be attempted before further drug administration.
Patients with renal insufficiency are apt to be unusually sensitive to digoxin.
The use of digitalis glycosides in the treatment of obesity is dangerous.
Precautions: Atrial arrhythmias associated with hypermetabolic and febrile states are particularly resistant to digitalis treatment. Care must be taken to avoid digitalis toxicity if digoxin is used to help control the arrhythmia.
Potassium depletion sensitizes the myocardium to digitalis and toxicity is apt to develop even with usual dosage. Potassium wastage may result from diuretics, corticosteroids, hemodialysis, malnutrition, old age, and long-standing congestive heart failure.
Acute myocardial infarction, severe pulmonary disease, or far advanced heart failure often are accompanied by a greater sensitivity to digitalis and its disturbances of rhythm.
Calcium may produce serious arrhythmias in digitalized patients.
With myxedema, digitalis requirements are less.
Incomplete AV block, especially in patients subject to Stokes-Adams attacks, may develop to advanced or complete heart block when cardiac glycosides are given.
Chronic constrictive pericarditis is apt to respond unfavorably to digitalis.
Idiopathic hypertrophic subaortic stenosis must be managed with great care.
Renal insufficiency delays the excretion of digitalis, and dosage must be adjusted accordingly in patients with renal disease.
Electrical conversion of arrhythmias may require adjustment of digitalis dosage.
Congestive failure accompanying acute glomerulonephritis requires extreme care in digitalization.
Rheumatic carditis, especially when severe, causes sensitivity to digitalis. If heart failure develops, digitalization may be tried with caution.
A digitalis preparation must be used with caution in cases of ventricular tachycardia, in which case it is indicated only if congestive heart failure is a contributing factor.
If the patient has been given digoxin during the previous week, or any other less rapidly excreted drug of the digitalis group during the previous two weeks, the dose of Lanoxin brand Digoxin must be reduced accordingly. Because of impaired renal function and excretion in elderly patients, they frequently require lower than recommended doses.
Dosage of digoxin must be carefully titrated and differences in the bioavailability of parenteral preparations, elixirs and tablets taken into account when changing patients from one preparation to another.
Adverse Reactions:
Allergic reactions.
Gynecomastia.
Overdose or toxic effects in children.
The toxic signs differ from the adult in a number of respects. Vomiting and diarrhea, neurologic, and ophthalmological disturbances are rare as initial signs. Cardiac arrhythmias are the more reliable and frequent signs of toxicity.
Premonitory signs of toxicity in the newborn are undue slowing of the sinus rate, sinoatrial arrest, and prolongation of the PR interval.
Overdose or toxic effects in adults.
Anorexia, nausea, vomiting, and diarrhea are the most common early symptoms of overdose in the adult. Uncontrolled heart failure may also produce such symptoms.
Headache, weakness, apathy, and visual disturbances (blurred vision, yellow vision) can be signs of digitalis toxicity in adults.
Ventricular premature ectopic activity is the most common arrhythmia, except in infants and young children where nodal and atrial arrhythmias are more common.
Excessive slowing of the pulse is a clinical sign of digitalis overdose.
Atrioventricular block of increasing degree may proceed to complete heart block.
NOTE: The ECG is fundamental in determining the presence and nature of these toxic disturbances. Digitalis may also induce other changes (as of the ST segment), but these provide no measure of the degree of digitalization.
Treatment of Toxic Effects Produced by Overdose: Digitalis is discontinued until after all signs of toxicity are abolished.
Potassium chloride may be given in divided doses totaling 4 to 6 g for adults (in children, 1 to 1.5 mEq per kg), provided renal function is adequate. ECG monitoring is indicated to avoid potassium toxicity, e.g., peaking of T waves.
Caution: Potassium should not be used and may be dangerous in severe or complete heart block due to digitalis and not related to any tachycardia.
Other agents which have been used in treatment of digitalis intoxication include lidocaine, quinidine, procainamide, and beta-adrenergic blocking agents.

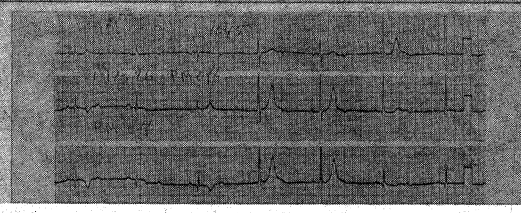
Dosage and Administration:
Recommended doses are practical average figures which may require considerable modification as dictated by individual sensitivity or associated conditions. Diminished renal function is the most important factor which requires modification of recommended or average doses. Clearance of digoxin from the serum is proportional to the glomerular filtration rate as estimated by renal creatinine clearance. The excretion of digoxin thus varies directly with creatinine clearance, and the ratio of the two clearances is near unity. As a result, an estimate of creatinine clearance gives a reasonable value for digoxin clearance, and a dose of digoxin may be reduced in proportion to the observed creatinine clearance.
Children 2 to 10 Years of Age.
The total dose of Lanoxin brand Digoxin for complete digitalization of children from 2 to 10 years of age is 40 to 60 µg per kg body weight. Initially, ¼ to ½ of the total digitalizing dose is given, followed by ¼ of the total dose at 6-hour intervals until digitalization is completed. Maintenance therapy is 20 to 30 percent of the digitalizing dose administered each day.
Children 10 Years of Age and Over.
Children 10 years of age and over require adult proportions by weight. Maintenance therapy is 20 to 30 percent of the digitalizing dose administered each day.
Adults.
The average digitalizing dose (i.e., amount accumulated) with digoxin tablets is 1.0 to 1.5 mg. A loading dose of 0.5 to 0.75 mg orally usually produces a detectable effect in 1 to 2 hours, which becomes maximal in 6 to 8 hours. Additional doses of 0.25 to 0.50 mg may be given cautiously at 6- to 8-hour intervals to full digitalization.
The usual daily oral maintenance dose is 0.125 to 0.50 mg (usually 0.25 mg). In elderly patients, 0.125 to 0.25 mg should be considered the average maintenance dose. In the presence of renal failure, the daily amount administered must be decreased by an amount proportional to renal impairment, in order to achieve therapeutic levels.

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

What's the Diagnosis?

THIS ELECTROCARDIOGRAPHIC CHALLENGE IS PRESENTED BY DR. SANFORD LEFF, FELLOW IN CARDIOLOGY, THE ROOSEVELT HOSPITAL, NEW YORK CITY.

A 71-YEAR-OLD man was admitted with a 24-hour history of dizziness, nausea, and precordial pressure. The ECG, (right), showed a sinus rhythm with 2:1 A-V block. The P-R interval of the conducted sinus beats is prolonged. There is no intraventricular conduction abnormality. Atropine 1 mg IV produced 1:1 AV conduction. Note Q waves with T wave inversions in the inferior leads, tall R waves in V_1 and V_2, and what appear as "hyperacute" tall, peaked T waves in the anterior precordial leads. For Dr. Leff's diagnosis, see page 7.



Heparin Injected by Thrombophlebitics Into Abdominal Fat Pad—20,000 Units Every Day

Continued from Page 1

sign. These patients are first tried on 1.2 to 2.4 grams of enteric-coated aspirin daily, along with mechanical measures, but are switched to outpatient heparin if they do not respond within two weeks.

The third group, chronic thrombophlebitics, have long-standing calf swelling, ulceration, changes in pigmentation and minimal pain. The physicians at Downstate now treat specific complications with surgery and apply the outpatient heparin regimen only when the chronic condition flares up and becomes acute. During the first years of their study, they found that the heparin treatment was ineffective against chronic manifestations.

Dose Reduced in Some Patients

Individuals assigned to outpatient therapy are trained to inject heparin — usually 20,000 units a day — subcutaneously into the abdominal fat pad. "We use this dose in all our patients except those who are very old and frail and those weighing under 100 pounds," Dr. Stillman told CLINICAL TRENDS IN CARDIOLOGY. "For them, we reduce it to 10,000 units daily."

The patients are also advised to use means to keep their feet elevated while in bed, to swim or to walk at least a mile a day if their condition permits, to take hydrotherapy — or at least a warm bath — daily, and to wear elastic stockings or elastic leg wraps. Extended bed rest is not prescribed, the Brooklyn surgeon emphasized, but the patients are told to avoid prolonged standing and excessive exposure to sunlight, which is believed to have a thrombogenic effect by causing a release of serotonin.

The regimen is continued until the patients show either a clotting time of over 20 minutes and symptomatic improvement, or a clotting time of over 30 minutes without other evidence of improvement. At this point, Dr. Stillman and his colleagues begin tapering the heparin dose.

"At the beginning of treatment we're on an every-day schedule," Dr. Stillman told CLINICAL TRENDS IN CARDIOLOGY. "Then we taper it to administration six days a week — usually eliminating the Sunday injection — then five days a week, then every other day. After that, the heparin injections are discontinued and the patients put on enteric-coated aspirin."

Simply reducing daily dosages during the tapering period is not feasible because the injections are prepackaged, he noted. "Also, we've found that the effect of heparin seems to last for two or three days anyway when it's given subcutaneously.



Dr. Stillman

"Furthermore, there's a psychological advantage doing it this way, a positive reinforcement. The patients know that when they start getting better there will be one day of 'vacation' a week when they don't have to stick that needle in themselves, and then two days, three days, and finally four."

The Downstate clinicians see their patients weekly until clotting times become stable; then once a month. Once the patients are switched to aspirin, they are seen twice at three-month intervals, and then at one and two years for checkups.

Pulmonary Symptoms Watched

"Of course, if the patient complains of any pulmonary symptoms during treatment, even the slightest, he's given an immediate chest examination, ECG and chest x-ray, and if there's any reason to suspect pulmonary embolus, a lung scan," Dr. Stillman said.

The incidence of complications caused by the heparin regimen was very small in the Downstate series, he reported, with only seven of the 407 patients developing clinically documented pulmonary emboli — none of them fatal — at any time during or after therapy. Abnormal bleeding during therapy occurred in nine of the thrombophlebitics, with all episodes being minor and easily resolved. They included epistaxis, menorrhagia, joint hemarthrosis, rectal bleeding and one case of retroperitoneal hematoma.

"A bleeding episode once controlled is not considered a contraindication to reintroducing heparin therapy while closely monitoring clotting studies and hematocrit," Dr. Stillman stressed. And as for other possible heparin side effects, the Brooklyn physican noted one case of reversible alopecia and several febrile or urticarial reactions, but no osteoporosis, thrombocytopenia or anaphylaxis.

Excluding two anomalous patients, one of whom has received heparin on an outpatient basis for the past 10 years, and suffers confirmed recurrence of thrombophlebitis whenever therapy is halted, the maximum period of treatment has been about three years, Dr. Stillman said.

'No Intravenous Therapy'

"The important thing," he continued, "is that the most common type of thrombophlebitic — one who presents with acute pain in one leg — can be treated outside the hospital, without intravenous therapy, and without coumadin — which seems to us to be more dangerous than heparin. On the basis of our experience, we can be 78 percent sure that within six months of this therapy, the patient will show improvement both symptomatically and on strain-gauge plethysmography."

This does not mean that the vascular system returns to its prethrombophlebitic condition, he added. "Our plethysmographic studies seem to show that all these patients have some persistent abnormalities, usually a decrease in venous filling time."

Recurrences of acute thrombophlebitis have not been common, however. Almost three quarters of the outpatients on the heparin regimen had no recurrence.

As an added benefit, chronic progressive venous insufficiency following acute thrombophlebitis was almost totally absent in the Downstate patient group. "We have not seen the chronically swollen thrombophlebitic limb we've observed in outpatients treated by other techniques," he concluded.

Managing MI in Aged Hold Special Problem

Continued from Page 1

for atypical as well as classic signs. Moreover, because the older patient may be senile or unable to verbalize his pain in the same way as a younger patient, it is often helpful to question family members or others close to the patient for more information.

Q — What complaints might be equivalent to pain in such older patients?

A — Syncope or epileptiform attacks may be particularly significant and may occur without pain or other warning as the onset of myocardial infarction. Since the cardiac origin of such attacks may be difficult to recognize clinically, a cerebral origin could be mistakenly diagnosed, especially in patients who have suffered earlier cerebrovascular accidents. When I see an acutely confused elderly patient, I always suspect cerebral ischemia secondary to a cardiac disorder. And, of course, stroke is also a quite common presenting sign in this group of patients.

Q — Wouldn't the ECG show the cardiac origins of this condition?

A — Recognition of the cardiac source may be complicated by the fact that abnormal ECGs, suggestive of acute myocardial ischemia, are recorded in some instances of cerebrovascular accident, even in the absence of organic heart disease. Similarly, in the elderly patient with acute MI, a drop in blood pressure may result in signs of cerebrovascular insufficiency and ischemia. Also, the symptoms of basilar artery insufficiency could become aggravated, and the patient might complain of visual changes, deafness, vertigo, ataxia and visual hallucinations. Cerebral thrombosis might develop. But the fundamental cause may be an MI.

Q — What other symptoms of MI are common in the older patient?

A — Dyspnea and exacerbation of heart failure are very common symptoms of myocardial infarction in this group. Next in frequency come the classic signs of radiating chest pain. And, of course, arrhythmias of all kinds, including complete heart block, are more common than in younger groups. Shock is also among the presenting signs.

Q — What features do you look for in the ECG of the elderly patient?

A — Three main electrocardiographic patterns may be observed in elderly patients with acute myocardial infarction. These changes usually depend upon the amount of collateral circulation that has developed over the years, and upon the severity, extent and location of the MI.

The most classic and characteristic form, though not necessarily the most common in older people, is the one in which the injury

Continued on Page 6

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Warning: May be habit forming.

1. Riedel HH: AM J Med 29:473, 1960

[redacted] 'I Prefer the CCU for My MI Patients' [redacted]

Continued from Page 5

produces marked ST elevations within 24 hours and is followed by a flattening and inversion of the T waves and by development of Q or QS waves due to transmural necrosis of the myocardium. Very tall T waves may sometimes be first noted minutes to hours after the onset. This pattern is the usual one in younger people.

The second involves progressive inversion of the T waves with no QRS changes, indicating that complete transmural infarction has not occurred. Elevated ST segments may be recorded initially, followed by deeply inverted T waves. Symmetrical, deeply inverted T waves in one or more precordial leads may also indicate subendocardial infarction in the anterior or lateral wall of the left ventricle. Such deep inversion may last for weeks or months before the T waves gradually improve.

The third ECG pattern consists of progressive ST depressions, usually greater than 1 millimeter, and slight T wave inversions in the limb and precordial leads, suggesting subendocardial infarction.

In my experience, more elderly patients with MI fall into the latter two patterns.

Very often, an acute arrhythmia may be due to acute MI (see ECGs on page 7.)

Q – What ECG features might be ambiguous?

A – The PR interval is usually a little longer in the elderly. A 0.22-second interval may be perfectly normal in a 80-year-old, whereas in a 50-year-old it's a little prolonged at a heart rate of 100 or less. T waves may be low or flat in older people without really being abnormal.

Q – Why is this?

A – There may be fibrosis, for instance, and I don't think this would be indicative of reduced coronary blood flow per se. Also, a fair amount of coronary artery disease involving several arteries is generally present, whereas in younger people only one artery may be affected, and this, too, may influence the ECG tracing and its interpretation.

Q – Are the same areas of the heart affected in the elderly patient with MI as in the younger one?

A – I don't know of any evidence suggesting this. But the older person may have developed a certain amount of collateral circulation over the years, and occlusion in one artery may be mistaken on ECG for occlusion in another one. Postmortems show that you can't diagnose the exact location of the MI from the ECG in older patients because of this alteration.

Q – In confirming the diagnosis of MI in the hospital by enzyme determinations, are there any age-related factors that might influence the classic criteria?

A – In general, the same criteria can be applied to all patients, but it is interesting to note that in older individuals the amount of enzymes in the heart may be decreased. Studies have shown this to be true of lactic dehydrogenase in particular.

On the other hand, the clinician should guard against making a diagnosis of myocardial infarction because the CPK level is high in the absence of other enzyme changes. Simply giving the patient an intramuscular injection may produce a significant rise in CPK levels.

Q – What is the reason for the small enzyme rises?

A – First, a collateral circulation may have developed; also, the older patient's heart is generally smaller and its intrinsic amount of enzyme may be reduced. Of course, the changes will still be very great in the patient with an overwhelming infarction and extensive damage.

Q – Turning to treatment, what guidelines would you give for hospital management of the elderly patient?

A – The first question that comes up – and it's a controversial one – concerns where in the hospital the older MI patient should be treated. I prefer that my own MI patients – regardless of their age – be treated in the coronary care unit. The incidence of complications is high in older patients, and I feel CCU treatment is particularly important for them because of the monitoring facilities available there. One can't afford to wait to initiate treatment as one can, to some extent, in younger individuals in better physical condition.

Q – What concomitant conditions are most likely to complicate treatment?

A – As I have said, the incidence of arrythmias is very high in older people, and this makes the elderly MI patient a particularly poor risk. These arrhythmias should be detected early and treated promptly. Second, of course, is the problem of congestive heart failure, which must always be expected in such patients; again, it must be detected early and treated vigorously. And third, there is shock.

Q – What are your guidelines in administering medication to older MI patients?

A – The first thing to remember is that older people are likely to have a chronic insufficiency of the detoxifying organs, so they're more prone to develop idiosyncrasies toward various medications. Generally they should receive lower doses than younger persons.

Q – What do you usually give these patients for pain?

A – I use meperidine or morphine, although the elderly are more sensitive to these medications and should be watched closely. What might be considered a normal dose for a younger patient may lead to respiratory depression, hypotension or urinary retention in the elderly. Another point to remember is that if morphine is given alone, it may cause nausea and vomiting, adding another burden to an already injured myocardium. But severe pain usually can be relieved by injecting 50 mg meperidine – or more if it's found necessary – or 8 mg to 10 mg morphine with atropine.

Q – Do you recomend the use of oxygen?

A – It may be useful to relieve anoxia. However, in the older patient it should be given nasally instead of in an oxygen tent. A tent often alarms the older patient and causes him to become confused and disoriented.

Q – What measures do you advocate to control arrhythmias?

Prompt Action Held Crucial in Acute Aortic Dissection

Continued from Page 2

progressed the entire distance and, as in the control group, one showed no progression at all. The other five showed progressions of 58, 55, 54, 50 and 36 percent. There was no change in the animals' systolic pressure, but myocardial contractility had been significantly depressed in these dogs by the administration of propranolol prior to the procedure.

Trimethaphan Preferred

In contrast, said Dr. Cleveland, none of the trimethaphan-treated animals demonstrated progression of aortic dissection upon sacrifice. Their aortic systolic pressure, which initially averaged 185 mm Hg, dropped to less than 90 mmHg before the surgical procedure and remained there during the 60-minute observation period, while their myocardial contractility decreased from a control value of 1,275 mm Hg/sec. to 890 mm Hg/sec.

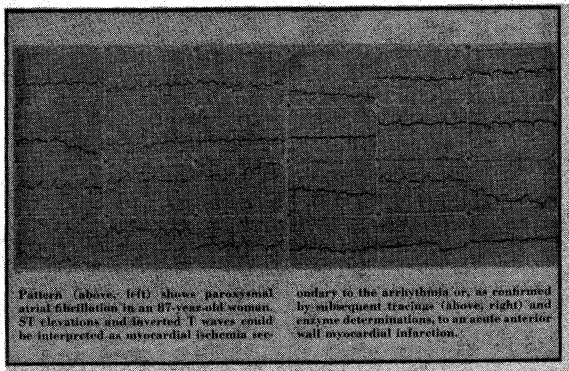
In a clinical situation, the Tufts researcher noted, the propranolol dosage used in his studies would be considered excessive and the significant depression of myocardial contractility observed in the dogs thus might not be duplicated. However, he added controlled hypotension to the 90 mmHg level is readily achieved in humans by the trimethaphan doses used in the experiments.

Associated with Dr. Cleveland in this investigation were Drs. Wilfred I. Carney Jr. and Harold F. Rheinlander. Their complete report is published in *Surgery* 78: 114-20, 1975.

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Pattern (above, left) shows paroxysmal atrial fibrillation in an 87-year-old woman. ST elevations and inverted T waves could be interpreted as myocardial ischemia secondary to the arrhythmia or, as confirmed by subsequent tracings (above, right) and enzyme determinations, to an acute anterior wall myocardial infarction.

A – For ventricular extrasystoles or ventricular tachycardias, procainamide can be given orally or intramuscularly, or lidocaine intravenously. Again, the doses should be relatively low. Excessive lidocaine or procainamide may be responsible for some of the acute confusion older people exhibit in the CCU.

If ventricular tachycardia or fibrillation develops despite these measures, emergency treatment – such as direct current countershock – can be most effective. In the event of paroxysmal ventricular tachycardia, electrical pacemaking may be the best means of control when pharmacologic methods fail. Digitalis is also indicated for some atrial arrhythmias, particularly if the patient is in heart failure.

Q – How do you manage congestive heart failure in the older patient?

A – Early signs and symptoms of congestive heart failure should be sought at each bedside visit, and therapy should be individualized and adjusted to the severity of the failure.

Q – Is sodium restriction helpful?

A – A diet that limits sodium to 1 gram or less will be beneficial to most elderly patients with acute MI. Salt restriction and diuretics should be started at the onset of pulmonary congestion. So long as the diuresis is not too rapid and electrolyte imbalance is avoided, the elderly patient usually responds well.

Q – If these measures fail, would you proceed to digitalize the patient?

A – Digitalization may indeed be helpful in treating congestive heart failure in the elderly. Their greater sensitivity to digitalis may dictate smaller doses, preferably orally, or perhaps intramuscularly, as well as observing the patient carefully for signs of toxicity. In patients with acute MI, it is best to avoid the IV route and to wait for 24 to 48 hours if possible. Otherwise, rapid administration of large doses of digitalis should be avoided, since this may provoke the onset of pulmonary edema and lead to digitalis toxicity and arrhythmias much earlier than in younger patients. It is possible to give half of an average digitalizing dose to an elderly patient and still obtain some useful inotropic effect.

Q – How do you treat shock in these patients, Dr. Harris?

A – In much the same way as in younger ones. Unfortunately, the prognosis for the older patient with MI and shock is particularly poor. Devices intended to assist the circulation may improve the outlook for younger patients, but are of less value in helping the geriatric patient with acute MI.

Q – What is your usual practice with regard to anticoagulants?

A – Ordinarily, I don't give anticoagulants to older MI patients who are otherwise good risks, unless they are already on anticoagulant therapy or if they have a history of pulmonary embolic phenomena or thrombophlebitis. Even in this second group, I prefer to ambulate the patients and prevent the complications that way.

Q – You mentioned ambulating your patients. Can this usually be done?

A – I don't believe the older patient has to be marooned in bed, especially following a small myocardial infarction. He can sit in his chair or walk around the room. This is important not only to reduce the risk of thromboembolism; older people have so much associated disease – arthritis or weakened lungs, for example – that when the physician confines them to bed, they deteriorate quickly.

Q – Do associated diseases change your treatment of the myocardial infarction?

A – Oh, yes. The real problem in treating these patients is that they have three strikes against them: the myocardial infarction, the associated diseases and the physiologic changes of the aging process itself. You might add a very important fourth factor – psychologic stress. The physician is really treating a variety of conditions.

Q – Does this lead to compromises in therapy?

A – Definitely. I've evolved what I call a goal-oriented system of managing elderly people generally. I have to find out what's more important to treat first.

Unquestionably the myocardial infarction is usually the life-threatening problem that requires priority. For example, many older people who come into the hospital with an orthopedic problem – a broken bone – may also have had an MI. In fact, the infarction may have been the reason the patient fell and broke the bone. When the MI is detected – and many of them aren't – treatment has to be directed against it first, naturally, and any orthopedic correction, which is usually aimed at preventing disability, has to wait until the MI has healed, usually three months after its onset.

Q – Does the reverse happen – another condition has to be handled before, or simultaneously with, the MI?

A – Yes. Arrythmias are actually the first priority in treatment. Or suppose you have a patient with myocardial infarction who develops a hemorrhage. The bleeding has to be treated concomitantly or the treatment for MI will fail. Giving the patient enough blood without throwing him into heart failure poses a judgment problem: How much blood do you give? How fast do you give it? These questions demand careful consideration for each patient. Shock and stroke are other conditions that must be treated immediately.

Q – What other factors have to be considered in the successful management of the geriatric MI patient?

A – I've mentioned psychologic stress. Its alleviation is especially important because older people are much less able to cope with it. Very often the infarction itself was triggered by some stressful condition.

In the coronary care unit, the stress factor increases. Older people are often frightened by all the machinery, oscilloscopes, ECG electrodes and so on surrounding them. The judicious use of tranquilizers and sedatives is therefore important, but even more so, I think, is an attitude of warm interest and reassurance on the part of the physician, nurse and staff. Indeed, this personalized support may be crucial to a successful outcome.

Answer to ECG Quiz

What's The Diagnosis?

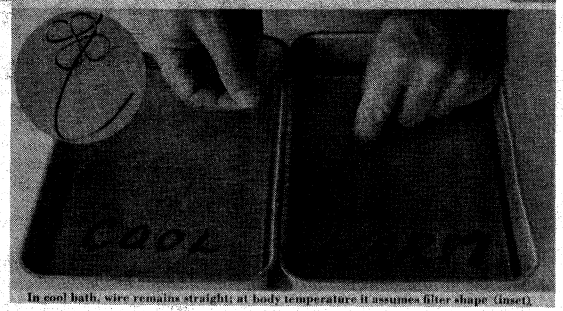
MOST LIKELY, it is patient has conduction delay above the bundle of His–Wenckebach type A-V block, 2-1. Inferior wall myocardial infarction is represented by the deep Q waves with T wave inversions, while the tall R waves in V_1 and V_2 indicate posterior wall myocardial infarction. The tall, peaked T waves in the anterior precordial leads suggest an acute ischemic process in the distribution of the left coronary artery. Subsequently, the patient developed anterior subendocardial infarction, manifested by low-grade enzyme elevations, S-T depressions and T wave inversions in V_3, V_4, and V_5.

Alloy With 'Memory' May Help Prevent Pulmonary Embolism

A SPACE-AGE METAL alloy capable of undergoing a programed metamorphosis may provide a simple means of preventing pulmonary embolism, according to a Boston radiologist. It would enter the vena cava in the form of a straight wire, he explained, and then, once inside, would automatically change its shape into that of a blood filter.

The new alloy – kept on the government's highly classified list until recently – "can exist in one or another of two predetermined forms, depending on its temperature," according to Dr. Morris Simon, director of clinical radiology at Beth Israel Hospital and professor of radiology at Harvard Medical school.

For its application as a blood filter, he told a recent Science Writers Forum held by the American Heart Association in Tucson, it is designed to exist outside the body as a fine, straight wire about 15-20 cm long. Upon reaching the vena cava through an angiographic catheter it responds to the body heat by twisting itself into a complicated shape previously imprinted in its "memory." The resulting filter, Dr. Simon added, is designed to lock itself into position at the desired level of the vena cava.



In cool bath, wire remains straight; at body temperature it assumes filter shape (inset).

A Simpler Approach

If the wire proves safe in humans – it has so far been tested successfully in dogs – it would represent a much simpler approach to preventing emboli from reaching the lungs than the traditional ligation or clipping of the vena cava or its obstruction with a Mobin-Uddin umbrella, he noted. The former procedure requires abdominal surgery, while the latter involves a cutdown in a jugular or femoral vein. "The 'memory' wire needs no other inlet than the angiographic catheter already introduced for diagnostic purposes," Dr. Simon said.

To prevent premature transformation during its passage through the catheter, the wire is bathed in a cool solution below its triggering temperature while it is being introduced. Once in place and in its filter form, it "effectively subdivides the cross section of the vena cava into a multitude of small openings that allow blood to pass through, but not emboli of any significant size," Dr. Simon observed. "Depending on its design, the openings can be about 2 to 4 mm in diameter."

The device would be used in patients in whom anticoagulants are precluded by the risk of internal bleeding – those who have suffered cerebral hemorrhages, undergone recent surgery, or sustained fractures or other injuries.

The underlying metallurgical breakthrough was achieved at the U.S. Naval Ordnance Laboratory as a result of work by Dr. William Buehler on nickel/titanium alloys (Nitinol) in the mid-1960's. The alloys were originally intended for use in space antennas. In addition to their unique shape memories, these alloys are nonmagnetic and are resistant to corrosion, oxidation and abrasion – all distinct advantages in biological applications.

In its straight-wire phase, the alloy used by Dr. Simon has to be kept at a temperature of about 50°F. Its transformation into the filter is completed at about 90°F, comfortably below body temperature.

The filter shape is programed into the wire by winding it onto a jig at the lower temperature and annealing it in a furnace at about 1,020°F. The wire is then cooled and straightened for introduction through the catheter.

"We have used clover leaf and spiral filter designs and are now moving on to a fine mesh produced by criss-crossing wire loops. The size of the mesh openings is only 2 mm square, small enough to block the passage of even tiny emboli," Dr. Simon reported.

Further animal experiments will be conducted to establish the alloy's efficiency, as well as its biologic effects on blood clotting, liver function and other bodily functions, he said. Tests on patients are foreseen in one to two years if the present research protocol continues to go well.

Collaborating with Dr. Simon on the metallurgical aspects of the filter is Professor Roy Kaplow of the Massachusetts Institute of Technology. His collaborators in clinical research are Drs. Edwin Salzman and David Freiman of Harvard.

"We believe," concluded Dr. Simon, "that the filter represents only the first of many in vivo applications of the remarkable thermal-memory alloys. There are other instruments of complex shape that could benefit from this method of easy insertion and emplacement, including devices for use in arteries, the heart, the bronchi, the skeleton, and the uterus."

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ISSUE HIGHLIGHTS

Managing Geriatric MI – Page 1

Self-Help in Thrombophlebitis – Page 1

Rx For Acute Aortic Dissection – Page 2

Litho U.S.A.

informative items for the **Doctor's Assistant**

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Volume 10 Number 5

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Office Management

The Patient's Medical Record and the Law

The legal status and importance of medical records is changing, according to physician/legal expert Harold L. Hirsh. Consider, for instance, that medical evidence now plays a role in about 75% of all civil cases and in about 25% of criminal cases brought to suit.

"Health care providers" such as private physicians or hospitals are responsible for keeping a patient's medical records confidential; failure to do so may constitute legal grounds for negligence or invasion of privacy. In the past, medical records were considered the possession of the private physician, the hospital, or clinic. Since the courts have ruled that a patient has the right to a full and frank disclosure of all facts related to his physical condition, this gives the patient exclusive control over information pertaining to his health. And, although the health care provider has a right to keep the original record, the patient has an absolute legal right to the information contained. He is therefore entitled to a copy.

Nurses, technicians, and orderlies whose work may be included in the patient's record also have a right to review records should they become "interested parties" in a legal case. In addition, insurance companies, attorneys, and health care providers have the right to inspect a patient's record with his permission.

In ruling on medical records, the court recognizes two types of ownership: 1) the traditional ownership of physical materials that comprise the actual records and 2) ownership of information recorded. Physical materials belong to the health care provider, but the information recorded belongs to the patient.

It's clear that a properly kept record may become the physician's legal witness, if need be. For this reason,

- Corrections, additions and deletions on medical charts and records should be made properly. Data should not be removed, tampered with or substituted once the record is made.
- All changes in a patient's records are best made in chronological sequence with adequate explanations regarding the reason for the change—again, without altering the original entry.
- There should be no attempt to improve legibility of the original entries.
- Records must be stored for the required statutory period.
- Poorly kept or inadequate records may be considered a breach of the accepted standard of medical care. This would be a factor in a malpractice case.

The patient is responsible for the cost of duplicating his medical records. Nonpayment of a medical bill is not a legal reason for failing to reproduce the record on request.

The court's decisions appear to be based on the legal principles that establish a patient's right to control his own body, his right to give informed consent to medical management, and his right to obtain complete medical information. The ultimate legal conclusion is that a patient has a "right of access" to his or her own records.

At the same time, the courts have established the *doctrine of professional discretion* regarding the preparation and dissemination of records. This means that a physician may decide to withhold part or all of the patient's record if he believes that the medical information will be detrimental to the patient. Reasons for any such refusal should be documented in writing.

Records are rightfully transferred to any physician who takes over the patient's care, in accordance with an AMA Judicial Council decree. *(Hirsh HL: J Fam Pract 2:213, 1975)*

New Computer in the Office

You'll surely have no problem in scheduling patients' appointments—or in billing—now that a minicomputer is being installed in your office. But B. Menkus, who understands the pitfalls, says there are several considerations. The first is whether your office computer will in fact do what's been promised by the salesman... then whether you can *afford* to have it work! Using a minicomputer requires more advance planning than most people realize. Furthermore, you'll probably need to be involved in the planning process more than you'd like. Then, various problems that your salesman cannot be expected to solve may develop only *after* the system is operating:

- **Data errors**—which often appear as billing mistakes. These are caused by minor typographical errors that occur in "feeding" information to the computer via typewriter. Because of their likelihood, data errors are "almost impossible to prevent and difficult to correct."
- **Employee salary escalation**—Successful computer operation requires a skilled operator familiar with the routine of your office. Trained individuals are hard to find—and once you've trained such a person, expect to see them tempted by a higher salary in some other office.

- **Inability to handle exceptions**—A standard data processing system designed to handle only routine situations is provided by the salesman. If a patient's bill is paid by her son-in-law who has a different name and address, the computer is befuddled unless it has been programmed (expensively) to deal with such unusual situations.

- **Unanticipated expenses**—Before the minicomputer can operate, your office may require expensive changes in electrical wiring, increased storage space, and even locked, specially insulated cabinets for tapes containing confidential data on patients.

On the other hand, using more complex computer systems, pioneering family-practice groups have fully computerized their patients' medical records with exciting results. Case histories are read from a TV-type screen. All records on file can be checked by computer to find out which patients need a "reminder" for a flu shot. The type of patient complaints reported can be periodically reviewed. You can rapidly obtain printouts of the patient's history for a consultation; complete the morning's billing in one hour; and even help to spot trouble-in-the-making by reviewing trends on a patient who has shown a steady rise in blood pressure over the past 6 months. *(Menkus B: Physician's Management 15[7]:59-GP, 1975; Patient Care 8[5]:68, 1974)*

Patient Relationships

When a Teenager Phones, It's Important!

So says George P. Rizzone, MD, pediatrician turned specialist in adolescent medicine. He finds that a phone call from an adolescent patient is rare unless something "very important is going on—either medically *or* psychologically." You may have observed this in your own office. At any rate, it's a good idea to give that adolescent priority. It's worth noting that Dr. Rizzone, a former pediatrician, now devotes his entire practice to adolescent medicine. Some pediatricians set aside one or two afternoons a week treating adolescents exclusively. Their assistants switch the Mickey Mouse/Bambi-type decorations and Sesame Street magazines to something more suitable for teenagers who are "too old" for the pediatrician—but still "too young" for the internist (ie, patients between the ages of 12 and 21). So the adolescent stage is an awkward one, even in medical practice!

How does adolescent medical care differ from pediatrics? Primarily you deal with the young patient, *not* the parent. For instance, on the initial visit, the patient and the parent may be asked to fill out separate confidential questionnaires.

You must try to gain the confidence and trust of difficult patients such as the withdrawn teenager with a sprained wrist who is secretly devastated by acne, awkward physical movements, and conflicts about emerging sexuality. That's why, besides treating the usual colds, sore throats, and sprains, Dr. Rizzone also counsels adolescent patients regarding their problems of sexual adjustment. Referral to a psychologist may be necessary. Another major responsibility of the specialist in adolescent medicine is family counseling, since the biggest problem of the adolescent may not be *medical* but rather the struggle between parent and teenager for control. This conflict becomes acute in a patient with a chronic disease such as diabetes; his parents may insist on supervising the administration of insulin, whereas he feels competent to care for himself. If parents are afraid to grant independence to the teenager with a chronic disease, both the parents and the patient may deny the severity of illness, to the detriment of the patient. The physician and his assistant are caught in the middle.

It's clear that the medical care of teenagers takes time and diplomacy, particularly because they mistrust adults. It takes people like you to help them feel secure while they cope with medical and psychological problems. *(Campion EW: Prism 3[8]:41, 1975)*

Talkies Rated 4-Star in Educating Patients

Machines that show films from behind the screen (so-called rear-projection devices) are helpful in educating a newly diagnosed heart patient, or perhaps a diabetic, about his disease. Teaching devices also make your work easier. During an office visit, the newly diagnosed patient is escorted to the console; you select and insert the appropriate film cartridge, seat the patient in front of the screen, press "on" and retreat until the film ends. He will then usually have additional questions for you or the doctor. Try to tie in the patient's *own* problems concretely to the points emphasized in the film. You must be familiar with the film's content as well as the patient's problems... In one survey of patient educational materials, a rear-projection system was rated most effective; next were written educational materials (such as flip charts) for "lecturing" a patient. And although booklets are the simplest, rated least effective are the booklets distributed around the waiting room. In contrast, those you hand *directly* to the patient are likely to be read. *(Neff J: Physician's Management 15[8]:35-D, 1975)*

Does Prenatal Education Really Make Childbirth Easier?

Many educational programs have been developed to improve the expectant mother's attitude toward childbirth and train her in the role she can play during labor. One of the best known, based on Pavlovian principles, was developed in Russia in 1945. It was introduced by Dr. Fernand Lamaze to Frenchwomen in 1951. The so-called Lamaze method, or variations of it, is widely taught in this country.

The Lamaze method is aimed at lessening the anxiety of the prospective mother by providing information that will correct any misunderstandings about childbirth. It tries to develop more positive attitudes toward pregnancy and thus, achieve a quicker, easier delivery. Since much of the anxiety surrounding childbirth

is thought to be related to the anticipation of pain, the expectant mother is taught how to relax so as to overcome physical tension that might produce pain. She is taught to concentrate on complex breathing techniques synchronized to changing uterine contractions. Thus, attention is shifted away from any pain associated with the contractions. Bodybuilding exercises are also recommended as a means of preparing for a comfortable delivery. Husbands are included in the program for both emotional and practical support. They become an important part of the labor and delivery process by being present in the delivery room.

Any anxiety-reducing effect of the Lamaze program could not be confirmed by psychologic testing. The Lamaze-trained primiparas and multiparas required less general anesthesia or nerve blocks than did multiparas who had not taken the course. Researchers conclude, therefore, that childbirth education courses such as the Lamaze program do have objective benefit. For more conclusive evidence, psychologic tests need to be targeted specifically toward the anxiety provoked by pregnancy and concerns about delivery, according to this group of psychologists.
(Zax M, et al: Am J-Obstet Gynecol 123:185, 1975)

Worth Noting

Tipster's Tips

Bone up on those medical terms—You may wish to consult the *Manual of History Taking, Physical Examinations and Record Keeping*, a text by E.E. Raus, Philadelphia, J.B. Lippincott Co., 1974. (588 pp, $18.00). This will introduce you to numerous medical terms, and better still, it provides a glossary of medical terms plus their lay equivalents. Procedures described require an understanding of normal and pathologic anatomy and physiology. This text has been recommended for physician's assistant programs.

To crush a tablet—Place the unopened packet containing the pill between the jaws of a hemostat. Clamp down several times until the pill is reduced to a fine powder. "Most of these packets are made of strong paper, and if care is taken, the patient will receive the entire dose."
(Smith FM: RN 38[7]:53, 1975)

About that Pap smear—Any sexually active woman should have Pap smears evaluated routinely; once a year is adequate for most women of childbearing age. However, cancer patients should be tested every 3 months during the first year, then every 6 months. Unless there are other risk factors, simply taking the pill or having an IUD does not necessitate semiannual examinations.
(Patient Care 9[11]:112, 1975)

Hold on to that slippery thermometer—Old enough for an oral thermometer ...too young to keep it in the mouth? Family physician, L.P. Lapin, takes the oral temperature of young children while they are lying down. If the thermometer does fall out, it drops only 2 or 3 inches to the examining table. What's more, children seem to keep a thermometer under the tongue more easily while lying down.
(Patient Care 9[5]:96, 1975)

Learning Systems for Physician's Assistants

The educational publishing division of Bobbs-Merrill (4300 West 62nd Street, Indianapolis, IN 46206) has published a series on Allied Health Occupations. These consist of softbound books with accompanying audio tapes, lab manuals and teachers guides (if needed) designed to provide the basics on 19 subjects such as medical office practice, medical records technology, physician's assistant techniques, patient-care techniques as well as the basics of anatomy, clinical chemistry and other important subjects. There are 19 core subjects; prices range from $4.95 to $7.95.

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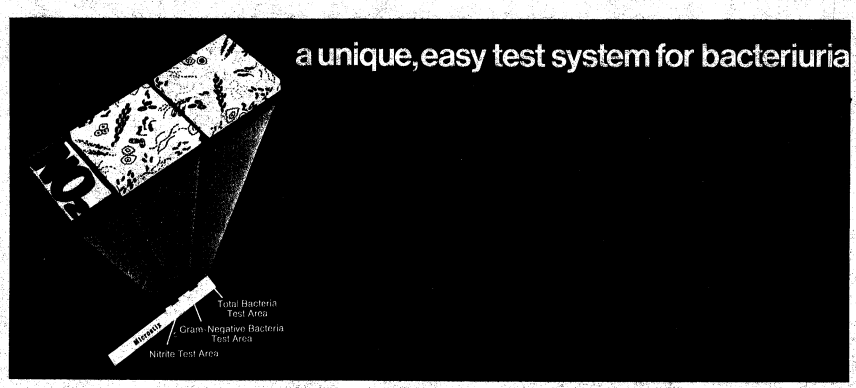
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tentacle-like microvilli. One researcher reports that those bristles disappear in patients with housewives' dermatitis. Cell formation becomes flat or misshapen after the hands are soaked in detergent. However, these cells seem to be normal in patients with psoriasis or eczema. A puzzling observation; soaking in water produces nearly the same destructive effect on the stratum corneum as detergent. It is believed that detergents exert a chemical effect 1) directly on keratin or 2) indirectly, by simply removing the normally protective oily film to expose the keratin. It seems that wearing rubber gloves for rinsing may be a good idea.

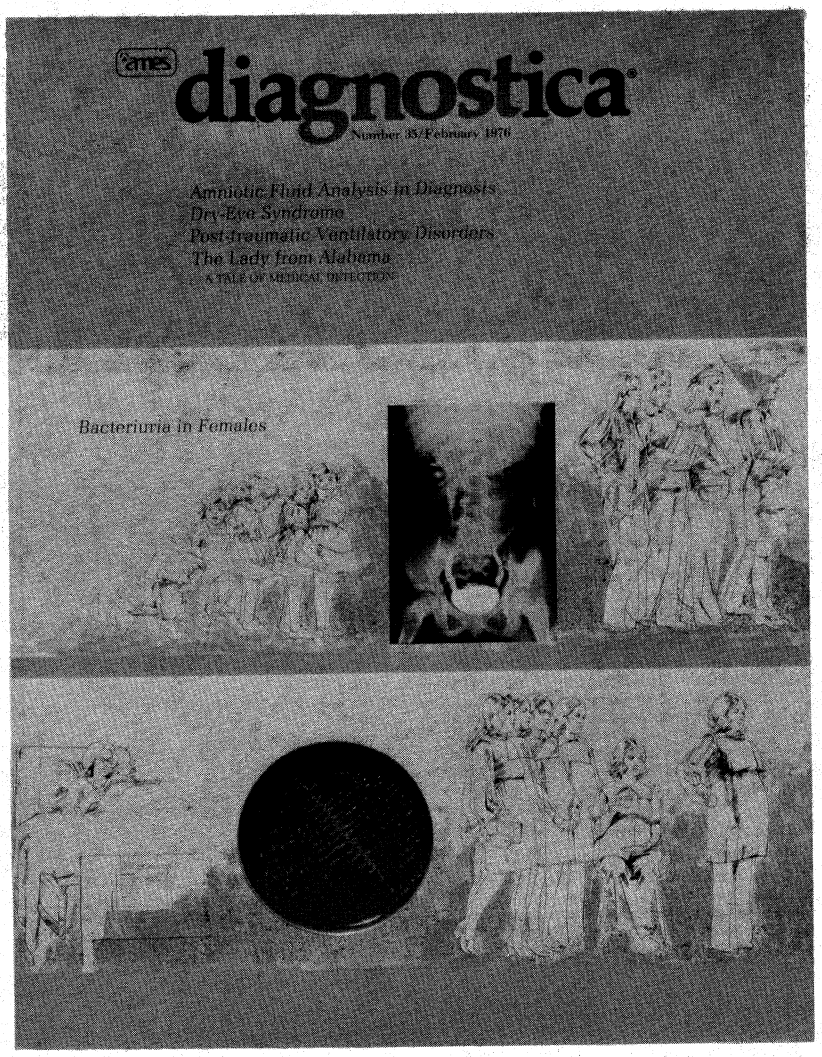
(Fam Pract News 5[11]:31, 1975)

Airport X-ray Machines: How Much Radiation?

According to Laurence E. Holder, MD, radiation has conjured up grotesque images of destruction ever since the atomic bombing of Hiroshima and Nagasaki... This may account for the emotional reaction some people have concerning x-ray machines installed as part of airport security. The Bureau of Radiologic Health, a Division of the Food and Drug Administration, is responsible for keeping radiation exposure as low as possible. Their guidelines require that radiation emitted outside the system not exceed 0.5 milliroentgens (mR) in one hour at 5 cm from the equipment. And in reality, exposures are *below* detectable limits... In the minute it takes you to walk past the detector, the radiation intensity is about 0.0021 mR. (The *roentgen* is a measure of exposure in air, whereas a *rem* is a measure of the amount of energy deposited in tissue.) The genetically significant dose of radiation is estimated as probably less than 20 mrem. Nonoccupationally exposed individuals should be limited to 170 mrem/yr.

(Holder LE: JAMA 233:1393, 1975)

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The Lady from Alabama
A TALE OF MEDICAL DETECTION
Bacteriuria in Females

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Number 35/February 1976

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Cover

As many as 15-20% of the female population will develop bacteriuria at some stage of life, notably in the preschool and elementary-school years or during pregnancy. Furthermore, the female child is prone to develop ureteral reflux as evident in the cystourethrogram. These and other factors that predispose toward bacteriuria in females—together with the ultimate possibility of permanent renal damage and the indications for diagnostic monitoring—are evaluated by Morris Notelovitz, MD, on page 4.

Articles

4 Bacteriuria in Females
by Morris Notelovitz, MD (Rand), MBBCH. MRCOG

12 Amniotic Fluid Analysis in Diagnosing Fetal Abnormalities
by Takashi Wagatsuma, MD

19 Post-traumatic Ventilatory Disorders
by William A. Cook, MD

Pictoclinic®

16 Dry-Eye Syndrome
by Marcia Spinak, MD, and Paul Henkind, MD, PhD

Tales of Medical Detection

27 The Lady from Alabama
by Richard D. Altick

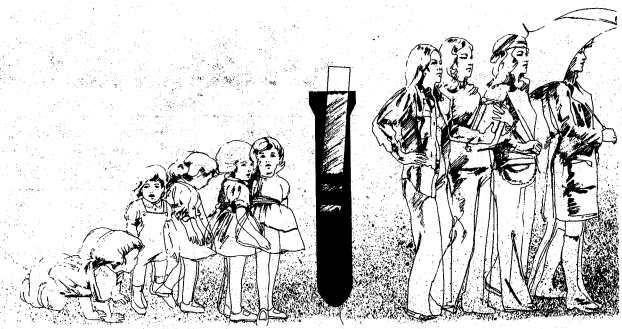
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Bacteriuria in Females
Detection, Prevalence, Implications

by Morris Notelovitz, MD (Rand), MBBCH, MRCOG*

Bacteriuria is one of the most common bacterial infections to affect females.[1] Because of its cyclic pattern, the number of females who develop bacteriuria at some stage of life is at least three times greater than the total who are bacteriuric at any one time.[2] On this basis, it is estimated that 15-20% of females in the economically developed countries may be affected at some stage of life.[1]

Prevalence

The prevalence of bacteriuria varies according to age, social background and ethnic group, as well as the clinical situation.[2] Screening surveys for bacteriuria among preschool children have shown that 1-2% of girls tested had infected urine, whereas this finding is rare among boys.[3-5] The incidence of bacteriuria decreases somewhat in girls at the secondary-school level.[6,7] An increased incidence is again observed during pregnancy, when 3-8% of pregnant women may be expected to develop significant urinary infections. The incidence of bacteriuria is also higher among hospital inpatients and outpatients than in the general population. Indwelling urethral catheters are clearly implicated,[8] with females having a higher rate of catheter-associated bacteriuria than males.[9]

Bacteriuria Defined

Urine, which is normally sterile, provides an excellent culture medium for many microorganisms. The clinical problem of differentiating a urine culture that is positive due to simple bacterial contamination from one that is referable to significant bacteriuria and clinical illness was resolved by Kass.[10-12] In pioneering studies, Kass found that patients with bacterial counts greater than 100,000 colonies per milliliter of urine usually had similar bacterial counts in subsequent specimens, and that the organisms present were known urinary pathogens. Low bacterial counts usually involved urethral commensals or mixed gram-negative and gram-positive organisms, suggesting that contamination had occurred. On re-examination, the urine usually proved negative for bacteria. The term *significant bacteriuria* therefore means that bacteria are actively multiplying in bladder urine, and that "true" infection exists when the bacterial count reaches 100,000 or more colonies per milliliter of urine. This value may be expressed as 10^5.

Asymptomatic bacteriuria. Covert or "asymptomatic" bacteriuria—which is extremely common—refers to significant bacteriuria in the *absence* of any subjective urinary symptoms. Only two fifths of the bacteriuric schoolgirls who were recently surveyed had subjective symptoms.[3] In contrast, Mond et al[13] found that 50% of the female patients seen in general practice for urinary complaints had bacteriuria. Obviously, the presence or absence of dysuria, urinary frequency, or nocturia—symptoms typical of urinary tract infection—can be misleading. One can only rely upon the accurate bacteriologic examination of the urine for accurate diagnostic information.

CLINICAL SIGNIFICANCE OF BACTERIURIA

Pyelonephritis and renal failure. A major concern about bacteriuria is its potential role in the develop-

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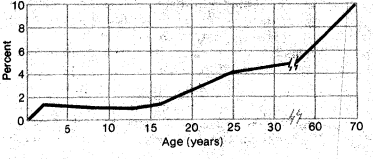
ment of chronic pyelonephritis and, thus, in renal failure. Follow-up studies of individuals who had documented bacteriuria at least ten years earlier were rather inconclusive; although some of them demonstrated radiologic evidence of chronic pyelonephritis with signs of renal malfunction and azotemia, the majority had no apparent renal defects. Inconclusive results were mainly due to the small sample evaluated.[1] But to obtain evidence of whether the treatment of bacteriuria actually protects patients against acquired renal lesions, a controlled clinical trial involving some 100,000 patient-years would be necessary.[11]

Asymptomatic pyelonephritis. There is increasing evidence that bacterial infection of the kidney need not be accompanied by the classic features of acute pyelonephritis, and that abnormal renal function may exist for months before the symptoms of pyelonephritis appear.[1,14] Brumfitt and Condie[14] referred to this as "asymptomatic" pyelonephritis. The wedge-shaped distribution of infection in the kidney suggests that the most likely invasion route is via the collecting tubules as a consequence of infection ascending from the bladder and ureters.[15,16] Hematogenous infection can occur, but usually only when bacteremia is manifested.[2] Early detection of bacteriuria and asymptomatic pyelonephritis is clinically important, because renal functional abnormalities—evidenced by a deterioration of renal concentrating ability and the presence of specific antibodies to kidney tissue—can be reversed by appropriate therapy. Normal renal function is then restored.[1]

Bacteriuria during pregnancy. The spontaneous cure rate of bacteriuria during pregnancy is estimated as only 8%, compared to 20-25% per annum[2] in nonpregnant women. The difference is probably due to the dynamics of incomplete emptying of the bladder during pregnancy. Therefore, the incidence of symptomatic infection is high among pregnant women; an estimated 20-40% risk developing pyelonephritis later in pregnancy.[2,14] The risk can be eliminated by treating a pregnant patient *before* clinical illness develops.[2]

Perinatal risk. Although maternal pyelonephritis is clearly associated with the occurrence of premature labor and perinatal loss, a possible contributory role of asymptomatic bacteriuria is less certain. According to some researchers,[2] 10-25% of bacteriuric women deliver prematurely. However, variables such as race, socioeconomic background, smoking habits, and a history of reproductive loss (ie, abortion, stillbirth, or neonatal death) also play a role. Women with bacteriuria who subsequently develop pyelonephritis during pregnancy do have a high risk of delivering prematurely; the risk may be reduced by treatment. Thus, Condie et al[17] and Elder et al[18] found

Prevalence of bacteriuria in females according to age.





that a high rate of premature births in pregnant women with bacteriuria was limited to those whose bacteriuria either relapsed after treatment or proved unresponsive to therapy. Patients whose infections cleared completely usually did not have an excessively high rate of premature delivery or other morbidity. Because recurrent disease is frequently associated with evidence of renal involvement, it appears that renal involvement *per se* predisposes toward excess perinatal risk, rather than the persistent bacteriuria.[19] In short, since the early treatment of bacteriuria prevents symptomatic disease, its prevention should substantially decrease the incidence of premature births accompanying symptomatic pyelonephritis.

Bacteriuria in schoolgirls. Although bacteriuria is clearly prevalent among schoolgirls, it is not known whether this may present an eventual health hazard. Screening surveys have shown that between 1-2% of schoolgirls have infected urine.[3-5,7] Approximately 30% also had vesicoureteral reflux, and 26% exhibited radiologic evidence of pyelonephritic scarring.[4,7] Most had little or no history suggesting previous urinary tract infection. Pyelonephritic changes are thought to develop from the combination of covert bacteriuria and vesicoureteral reflux. Pyelonephritic scarring is probably nonprogressive unless it is associated with severe vesicoureteral reflux.[20] Most vulnerable in this respect are children under the age of five,[21] since renal scarring is more likely to occur when the kidney is not fully grown. Persistent bacteriuria in children is associated with a genitourinary abnormality in 5-40% of cases.[1]

Bacteriuria and hypertension. Kass estimates that 10-20% of hypertensive females may have an associated bacteriuria. However, it is not clear whether the bacteriuria itself leads to hypertension, or whether the renal vascular disease and resulting kidney damage predispose toward secondary infection. Available data favor the hypothesis that bacteriuria is associated with hypertension directly, rather than indirectly through secondary infection.[1]

Catheterization and bacteriuria. Urinary infection acquired during the puerperium or after gynecologic surgery frequently is due to catheterization. Brumfitt et al[22] studied 320 women known to have had sterile urine before delivery. Of women who were not catheterized, 4.7% developed infected urine during the puerperium. In contrast, 9.1% of the women catheterized only once for specimen collection developed urinary infection. Of those in whom catheterization was performed for clinical reasons, 22.8% developed bacteriuria. In women with urinary retention, the incidence rose to 40.6%. The majority remained asymptomatic, and the urinary infection either resolved spontaneously or cleared following one course of chemotherapy.

Catheterization following gynecologic surgery is also associated with a high incidence of urinary infection, especially in those with indwelling catheters and open drainage. In contrast to the rapid response of urinary infections during the puerperium, bacteriuria that develops after pelvic surgery frequently persists. Simmons and Baker[23] studied 47 women who had undergone vaginoplasty six years previously; in 31.9%, significant bladder infection was discovered. In all instances, infection was attributable to operation and catheterization. Although such infections usually do not produce serious renal damage, the sequence of catheterization followed by bacteriuria, pyelonephritis, bacteremia and, ultimately, death has been reported.[1] Such complications can be prevented by applying appropriate techniques in carrying out



Cystogram illustrating bilateral ureteral reflux in 6-year-old girl.

catheterization and by using closed-circuit drainage systems.[8]

DIAGNOSIS OF BACTERIURIA

Pour-plate technique. The accepted methodology for diagnosing a significant urinary tract infection is quantitative bacteriology; the standard procedure for counting bacterial colonies is the pour-plate technique. According to this method, either blood agar or MacConkey's agar plates are inoculated with aliquots of undiluted urine obtained with standardized sterile loops or pipettes, preferably within minutes of voiding. Plates are incubated overnight at 37°C, with blood agar in an atmosphere of increased CO_2 tension. Colonies are counted the next day. It is recognized that this standard laboratory technique is liable to variation and error. This has led to the evaluation of other diagnostic methods, many of which provide accurate bacteriologic information and are also convenient and economical.

Dip slides. Brumfitt[24] described a simple diagnostic method using a strip of sterile filter paper that is first dipped in urine, then placed on a small agar container and incubated. This technique provides good results, but the differentiation between "doubtful" and "negative" findings is frequently difficult. The dip slide appears to provide the best approach to a fully quantitative method. Dip slides, now available from various manufacturers, have essentially the same design consisting of a glass or plastic slide coated on both sides with nutrient agar or an inhibitory selective agar. Slides are inoculated immediately after urine collection and incubated overnight in their sterile containers. Bacterial colony counts are then estimated by comparing them with the colony density depicted on manufacturers' charts. Dip-slide testing has been extensively evaluated in adults and children; results compared favorably with the pour-plate technique used as control.[3,25-27] A more recent innovation is the application of antibiotic sensitivity discs to the lower end of the dip slide prior to incubation.[27] A zone of inhibition around the disc indicates that the organism is sensitive to that antibiotic. Thus, the practitioner may obtain both diagnostic and therapeutic information within 24 hours of urine collection.

Chemical method: glucose oxidase test. Chemical methods have also been developed for diagnosing bacteriuria. Scherstén and Fritz[28] used the fact that urinary glucose concentrations of less than 0.1 μmol/l occur in the presence of significant bacteriuria provided that: a) the patient is fasting; b) the urine has remained in the bladder for 4-6 hours. Low urinary glucose concentrations can be detected by a commercially available paper-strip test reagent (glucose oxidase) that reflects the small amounts of sugar utilized during bacterial multiplication. A negative result is indicated by the blue color that develops within 10 minutes, while a positive result—equivalent to a urinary glucose concentration lower than 0.1 μmol/l—is indicated by the absence of a color change. This test's major drawback is that it requires a urine sample from a fasting patient; failure to adhere to this requirement leads to false-negative results, undesirable in any screening device. For example, in one recent survey of schoolgirls screened for bacteriuria,[3] the glucose-oxidase test strip indicated a false-negative result in 20.8% while the dip-slide technique produced no false-negatives.

Chemical method: nitrite test. Still another chemical test is based on the observation that the Griess reagent turns pink in the presence of nitrite formed during the reduction of urinary nitrate by certain gram-negative pathogens such as *Escherichia coli*. This commercially available test can detect 60-70% of cases of significant gram-negative bacteriuria.[29,30] Sensitivity is enhanced by withholding micturition for a few hours, and also by waiting at least one minute before interpreting the test result.[29] This method is far from ideal, as—in the author's experience—approximately 30-40% of false-negative results can be anticipated. False-positive results are infrequent.[29] According to a recent report by Kunin,[31] self-administration of the nitrite test by women who participated in a screening program resulted in 90% detection of those with bacteriuria when three different

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morning specimens of urine were tested. The results were confirmed by quantitative urine culture.

Combined nitrite test, colony counting, and gram-negative reaction. More recently, a unique diagnostic test strip has been introduced which incorporates a "nitrite-sensitive" pad for immediate identification of a gram-negative infection plus two culture pads: one identifies the *total* bacterial colony count and the other, gram-negative pathogens. The test strip is presented in a sterile plastic envelope. After the strip is inoculated with urine, the nitrite reaction is read and the strip is replaced and sealed in its container, then incubated for 24 hours. Bacterial density is interpreted according to a chart provided by the manufacturer. Advantages provided by this test medium are a) an *immediate* result that is accurate in about 60% of significant gram-negative infections, and b) it distinguishes gram-negative from gram-positive pathogens. In a survey of 500 unselected female hospital attendants,[32] the author detected a 2.0% incidence of asymptomatic bacteriuria using this method, as compared to the 2.8% detected by a standard laboratory pour-plate culture.

Microscopic examination. Direct microscopic examination of the urine is frequently used as a test for bacteriuria. Its advantage is that smears of uncentrifuged urine can be stained and examined within minutes. Comparing the Gram-stained urine smear with bacterial counts in urine, Norden and Kass[33] reported a 20% error by the Gram-stain technique.

Pyuria, which is defined as two or more white blood cells per microscopic field (centrifuged urine examined by high-power microscopy), is usually associated with urinary infection. There are many other causes of pyuria besides infection, so that as compared to bacterial colony counts, this criterion yields an unacceptably high rate of false-negative results.[2] Furthermore, the absence of pyuria is not a reliable criterion for excluding the presence of bacteriuria.

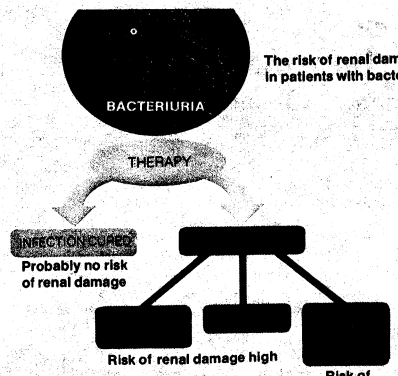
COLLECTING URINE SPECIMENS

Accuracy of any one of the diagnostic methods described is directly related to the manner of urine sampling. For example, urine retained in the bladder for approximately 4-6 hours before collection enables any bacteria present to multiply, and thus greatly enhances test sensitivity. This is noticeable with the nitrite test in particular.[30] An early-morning urinary specimen is therefore ideal, provided that the test reagent can be inoculated immediately or within one hour of collection. Alternatively, the specimen should be refrigerated at 4-6°C until cultured.

The "casual" collection of urine or inadequate cleansing may produce contamination and unreliable results. While transabdominal bladder aspiration is possibly the ideal method of urine collection, it is clearly impractical for screening purposes. A clean-catch specimen is usually obtained easily: All that is required is that the patient wash her hands thoroughly, cleanse the introitus front to back using four sterile gauze swabs soaked in a 10% green-soap solution[2] and separate the labia widely while micturating. Edwards et al[3] obtained excellent results among schoolgirls by requesting them to void upon awakening directly on both sides of a dip slide—the so-called "dip stream."

PREDICTING RENAL INVOLVEMENT

Care must be exercised when assessing the significance of bacteriuria and deciding on the need for treatment, considering that the rate of spontaneous disappearance of bacteriuria in nonpregnant women is about 20-25% per annum.[2] Recurrence of bacteriuria, appearance of symptomatic disease, or demonstration of renal-tissue invasion (especially if the patient is asymptomatic) will require careful assessment, treatment, and follow-up. With regard to diagnosing renal involvement, it is known that patients with pyelonephritis are unable to concentrate the urine maximally. This also has been noted in bacteriuric patients who are asymptomatic.[2,34] Patients unable to concentrate their urine above 700-



800 mOsm are more likely to develop symptomatic renal disease.[2] Treatment of bacteriuria in such cases usually improves the renal concentrating ability.[2]

Antibody titer and prognosis. Another major contribution to detecting renal involvement in a patient with asymptomatic bacteriuria is the demonstration of a specific antibody response to infecting bacteria, as it correlates with histologic evidence of pyelonephritis.[35] Patients having the highest titer of specific antibody are more likely to have renal involvement. Successful treatment is associated with a lower antibody titer; conversely, a lack of therapeutic success is linked to persistently high titers. As in patients with renal-concentration defects, women having high antibody titers—especially in early pregnancy—risk developing renal symptoms. There is a close correlation between the severity of a renal-concentration defect and the antibody titer.

Finally, Wren,[36] who quantitated the leukocytes excreted in urine of pregnant women, found that both symptomatic pyelonephritis and the delivery of infants of low birth weight were more common among bacteriuric women having quantitative pyuria than in bacteriuric women without pyuria. To some extent, quantitative pyuria was predictive of bacterial infection of the kidney.

WHO SHOULD BE SCREENED FOR BACTERIURIA?

Whether large-scale screening should be carried out to detect asymptomatic bacteriuria in females must be considered judiciously until the natural history of untreated bacteriuria—and its etiologic role in renal disease—has been elucidated. At present, it appears that screening girls younger than five years is justified because of vulnerability to intrarenal reflux and infection followed by permanent kidney scarring. The screening of pregnant women for bacteriuria is also justified, since an estimated 70-90% of cases of potential pyelonephritis associated with pregnancy can be eliminated on one screening, which pinpoints bacteriuric patients for antibiotic treatment. However, pregnant patients should be tested on at least two occasions, since an estimated 1% will later develop bacteriuria.[2] Some authors[2,37] advocate that—just as for proteinuria—one should routinely check for bacteriuria during pregnancy.

Other valid indications for the routine testing of bacteriuria include: patients who have undergone catheterization of the bladder; following treatment of urinary tract infection in order to establish a clinical cure, or to detect an asymptomatic relapse or disease recurrence. Finally, it is the author's opinion that each infection of the urinary tract should be regarded as a potentially chronic problem.

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Amniotic Fluid Analysis in Diagnosing Fetal Abnormalities

Some Clinical and Experimental Observations

by Takashi Wagatsuma, MD*

Although the genesis and mechanisms leading to formation of amniotic fluid have not yet been completely clarified, it is obvious that the amniotic sac and its fluid constitute a "fetal compartment" surrounded by the chorion and the placenta, both of which are products of conception. Although water, electrolytes and other solutes contained in the amniotic fluid may originate in part from the mother, the cells and macromolecules contained in this fluid are of fetal origin, and hence would logically reflect fetal properties. Therefore, sampling the amniotic fluid by amniocentesis would appear to be a useful means of detecting genetic disorders of the intrauterine fetus.

Clinical Applications of Amniocentesis

The first systematic attempt to sample amniotic fluid for the purpose of obtaining information about the condition of the fetus was conducted in 1952 by Bevis.[1] This researcher determined the content of bile pigment in cases of erythroblastosis fetalis due to Rh isoimmunization of the mother. Then, in 1960, Riis and Fuchs[2] first applied the examination of amniotic fluid in their investigation of sex-linked hereditary diseases.

During the past few years, amniocentesis (ie, removal of a sample of amniotic fluid by percutaneous transabdominal puncture) has been gaining importance as a means of detecting genetic disorders and congenital abnormalities *in utero*.[3-8] Table 1 lists the indications and tests used in diagnosing various fetal conditions.

Prenatal detection of chromosomal aberrations. The cytologic and cytogenetic study of cultured or uncultured amniotic-fluid cells has become established as a practical diagnostic technique. Interpretation of results helps in providing adequate genetic counsel to mothers having a high risk of bearing a child with chromosomal abnormalities. Down's syndrome (mongolism) is one of the many genetic diseases which may be diagnosed by cytogenetic techniques. Among the other diagnosable chromosomal abnormalities is 13-trisomy, a condition characterized by multiple congenital anomalies.

Enzymes in amniotic fluid. A new and challenging approach to the prenatal diagnosis of hereditary disease is the analysis of enzymes contained in cultivated or noncultivated cells collected from amniotic fluid. Reviews of enzymatic studies describe at least 15 enzymes isolated from cultivated cells. Numerous metabolic disorders have recently been diagnosed in the fetus by enzyme analysis.

Sex-linked hereditary disease. The antenatal determination of sex is useful in preventing sex-linked hereditary disorders. Although this approach does not permit one to make a direct diagnosis of all sex-linked diseases, it is highly useful since only the male suffers from these diseases and, thus, selective preventive measures can be taken. The technique should only be used as a medical indication for therapeutic abortion when sex-linked disease is a distinct possibility. Otherwise, the test could be exploited by those having a strong preference for a child of a particular sex.

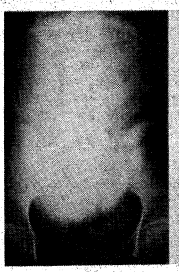


Fig. 1.—Normal human fetus as shown by fetography.



Fig. 2.—Fetograph reveals anencephalic human fetus.

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Table 1.—Amniotic-Fluid Analysis

INDICATION	FETAL OR MATERNAL CONDITION
Antenatal diagnosis of congenital abnormalities (in general)	Chromosomal aberrations Inborn errors of metabolism Sex-linked hereditary disease Congenital adrenal hyperplasia
Diagnosis of fetal malformations	Congenital malformations Hydramnios Pregnancy with diabetes
Assessment of fetal maturity	Rh incompatibility Pregnancy with diabetes Pregnancy with uncertain dates Severe toxemia
Amniotic water volume	Hydramnios
Fetoplacental function	Placental dysfunction

Consequently, this approach could present a serious medicolegal problem — at least in some countries.

Congenital adrenal hyperplasia. Jeffcoate et al.[9] in 1968, successfully predicted the diagnosis of congenital adrenal hyperplasia from levels of 17-ketosterones and pregnanediol estimated in amniotic fluid; levels were markedly elevated as compared to those of four normal pregnancies. However, Fuchs and Cederqvist[10] do not believe that one can predict congenital adrenal hyperplasia solely on the basis of hormonal levels in amniotic fluid.

Amniography and fetography. The ability to detect either hereditary or nonhereditary fetal malformations would be extremely useful. Techniques directed toward this goal essentially rely upon visualization of the fetus. Four diagnostic approaches have been taken: roentgenography, amniography or fetography, ultrasonic scanning, and direct visualization. Amniography or fetography are modifications of the classic x-ray technique of visualizing the intrauterine fetus. Contrast medium is injected after amniocentesis is carried out. Soft-tissue abnormalities in the fetus, such as meningocele, displace the opacified amniotic fluid, permitting the intrauterine detection of abnormalities. Some results of fetography performed in our department are illustrated in Figures 1 and 2. The first shows a normal fetus; the second, anencephalus.

Assessing fetal maturity. There are instances in which pregnancy must be terminated prematurely in order to save the infant's life. This may occur, for example, in some instances of severe toxemia, prolonged pregnancy, Rh incompatibility, or diabetic pregnancy. In these and certain other conditions, it would be enormously helpful to confirm gestational maturity when any doubt exists and, also, to detect early signs of any fetoplacental dysfunction. Presently available diagnostic methods include: fetal heart rate and electrocardiogram; fetal electroencephalogram; sonography; amniography; and urinary estrogen value. However, these tests do not always provide the information desired. With increasing confidence being placed upon amniocentesis, new test methods are now appearing in the literature. It seems reasonable to suggest that the evaluation of amniotic fluid—the fetal environment—will one day provide maximal information regarding various fetal conditions.

Amniotic-fluid volume determinations. Several methods now available for determining the amniotic-fluid volume by amniocentesis are clinically important in diagnosing hydramnios and oligohydramnios.

Clinical Experience with Amniocentesis in 91 Women

Between July 1966 and May 1973, 141 amniocenteses were performed in 91 women at the Department of Obstetrics and Gynecology of the University of Tokyo. Indications for the procedure are listed in Table 2. Among these are genetic counseling, diagnosis of fetal anomalies, Rh incompatibility, and assessment of fetal maturity, which

Table 2.—Indications for Amniocentesis in 91 Cases*

INDICATION	NUMBER
Assessment of fetal maturity	37
Genetic counseling	22
Therapeutic amniocentesis for hydramnios	16
Management of Rh isoimmunization	13
Feto-placental function test	6
Previous fetal anomalies	4
Detection of fetal anomalies	3

*Two patients had more than one indication for amniocentesis.

Table 3.—Results of Amniocentesis: 141 Procedures in 91 Patients

TIME PERFORMED (Gestational Week)	TOTAL (No.)	SUCCESSFUL (No.)	UNSUCCESSFUL (No.)
14-16	3	1	2
16-20	13	12	1
20-24	18	17 (2)	1
24-28	11	10	1
28-32	27	26 (2)	1
32-36	25	27 (4)	1
36-40	32	30 (5)	2
40	6	6	1
Total	141	131 (13)	10

*Numbers in parentheses indicate cases in which diagnosis could not be made because of anterior presentation of placenta.

comprise 70% of all indications for this procedure. In a few instances, amniocentesis was not possible due to an anterior placenta. Nonetheless, the rate of successful diagnoses obtained in the entire group was extremely high (Table 3).

Risk to the mother. There are several possible risks of amniocentesis that may affect the mother; however, only a few instances of maternal morbidity have been observed. These risks may include:

- Amniotic-fluid embolism
- Hemorrhage
- Peritonitis
- Abdominal pain
- Abortion (premature labor)
- Abruptio placentae
- Premature rupture of membranes

Risk to the fetus. Although fetal complications of amniocentesis are relatively more frequent than maternal complications, fetal effects attributable to the procedure are rare. They have included:

- Death due to disruption of fetomaternal circulation or amnionitis
- Injury to umbilical cord
- Hemorrhage
- Abortion or premature labor due to ruptured membranes
- Fetomaternal transfusion
- Possible teratogenic effect

Experimental Studies of Amniocentesis

The possibility of a teratogenic effect of amniocentesis recently attracted the attention of several investigators. In certain small experimental animals such as mice or rats, there occurs a phenomenon known as the "post-amniocentesis dysmelic syndrome." Dr. Jimbo of our department performed amniocentesis unilaterally on a uterine horn of Wistar rats on day 15.5 of gestation. Fifty-four fetuses were delivered on day 21 and examined histologically; animals delivered from the opposite uterine horn served as controls.

As shown in Table 4, in the experimental group 43 fetuses were aborted; 30% showed adhesions to the amniotic membrane. In the control group, two fetuses died *in utero* and none of the 52 survivors showed any significant abnormalities. Ten of the 11 viable fetuses from the uterine horn subjected to amniocentesis had

gross malformations such as cleft palate (8 of 10 animals), or dysmelia affecting the forelimbs or hind limbs. Figure 3 illustrates a rat fetus with dysmelia of the left forelimb. Also, the abdominal wall of this fetus was thinner than that of control animals.

Neither the high incidence nor the extent of fetal complication following amniocentesis in animals has been observed in the human. Factors relating to differences in teratogenic effect of amniocentesis between animals and man include:

- Species differences in reacting toward potential teratogenic factors
- Species differences in anatomy of uterus, placenta and chorionic membrane
- Differences in the amniotic water volume
- Differences in adhesion potential of amnion to fetus causing deformity
- Differences in gestational age at which amniocentesis was performed

Conclusion

Amniocentesis has provided important data on fetal maturity, sex, and chromosomal abnormalities, and is an invaluable tool for genetic counseling. Neither the incidence nor severity of complications observed in mice and rats following experimental amniocentesis is found among humans. Species differences may account for this discrepancy. However, a need exists for additional clinical information on the risks of amniocentesis considering both the mother and the fetus. Clearly, long-term postpartum follow-up of the infant is needed. Only then can the risk-benefit ratio of the procedure be accurately assessed.

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Table 4.—Comparative Results of Amniocentesis in Rats

	CONTROL (Untreated)	TREATED (Amniocentesis)
Number of fetuses	54	54
Aborted	0	43
Intrauterine death	2	0
Viable fetuses at birth	52	11
Visible malformation	0	10



Fig. 3.—Rat fetus from uterine horn subjected to amniocentesis; dysmelia of the left forelimb is apparent.

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Dry-Eye Syndrome

by Maria Spinak, MD*
and Paul Henkind, MD, PhD†



Fig. 3.—Lesions of the skin and mucous membrane including eye involvement as found in Stevens-Johnson syndrome.

Fig. 1.—Dry spots on the cornea associated with a deficiency of mucus.



Fig. 2.—Superficial epithelial erosions of the cornea due to persistent dryness.

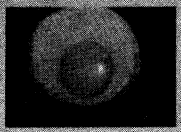


Fig. 4.—Stevens-Johnson syndrome with symblepharon.



The dry-eye syndrome may be caused by an abnormality of any component of the normal tear film or by some irregularity in the position or anatomy of the eyelid. Keratoconjunctivitis sicca—which refers to the involvement of both the conjunctiva and cornea—is generally caused by the inadequate moistening of the corneal surface due to a decreased flow of tears. It may occur as a strictly localized ocular condition, but more frequently it accompanies systemic disease.

Diagnostic procedures used to define the cause of dry-eye syndrome include Schirmer's test, cytologic examination of tissue scrapings or biopsy, and electrophoretic or microbiologic assay of tears.

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The normal eye is well lubricated by a film of tears comprised of three layers: mucoid, watery, and oily, respectively. If any one of these physiologic lubricants becomes deficient, the individual develops a "dry-eye syndrome" or *keratoconjunctivitis sicca*. Although it may be primary (localized to the eye), this syndrome not infrequently accompanies systemic disease.

In a patient suffering from a dry-eye syndrome, the anterior surface of the eye loses its usual glistening appearance and may appear dry. The patient usually complains of irritation, burning or discomfort, but often the eye "feels moist"; rarely is dryness a subjective complaint. Visual function will ultimately be impaired if the corneal surface develops irregularities as a consequence of persistent dryness.

Composition of Tear Film Layer

Of the three components that normally comprise the tear film, the innermost is 1) a *mucoid* layer elaborated by conjunctival goblet cells. This layer coats the epithelium of the conjunctiva and cornea. Spread over the mucoid layer is 2) a middle *watery* layer composed of lacrimal fluid secreted by the major and accessory lacrimal glands. Floating on the watery layer is 3) a lipid monolayer derived from meibomian gland secretion. This outer *oily* layer helps to prevent tears from evaporating or spilling over the boundary of the eyelids. Abnormality of any tear film component, irregularity of eyelid position, or other anatomic disturbance can induce the dry-eye syndrome.

What Causes an Abnormal Tear Film?

Mucous deficiency. A deficiency of mucus in tears will produce dry spots on the cornea because of the hydrophobic nature of the exposed epithelium (Fig. 1). If dryness



Fig. 5.—Sjögren's syndrome with swollen parotid glands, dry mouth, and arthritis.



Fig. 6.—Conjunctivitis and blepharitis due to a lack of tears.



Fig. 7.—Debris and mucous threads in the inferior fornix.



Fig. 9.—Diminished lacrimation is demonstrated by using filter paper to demonstrate wetting at Schirmer's test.



Figs. 8 a,b.—Rose bengal staining of the conjunctiva and cornea, a diagnostic test for dry eye.



persists, the superficial epithelial layers will erode with ensuing corneal ulceration (Fig. 2). Among the conditions that can scar the conjunctiva and thereby create a secondary deficiency of mucus are the Stevens-Johnson syndrome (Figs. 3,4), toxic epidermal necrolysis, benign mucous-membrane pemphigoid, avitaminosis A, trachoma, chemical burn, and the postvaccinial syndrome.

Lacrimal fluid deficiency. The most common cause of the dry-eye syndrome is an underproduction or absence of the middle watery layer comprising the lacrimal fluid. The ocular abnormality may be primary; however, most affected patients have associated systemic disease. Sjögren's syndrome is the classic example (Fig. 5). Such patients suffer from dry mouth, arthritis and, in some cases, swelling of the parotid glands. Other disorders linked to the dry-eye syndrome include rheumatoid arthritis, osteoarthritis, sarcoidosis, psoriatic arthritis, systemic lupus erythematosus, scleroderma, polyarteritis nodosa, and familial dysautonomia (Riley-Day syndrome).

The patient with a lacrimal-fluid deficiency usually has bilateral ocular disease manifested by erythematous eyelid margins (Fig. 6); a diminished or absent tear meniscus; debris in the precorneal tear film, ie, mucous threads in the inferior fornix and beneath the upper eyelid (Fig. 7); superficial punctate erosions of the cornea and conjunctiva demonstrable upon rose-bengal staining (Figs. 8 a,b); and the appearance of epithelial filaments on the cornea.

Schirmer's test. A common diagnostic procedure for detecting dry-eye disorders is Schirmer's test (Fig. 9) in which a strip of folded filter paper standard for this test is inserted behind the outer third of the lower eyelid with approximately 25 mm of the strip protruding downward. The patient may keep the eyes open during this 5-minute procedure. If at that time less than 10 mm of the free end of the strip is wet, a deficiency of lacrimation is indicated.

Oily-layer deficiency. A rare cause of dry eye is a deficiency of the oily layer of the tear film. Such deficiency may result from the atrophy of the meibomian glands following—for example—irradiation of the eyelids.

Loss of anatomic integrity. The normal tear film can also be disrupted by a poor approximation of the eyelids to the globe of the eye as is associated with either entropion or ectropion, and with defects and irregularities of the eyelid margins.

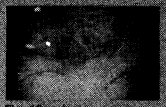


Fig. 10.—A closeup view of the lower eyelid reveals a marked irregularity of inner margin.



Fig. 11.—Keratinization of epithelial cells observed in conjunctival scrapings.

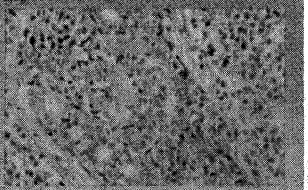


Fig. 12.—Advanced stage of dry eye with proliferation of squamous epithelium typically associated with xerosis.

Fig. 13.—Periductal lymphocytic infiltration with atrophy of the acini in the lacrimal gland.



Fig. 14.—Epimyoepithelial islands due to focal hyperplasia of ductal cells, characteristic of Sjögren's syndrome.



(Fig. 10). A break in the normal tear film with subsequent damage to the underlying epithelium may also result from inturned eyelashes (trichiasis), perilimbal elevations following conjunctival surgery or developing during phlyctenulosis, wearing contact lenses, or the incomplete closing of eyelids during sleep.

Other Tests for Dry Eye

Cytology. Early in the disorder, a conjunctival scraping will reveal prexerosis of the epithelial cells (Fig. 11). As the disorder progresses, the keratinization of epithelial cells increases, mucous-laden cells may appear and purplish mucous threads become abundant. Diphtheroid bacilli typically associated with xerosis become apparent at this stage (Fig. 12).

Biopsy. Histologic examination of the conjunctiva may reveal an atrophied conjunctival epithelium, edematous underlying tissue, and a reduced number of goblet cells or their absence. Upon examining the lacrimal gland histologically, one observes degeneration of acinar structures with periductal infiltration by lymphocytes (Fig. 13). The formation of epimyoepithelial islands is characteristic of Sjögren's syndrome (Fig. 14).

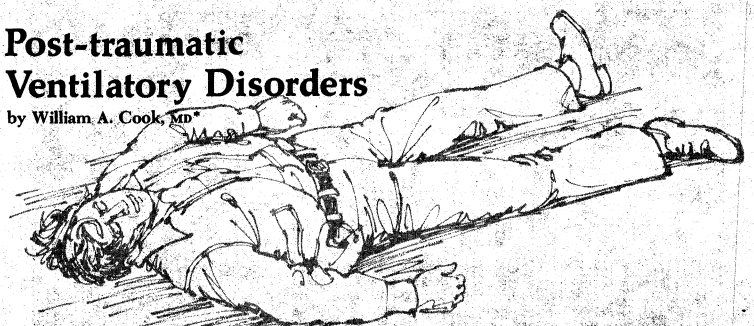
Other diagnostic tests. Either electrophoretic or microbiologic assay will reveal a decrease or absence of lysozyme, an enzyme normally present in tears.

Treatment

Efforts should be made to restore the normal tear layer. Measures to prevent corneal dryness include the application of artificial tear solutions, eg, eyedrops. In some cases one may prescribe contact lenses to be used with eyedrops consisting of 0.5N saline. In a patient with a severe permanent decrease in the tear flow, occlusion of the lacrimal puncta by means of a fine electrocautery needle helps to conserve the supply of tears by preventing their escape. A miniature pump-like device is available for implantation in the upper or lower eyelid to produce synthetic tears automatically. In certain patients with severe dry eye but without Sjögren's syndrome and parotitis, one may choose to transplant Stensen's duct. Additionally, antibiotic eyedrops are administered to the patient having a concurrent staphylococcal infection of the eye.

Post-traumatic Ventilatory Disorders

by William A. Cook, MD*



Among the most widely investigated—yet incompletely understood—ventilatory phenomena is sudden pulmonary failure following surgery or traumatic injury. Various post-traumatic ventilatory disorders may arise in severely injured patients. Examples are the so-called "low flow" lung syndrome or shock lung, pulmonary burns, wet-lung syndrome, pulmonary fat embolism, ventilator pneumonia, and hematogenous pneumonia. Therapeutic difficulties may arise from either a confused or an inadequate differential diagnosis. For clinical emphasis, therefore, these disorders will be discussed in the order in which their manifestations are seen clinically.

Pulmonary burns and wet lung are each caused by a direct injury to lung tissue so that their onset is immediate. Low-flow lung and fat embolism each have an intermediate onset, whereas ventilator pneumonia and hematogenous pneumonia are the last to occur, and may become superimposed on any preexisting ventilatory disorder.

IMMEDIATE ONSET

Wet-Lung Syndrome

This type of post-traumatic pulmonary disorder results from a severe bruise to lung tissue itself. During World War II, Brewer and coworkers[3] observed the post-traumatic wet-lung syndrome in casualties who were exposed to a blast or who had suffered a crushing blow to the thorax or abdomen. In such instances, compressive energy is transmitted via the chest wall or diaphragm to the lung confined, as it is, in the thoracic cage. The sudden compression of lung tissue ruptures the small vessels and produces hemorrhage and edema, both interstitial and intraalveolar. Wet lung is suspected when there is a history of compression injury together with an early onset of pulmonary symptoms. Ordinarily this disorder can be identified when the patient is admitted to the emergency department.

In a patient with the wet-lung syndrome, the chest x-ray shows diffuse haziness—either local or widespread—in one or both lung fields. Upon physical examination, one finds rales and rhonchi; and, if the lung is badly injured, there may be evidence of consolidation. Hemothorax or pneumothorax, or both, may be present (Fig. 1a). The bruised lung may be identified by reexpanding the collapsed lung that is on the injured side of the body (Fig. 1b). Arterial blood-gas analysis reveals hypoxia whose severity is consistent with the severity of lung-tissue injury. Hypoxia is related to a loss of compliance that results from the exudation of plasma and which, consequently, interferes with surfactant function. Factors that contribute to hypoxia include:

- Mechanical restriction secondary to pulmonary-tissue edema
- "Physiologic shunt" effect in perfused but nonaerated lung tissue
- Restrictive effect of injury on the chest wall

Fortunately, most patients who suffer from the wet-lung syndrome will respond well to appropriate treatment (Fig. 1c).

Pulmonary Burns

Pulmonary burns are usually associated with facial burns or with burns sustained in a confined space, and are therefore suspected in any patient who has suffered severe burns under those conditions. Cytologic examination of the sputum (ie, exfoliative cytology) helps to pinpoint the diagnosis. Furthermore, such patients often require tracheostomy; visual inspection of the airway at that time may confirm the diagnosis by disclosing edema, erythema, or actual scorching of the tracheal mucosa.

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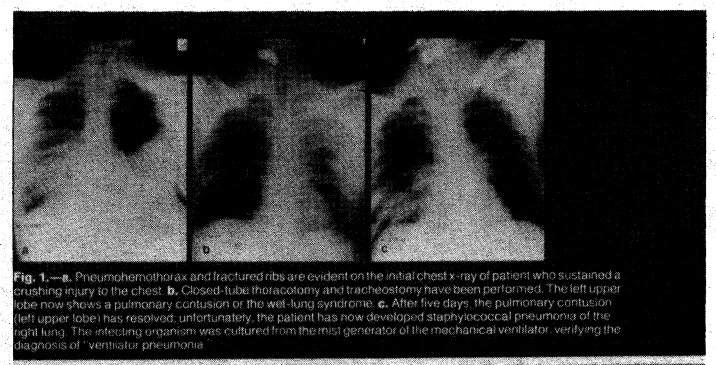


Fig. 1.—a. Pneumohemothorax and fractured ribs are evident on the initial chest x-ray of patient who sustained a crushing injury to the chest. **b.** Closed-tube thoracotomy and tracheostomy have been performed. The left upper lobe now shows a pulmonary contusion or the wet-lung syndrome. **c.** After five days, the pulmonary contusion (left upper lobe) has resolved; unfortunately, the patient has now developed staphylococcal pneumonia of the right lung. The infecting organism was cultured from the mist generator of the mechanical ventilator, verifying the diagnosis of "ventilator pneumonia."

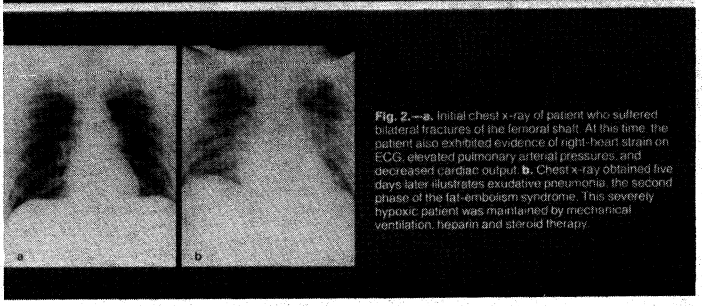


Fig. 2.—a. Initial chest x-ray of patient who suffered bilateral fractures of the femoral shaft. At this time, the patient also exhibited evidence of right-heart strain on ECG, elevated pulmonary arterial pressures, and decreased cardiac output. **b.** Chest x-ray obtained five days later illustrates exudative pneumonia, the second phase of the fat-embolism syndrome. This severely hypoxic patient was maintained by mechanical ventilation, heparin and steroid therapy.

Early in the clinical course of a patient with pulmonary burns, the chest x-ray usually reveals a diffuse alveolar exudate with local areas of atelectasis due to the presence of edema fluid in the alveoli and to bronchial blockage by exfoliated epithelial debris. On arterial blood-gas analysis, one finds that the pH is lowered while Pco_2 is increased. Hypoxia is caused by the inability of oxygen to diffuse into alveoli and across the edematous alveolar-capillary membrane. A "physiologic shunt" effect may ensue if the alveolar-capillary units are perfused adequately without being ventilated. A lack of oxygen diffusion and the resulting shunt effect can be identified on pulmonary function testing, ie, nitrogen or helium "washout," and by determining the pulmonary diffusion capacity of carbon monoxide.[5] In the presence of reduced ventilatory function, nitrogen or helium washout tests reveal a definite "slow space" effect related to those areas of alveolar hypoventilation. Diffusion capacity is reduced by interstitial and intra-alveolar edema.

INTERMEDIATE ONSET

Fat Embolism in Lung Following Injury

Soon after a severe injury, fat may be liberated into the blood from fractured bones. It has been suggested that fat globules may also be formed in blood by the aggregation of chylomicrons. Fat emboli are entrapped by small pulmonary vessels, leading to the *first* stage of pulmonary fat embolism which is characterized by the following: elevated pulmonary arterial pressures; right-axis deviation demonstrated on the electrocardiogram; decreased cardiac output and low arterial pressure; enlargement of the right heart and the right pulmonary artery (observed on x-ray, Fig. 2a); and an apparent increase in physiologic "dead space" detected by pulmonary function testing.[12] The latter is due to the lack of perfusion of alveoli, which may or may not be ventilated.

At this stage, the diagnosis may be confirmed either

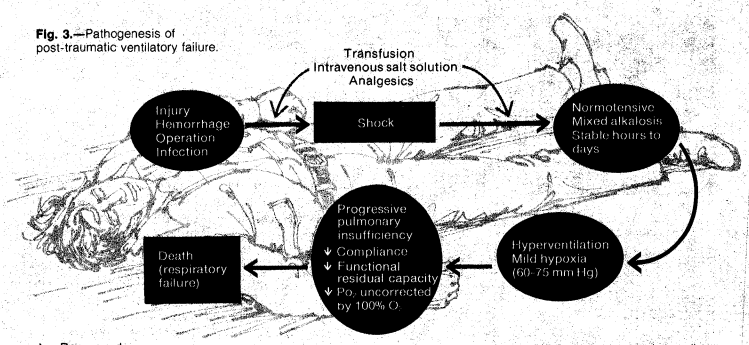


Fig. 3.—Pathogenesis of post-traumatic ventilatory failure.

Adapted from Rogers, RM: Shock lung, or respiratory distress syndrome of adults. *Continuing Educ* 3:26, 1975.

by detecting free fat in the urine or increased activity of serum lipase. Early diagnosis may be lifesaving. Administration of heparin is recommended on the same basis as in thromboembolic disease.[4] Besides increasing blood flow through the pulmonary capillaries, heparin may help to reduce the aggregation of chylomicrons and fat globules so that they can be transported through the pulmonary bed.

As the fat embolism breaks down, it produces a *second* stage of pulmonary involvement in the form of exudative pneumonia. The latter results from a reaction of the pulmonary parenchyma to the products of lipid breakdown.[2] Even though x-rays clearly reveal exudative pneumonia (Fig. 2b), physical examination of the patient may be remarkably unrevealing, since the pulmonary involvement is primarily interstitial. Arterial hypoxemia usually is present and often progressive. Pulmonary function tests reveal a marked diffusion block, ie, alveolar-capillary block due to an inflammatory response. The latter may be controlled by administering massive doses of steroids which purportedly stabilize the cell membrane and thereby lessen the release of lysosomal enzymes. However, if the condition progresses, a decrease in pulmonary compliance and the closure of small airways leads to progressive hypoxia which, together with the oxygen-diffusion block, results in arterial oxygen desaturation. This condition is extremely difficult to treat, even by controlled mechanical ventilation. Some benefit may be obtained by administering concentrated oxygen and continuous positive end-expiratory pressure.[12]

Low-Flow Lung Syndrome

During the Vietnamese war, casualties were rapidly evacuated from the battlefield to medical units at which they were given resuscitative as well as definitive care by surgeons and paramedical personnel. This led to the highest ratio of survivors to fatalities documented during wartime. But a new clinical problem then became apparent: after successful resuscitation from shock, some of the wounded died of pulmonary complications 4-7 days following injury—despite effective ventilatory support. Often patients without chest injuries—but with extensive injuries to other parts of the body—were affected. This lethal syndrome of post-traumatic pulmonary failure (shock lung or low-flow lung) is today well recognized as a potential complication of any severe injury that leads to shock.

Moore and coworkers[9] classified the pathophysiologic phases of post-traumatic ventilatory failure as follows:

- Phase I: Injury, resuscitation, and alkalosis
- Phase II: Circulatory stabilization and beginning of respiratory difficulty
- Phase III: Progressive pulmonary insufficiency
- Phase IV: Terminal hypoxia with hypercarbia and asystole

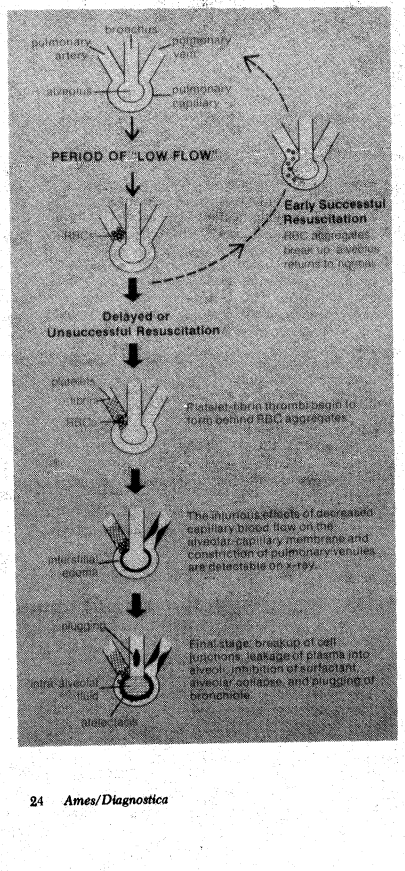
The lethal progression from injury through resuscitation to the state of terminal hypoxia is illustrated in Figure 3.

The most important diagnostic clue to low-flow lung is a history either of shock or low cardiac output following trauma. Pulmonary complications may follow any condition that leads to low cardiac output,



Fig. 4.—Microphotograph of animal lung (*in vivo*) following hypovolemic shock. Capillaries around the central alveolus are engorged with erythrocyte aggregates.

Fig. 5.—Pathophysiology of low-flow lung.



with the probable exception of cardiogenic shock. In our laboratories, we have observed the onset of the disease process in animals only five minutes after hypotension had been induced by hypovolemia. Microscopic studies of intravascular events reveal the aggregation of formed elements of the blood—particularly the erythrocytes—into progressively larger clumps (Fig. 4). Such aggregates tend to settle in relatively dependent lung areas, while plasma streams toward the upper areas. Pulmonary capillaries may then become blocked by the aggregates. If the patient is resuscitated in time, the aggregates will break up and reenter the circulation. However, platelet-fibrin thrombi may form behind the aggregates leading to a reactional edema of the perivascular tissues; this process goes on to destroy the endothelial and epithelial cells of the lung, producing exudation of plasma into alveoli, loss of surfactant function, decreased compliance, and atelectasis (Fig. 5). Occasionally, the process may actually be aggravated by resuscitative efforts.[8] Experiments in our laboratory suggest that simply by administering large volumes of crystalloid solution (eg, glucose, saline), one can trigger the aggregation of erythrocytes to cause interstitial edema of the lung.

In addition to the cellular effects of the low-flow state described, it seems likely that a humoral component causes constriction of the small veins.[11] The combination of all these factors produces a maldistribution of air in the lung and progressive alveolar-capillary block. As in patients with fat embolism, the clinical onset of post-traumatic pulmonary failure may be insidious because the initial pulmonary reactions are interstitial; few, if any, physical abnormalities are apparent. Characteristically, the patient experiences arterial oxygen desaturation following an apparently successful resuscitation from the initial shock state (Fig. 6a). Hypoxia supervenes within hours, and even though one hears normal chest sounds on auscultation, the x-ray shows a diffuse infiltration of both lungs (Fig. 6b). The patient breathes deeply and rapidly; there is marked metabolic acidosis, arterial oxygen desaturation and, terminally, the carbon-dioxide level increases in arterial blood. The most prominent finding is hypoxia which is secondary to a severe diffusion block produced by intra-alveolar septal edema (see Fig. 5). As the process continues, atelectasis and pulmonary congestion become superimposed.

LATE ONSET

Ventilator Pneumonia

A pulmonary complication observed in some patients who have required assisted ventilation is best known as "ventilator pneumonia." This is the last pulmonary complication to appear, and becomes superimposed upon the existing pulmonary disorder. Ventilator pneumonia is caused by the inhalation of warm, moist air which encourages a rapid proliferation of transient bacteria originating in either the device or the patient's respiratory tract.

Each stroke of the ventilator may drive bacteria deep into the lungs. Since the phenomenon of antibiotic resistance is not uncommon in a patient who has already received several courses of antibiotics, treatment of ventilator pneumonia may be difficult. Serial sputum cultures help one to make an early diagnosis. Prophylactic measures should be employed to help prevent ventilator pneumonia, such as the clean maintenance of machine circuits together with aseptic care of the tracheostomy wound.[14]

Typically, the chest x-ray of a patient who has ventilator pneumonia shows a more localized pulmonary infiltration (Fig. 1c) as compared to other respiratory syndromes. Physical examination reveals the classic signs of consolidation and fluid in small airways. Hypoxia is present; pulmonary function tests reveal abnormalities in ventilatory quotient, alveolar-capillary block, and perfused nonventilated lung tissue with physiologic shunting. It is at this advanced stage that the patient may also develop hematogenous pneumonia.

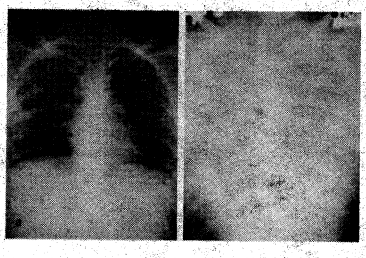


Fig. 6.—a. Chest x-ray of female patient six days after she fractured the pelvis and left femur during a fall related to a hypotensive episode. The lungs are essentially clear. **b.** Chest x-ray of the same patient 18 hours later shows a diffuse infiltration of both lungs. During this period she became severely hypoxic, but exhibited no signs of pulmonary disease on auscultation. Despite mechanical ventilation and a period of extracorporeal oxygenation with a membrane oxygenator, the patient died.

Hematogenous Pneumonia

Hematogenous pneumonia, which may develop at a late stage of any septic disorder, is seen on x-ray as a patchy, disseminated infiltrate. Commonly it follows massive burns, but may be associated with any septic, post-traumatic state. During the early stages of hematogenous pneumonia, the patient may present few significant physical findings because of the interstitial nature of the disease. This type of pneumonia is highly lethal; it responds—if at all—to therapy directed at both the pneumonia and the septic focus.[7]

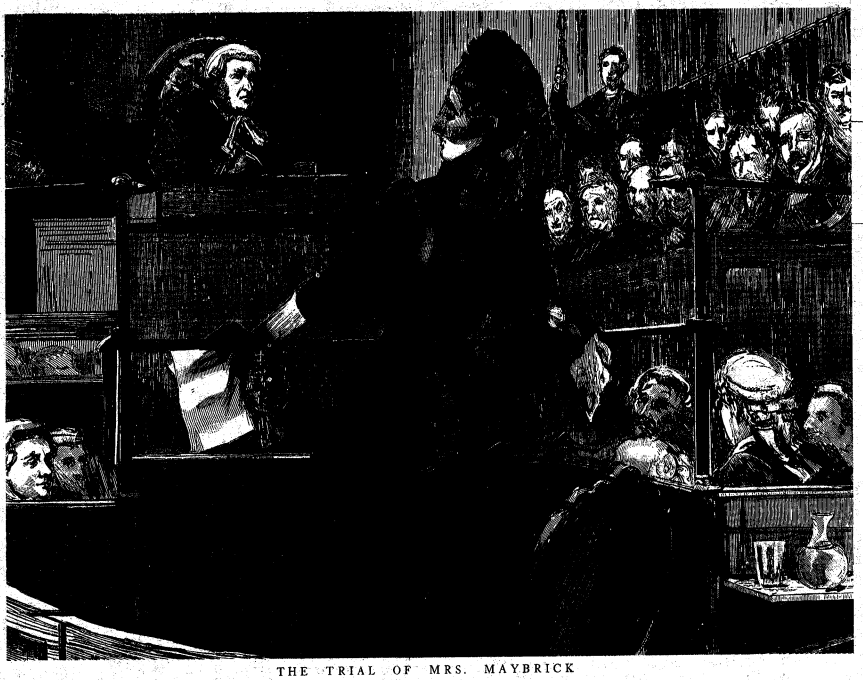
Summary

Knowledge of the pathogenetic sequence and time of clinical appearance of the various pulmonary disorders described should help to clarify what has been a confusing group of post-traumatic problems; thus, it will lend specificity to treatment.

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THE LADY FROM ALABAMA

by Richard D. Altick



THE TRIAL OF MRS. MAYBRICK

Time: 1889
Place: Liverpool, England
Category: Fact

The case of Florence Maybrick offers, if anybody is disposed to look at it that way, a fortuitous conjunction of Jack the Ripper and Henry James. The former's association with Mrs. Maybrick is chronological; the sixth victim attributed to him by some authorities, and, as it turned out, the last according to anyone's count, was found on July 18, 1889, and Mrs. Maybrick's trial opened in Liverpool thirteen days later. Henry James's association is of a very different, much subtler

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She was accused as a poisoner, even though her "victim's" prescriptions included arsenic, cascara, henbane, iridin, jaborandi, morphine, papain, prussic acid, and strychnine.

sort, perhaps not really demonstrable: but one thinks of him because Mrs. Maybrick participated in that memorable exodus of marriageable young women from America to Europe between the end of the Civil War and turn of the century which so often hovers in the background of James's fiction. It is altogether too fanciful to see her as a small-scale, bourgeois replica of a Jamesian heroine—and yet...

Mrs. Maybrick was born Florence Elizabeth Chandler, the daughter of a banker in Mobile, Alabama; her husband, twenty-four years older than she, was a Liverpool cotton merchant temporarily resident in America, where they were married in 1881.*

The story of the Maybrick murder is a simple one, the complications in the trial record being almost exclusively due to the battle of the toxicologists being once more rejoined. On May 11, 1889, James Maybrick died "under mysterious circumstances" in his home, after an illness attended by many distressing symptoms. The principal evidence against his wife was the presumption of motive. In March she had spent a few nights in London with another man, and later the same month, after their return from the Grand National Steeplechase, where they had happened to meet him, she and her husband had had a violent argument, in the course of which he had given her multiple bruises and a black eye. Only the pleas of a servant and the family doctor dissuaded her from leaving him. Moreover, a month later, a week or so before her husband's final illness began, she had bought flypaper at a chemist's, even though flies were not yet in season and there was some flypaper left in the kitchen from last year. A servant saw her soaking the paper in her bedroom basin to remove the arsenic coating. Her explanation was that she wanted to make a cosmetic solution to clear her complexion, as she was planning to accompany her husband to a ball. At the trial, some evidence was produced that arsenic was occasionally favored as a complexion aid or, alternatively, as a depilatory cream. Another chemist from whom Mrs. Maybrick had bought a dozen flypapers in April said, "I can speak to the fact that ladies came to buy flypapers when no flies were about." But the line of inquiry this statement invited—the possibility that certain other ladies' husbands subsequently died of violent gastric disturbances—was not pursued.

Combined with this indisputable possession of arsenic was the fact that Maybrick's nurses reported some apparent sleight-of-hand on his wife's part with the beef juice that the doctor had ordered given to the patient. A search of the house after his death revealed the presence of arsenic in tiny or more significant amounts on a rag, in one of Mrs. Maybrick's handkerchiefs, in a bottle of aperient mixture, in a bottle of glycerine, in a packet marked "Poison for Cats" (*sic*—not "rats"), and elsewhere. All told, the analysts estimated that the arsenic found scattered about the house was enough to kill fifty people. But all this was circumstantial evidence, and in its totality it did not constitute a crushing case against the young woman.

A comparison of this trial conducted in 1889 with those of 1856-65 reveals how far the Age of Science had progressed since those remote days when toxicology and forensic medicine were in their hesitant, inexperienced infancy. The medical evidence which occupies by far the greater part of the Maybrick transcipt (the scene of virtually the whole reconstructed drama is the victim's sickroom) has a much more scientific air about it, a greater assumption of authority. Home Office analysts were not forced to admit, as one previous analyst had, that the poison revealed by their analysis was derived from their analytic tools. But the trouble was that along with heightened authority should have come consensus; and no such consensus was reached. The doctors disagreed as violently as ever.

Was Maybrick's death really caused by arsenic? Some expert witnesses testified that it was, while others, equally expert, testified with equal assurance that it was not. The defense labored mightily to show that Maybrick suffered from gastroenteritis. Certain it was that, despite a basically healthy constitution, he was a hypochondriac of

*There is delicious reverse snobbery in the statement of James G. Blaine, the Secretary of State in Benjamin Harrison's cabinet, when he wrote in support of his countrywoman's reprieve: "That she may have been influenced by the foolish ambition of many American girls for a foreign marriage, and have descended from her own rank to that of her husband's family, which seems to have been somewhat vulgar, must be forgiven to her youth, since she was only eighteen at the time of her marriage."

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long standing, addicted to taking all kinds of medicines. Medicines were a favorite topic of his in conversation, and he eagerly followed the advice of his friends, sometimes doubling the recommended dose to be sure of good results. Evidence was brought from Norfolk, Virginia, where he had lived for a time, and from Liverpool itself, to the effect that he had sampled virtually the whole pharmacopoeia, with special attention to substances normally deemed lethal in sufficient quantity but including also such relatively benign items as quinine compound, cardamom pills, and Seidlitz powders. He was in the habit of dropping into a chemist's shop in downtown Liverpool from two to five times a day for a "pick-me-up" laced with liquor arsenicalis, the quantity of which was steadily increased over ten years. If this suggests sheer aberration on the part of the cotton broker, it must be noted that, as his chemist testified, many other level-headed businessmen he knew liked their daytime potations strengthened with arsenic. Whatever the practice says about Maybrick personally, it says a good deal more about the pressures of Liverpool business life.

By May 1889, one would therefore assume, Maybrick had built up a high tolerance for a wide range of chemicals, pharmaceutical and nonpharmaceutical, against those last days when, as a letter to *The Times* after the trial put it, his stomach served as "a druggists' waste-pipe." For his malady, whatever it was, his doctors prescribed, in addition to measured doses of arsenic, a variety of substances including strychnine, jaborandi, cascara, henbane, morphia, prussic acid, papaine, and iridin. It was a formidable intake for a patient of whom one physician testified, "He seemed to be suffering from nervous dyspepsia. I should say that he was hypochondriacal." But neither nervous dyspepsia nor hypochrondria would seem adequate to account for the voluminous and the disagreeable symptoms Maybrick suffered toward the end, except on the not unreasonable premise that the curative regimen was worse than the disease. In view of his well-attested appetite for those many drugs, one wonders why his wife should even have been suspected of doing away with him. If she was intelligent enough to soak flypaper in a basin, she was also surely intelligent enough to realize that nature or the doctors, or both, would eventually do her work for her. Still, considering how long he had been ingesting those potions and powders without harm, she may be forgiven for doubting that, in his case, nature was to be much relied upon.

The technical and contradictory nature, repetitiveness, and often the repulsiveness of the medical testimony do not recommend the trial record to the layman's attentive reading. But it is lightened by occasional passages in which the atmosphere and action of the courtroom are vividly evoked. At one point, for example, an expert witness handed in as evidence a glass tube containing a "film" composed of arsenic crystals obtained from Maybrick's liver. The judge, James Fitzjames Stephen, tried two high-power magnifying glasses but was unable to see the film and asked defense counsel to point it out to him. Counsel very properly admonished, "You must look for it yourself, my lord." Stephen tried again, this time with the court chaplain's slate hat as background. After a pause of some time, during which the court was silent, his lordship handed back the tube to the jury, with his two glasses, explaining that one was much more powerful than the other, but the more powerful one required to be so near to the eye on the one hand, and so close to the object on the other that it was very difficult to manage. The jury then proceeded to use the glasses of his lordship, the black coats of their fellow-jurymen being used as a background.

That the judge had to be instructed by counsel as to his proper conduct when presented with an exhibit is a small indication of what was, in fact, a governing circumstance in the trial: his evident incompetence. James Fitzjames Stephen, a brilliant lawyer, legal historian, and man of letters, had become a judge of the high court in 1879. But by now it was obvious that his mental powers were no longer what they had been. H. B. Irving wrote of this disturbing state of affairs with exemplary delicacy:

"It is impossible for the historian of the Maybrick case to ignore the statement, frequently made, that at the time of her trial the judge's mind was suffering from the early attacks of an insidious disease which, two years later, compelled Sir James to retire from the bench. The judge



had a slight stroke of paralysis at Derby in 1885, and had been obliged to give up work for a time. Whether on his return to work his mind had entirely recovered its former vigor, or was suffering a gradual loss of strength, is a matter that need not be discussed. But those familiar with the judge's powers during his earlier years on the bench may well doubt, in reading the report of Mrs. Maybrick's trial, whether those powers were as conspicuous and effective in the trial of her case as they would have been had it taken place some few years before. Of the judge's scrupulous anxiety to be fair, just, and considerate towards the prisoner no impartial reader can doubt."

Perhaps it is unfair to cite the following excerpt from the judge's summing up as evidence of his failing intellect, but to most Englishmen it would be otherwise inexplicable: "The next date after that took place is the Grand National 'something.' I don't know whether it is a race, or a steeplechase . . . but it is something called the 'Grand National,' as if everybody knew what the substantive was—but the Grand National took place on the 29th March." Counsel at the bar and spectators must have mentally shaken their heads at such ignorance. In any case, Stephen's conduct of the trial called forth much sharp comment in both the press and the legal profession and provided the chief basis for later action in Mrs. Maybrick's behalf. Stephen left the bench two years later, was created a baronet, and died in a private asylum in 1894.

Florence Maybrick was found guilty and was sentenced to death. But the case had aroused so much interest, and her plight so much sympathy, that almost half a million persons throughout the kingdom signed petitions to the Home Office urging her reprieve. Among those who circulated the petitions were members of the Liverpool Exchange (who obtained signatures over quick ones laced with arsenic at the corner chemist's?) and medical men, who argued that the quantity of arsenic found in Maybrick's body was too small to have caused death and that the symptoms observed during his last illness did not point to arsenic poisoning; some of the classic manifestations were not present. Some members of Parliament signed the petitions. Public meetings in Mrs. Maybrick's support were held in London and Liverpool, and, since Mrs. Maybrick was American by birth, leading officials and citizens of the United States also pleaded in her behalf. Among the names subscribed to the petitions were those of Cardinal Gibbons, Vice-President Levi P. Morton, members of the cabinet in addition to Secretary of State James G. Blaine, and high army officers.

At length, public opinion had its way to the extent that the condemned woman's sentence was reduced to life imprisonment. Mrs. Maybrick was released after fifteen years and returned to the United States, living first in Highland Park, Illinois, then in Florida, and finally in South Kent, Connecticut, as an eccentric old woman in a shack overrun with cats. She died in 1941. The beginning of her story, in the promise of a transatlantic marriage—overlooking the "vulgar" origin of her husband—may have been fit for Henry James's pen, but the long unhappy sequel would have had to be written by another hand.

Vol. 2, No. 3 March 1976

Primary Cardiology

Cardiovascular Medicine for the Primary Care Physician



INTERMEDIATE CORONARY INSUFFICIENCY

Key to therapy: Is it benign or more serious?

By John J. Sampson, M.D.



DIGITALIS AND ACUTE MYOCARDIAL INFARCTION

There are conflicting practices, opinions

By Edgar Haber, M.D.



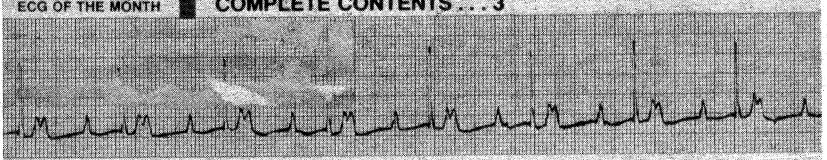
ANTICOAGULANTS: WHEN TO USE—AND HOW

First of a series

By Louis M. Aledort, M.D.

ECG OF THE MONTH

COMPLETE CONTENTS . . . 3



For ECG Analysis See Page 48

Vol. 2 No. 3 March 1976

Primary Cardiology

Cardiovascular Medicine for the Primary Care Physician

Warren J. Taylor, M.D. 10
Care of the Cardiac Surgical Patient
Requires step-by-step planning

Gordon K. Danielson, M.D. 14
Current Status of Prosthetic Valves
Which patients are candidates?

H.J.C. Swan, M.D.
William Ganz, M.D. 20
Diagnosis & Treatment with the Balloon Catheter
Its use is being extended

Arthur A. Sasahara, M.D. 26
Diagnostic Sequence for Pulmonary Embolism
Reducing that high mortality rate

John J. Sampson, M.D. 30
Minor Infarctions: What to Look For
Are they benign or more serious?

Louis M. Aledort, M.D. 38
Anticoagulant Drugs—When to Use, and How
First of a series

Max Harry Weil, M.D. 42
Pulmonary Edema: Treatment Depends on Cause
Replenishment of plasma volume is important

Edgar Haber, M.D. 46
Should Digitalis Be Used in Treating Acute Myocardial Infarction?
There are conflicting practices, opinions

DEPARTMENTS

5 **Editor's Page** Is the lecture obsolete? Edward Gipstein, M.D.
8 **Letters to the Editor**
18 **Photo Feature** Internal heart functions are monitored by noninvasive technique
34 **Case History** Sanford S. Zevon, M.D.
48 **ECG of the Month** Atrioventricular block

PRIMARY CARDIOLOGY™ publishes original articles by leading cardiologists to assist the primary care physician in the diagnosis and treatment of cardiovascular and related illnesses frequently encountered in his medical practice.

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Editor's Page

Is the Lecture Obsolete?

Pressures on the physician to update his knowledge come from many sides. The strongest motivation for continuing his education should, of course, come from the rapid proliferation of new medical findings, and the physician's desire to provide good medical care. But regardless of their personal drive to learn, all physicians will have to face the likelihood that mandatory continuing education—now legally required in a minority of states as a condition of relicensure and continuing hospital affiliation—will some day be required throughout the United States.

Against this background it is disturbing to read the attacks by some writers in medical journals on mandatory continuing education in general and on the lecture format in particular.

These critics assert that the majority of continuing medical education programs rely heavily on the lecture method; that the flow of information is all one way—from the platform to the audience—discouraging the active participation of the learner and fostering passivity and resentment; that compulsory attendance at such programs arouses resistance to learning; and that there is no evidence that physicians render better patient care as a result of their attendance at these courses.

Some critics say that mandatory continuing education and formal lectures should be replaced by a voluntary form of continuing self-education, in which the physicians could select problem-centered learning opportunities directly related to his practice.

Superficially, some of these arguments seem persuasive. There is no denying the destructive effect of a lecture delivered by a speaker who has little ability to hold the interest of a large audience; who has not organized his material well or simplified it appropriately for the occasion; and whose slides are so crowded with data printed in small, pale type that they cannot be read beyond the third row.

But there is a right and a wrong way to carry on any form of medical education, whether it is done through lectures, seminars, journal articles, bedside teaching, or audiovisual teaching aids. Each of these methods, performed badly, wastes the physician's time. Each one, when it is performed well, makes a contribution to medical education which is unique to that method, and not replaceable by any other.

Let us look at the advantages of the lecture when it is delivered at a high level of competence. A stimulating presentation by a specialist who is also a gifted communicator can provide large audiences with a valuable perspective—the result of his survey and crystallization of a tremendous amount of research. In an hour or less, a good speaker can illuminate what is known, what is new, and what is still to be explored, alerting the individual physician to developments that may have immediate bearing on his practice. This also helps the physician to focus his reading in a field crowded with more journals than he could ever survey and evaluate on his own. *(continued page 6)*

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Editor's Page

(continued from page 5)

This is the kind of educational experience we try to provide twice a month through the Medical Education Program at Lawrence and Memorial Hospitals in New London, Connecticut. For the past thirteen years we have used the lecture format extensively. Participating physicians pay tuition, and they come voluntarily, in large numbers, from out-of-state as well as from the immediate area. Subject matter represents many disciplines, and physicians are offered course material closely related to problems they face in their practices. At the end of each lecture, a question-and-answer period provides at least an adequate opportunity for audience participation and for resolution of points that may be unclear.

Since much of what can go wrong with lectures is due to the quality of the lecturer rather than to the lecture format itself, we screen our speakers carefully. They are selected, not only for scholarship, but also for their ability to speak well, to make suitable slide and other visual presentations, and, in general, to have a strong impact on a large audience.

At the end of each session, course participants are asked to fill out questionnaires evaluating the content of the lecture, its value to them, and the effectiveness with which it was delivered. We share the results—bad or good—with the lecturers.

Fortunately, many successful administrators, research scientists, and clinical specialists are also good communicators, and they are invited back again. But there are other equally accomplished physicians who are brilliant in their specialties, or effective at directing large research projects or conducting intensive bedside teaching; and yet they do not have the special gifts required to organize or simplify their material into a good lecture and to present it with flair to a large group. For their sakes, and in the interest of a successful educational program, we feel that they should know how they have been evaluated, and we do not ask them back again.

Response to our lectures suggests to us that this program, and many others like it in this country, constitute one of several possible forms of voluntary, continuing self-education, in which physicians take responsibility for upgrading their knowledge. Despite their supposedly passive roles in a lecture situation, the participation of these physicians in question-and-answer periods, and their responses to evaluation questionnaires, indicate to us that they are involved in some form of learning and they find the courses rewarding.

Of course, we cannot prove that these rewards are passed on in the form of changed physician behavior and better patient care. But we strongly doubt the validity of studies that claim to measure the relationship between a medical education course and subsequent changes in patient care. To quote a writer on this subject in a recent issue of JAMA:

> *There are many controlled factors that influence physician behavior and treatment outcome that were not, and could not be, identified or measured in such studies. While the studies fail to find a change, they also fail to prove that no change occurred.*

Because our experience with the lecture format has been positive, we are interested in seeing that it is preserved as an effective instrument. To do this, more teaching talent must constantly be developed. Perhaps we can look for this talent among the ranks of medical school instructors and teaching fellows, many of whom are effective communicators. If they had more opportunities to represent their departments on the lecture platform, we might see the rise of an important new group of medical lecturers. Since we can expect growing demands to be made on the physician to engage in continuing education, all possible encouragement should be given to the development of such a corps of effective medical educators. □

Edward Gipstein, M.D.
Director of Medical Education, Lawrence and Memorial Hospitals New London, Conn.

Letters to the Editor

We welcome comments, criticism, questions, and suggestions from our physician-readers and will occasionally print letters we receive. Please write to Editor, PRIMARY CARDIOLOGY, 488 Madison Ave., New York, N.Y. 10022

D.O. Specialist

You have made a slip in the October 1975 issue I would like to correct. Next to each doctor's name, you indicate his area of specialization. If a physician is a D.O., it is taken for granted he is a practitioner of osteopathic medicine. If a physician is an M.D., it is taken for granted he is a practitioner of allopathic medicine.

If you were to look up in the Directory of the American Osteopathic Association you will note there is no specialization called osteopathy. There are 54 types of specialization that osteopathic physicians are trained in. Your listing as Dr. Nash's specialty "osteopathy" is an anachronism. For your information, Dr. Nash is certified in diagnostic roentgenology. I trust that in your future issues you will be more specific.

Warren Wolfe, D.O.
Cherry Hill, New Jersey

Aspirin's Role

I have read with interest your note in Clinical Horizons entitled Two Studies Check Aspirin's Role in Stroke, Heart Attack Prevention, in January's issue of PRIMARY CARDIOLOGY. Aspirin has been considered a drug potentially capable of preventing arterial thrombotic diseases. It has been studied in many places, and using varied clinical models. At present, however, it is not clear-cut that it will prevent thrombosis.

Other drugs which can alter platelet function have also been studied for their ability to prevent arterial thrombosis. Persantine (dipyridamole), a drug long known as a coronary artery dilator, is capable of inhibiting platelet uptake of glucose and adenosine. In addition it inhibits platelet cyclic AMP phosphodiesterase. Only in greater than physiologic concentrations does this drug alter platelet aggregation. However, this drug is one of only a few which has clearly demonstrated its in-vivo ability to significantly inhibit valvular-induced embolic disease.

In addition, in clinical settings which alter arterial vessel walls and lead to increased platelet turnover (decreased survival) dipyridamole alone or in combination with aspirin (thereby allowing a lower dose of dipyridamole) can return platelet survival to normal.

The P.A.R.I.S. (Persantine Aspirin Reinfarction

Study), is now in progress. Twenty clinical centers have already entered more than eight hundred patients with an aim for recruitment of two thousand. Patients will be randomized to aspirin alone, aspirin plus dipyridamole, or placebo. These patients will be followed in similar fashion to those described in your article. Eligibility is also similar, with a previous documented myocardial infarction being mandatory.

It is important to realize that only by studying prospectively in double-blind fashion the multiple potential antithrombotic agents, will we be secure in identifying those drugs with clinical efficacy.

Louis M. Aledort, M.D.
Executive Secretary
P.A.R.I.S. Policy Board
New York, New York

No Garbage

I am happy with your subject matter and very happy with your concise, no garbage, presentation. Many thanks.

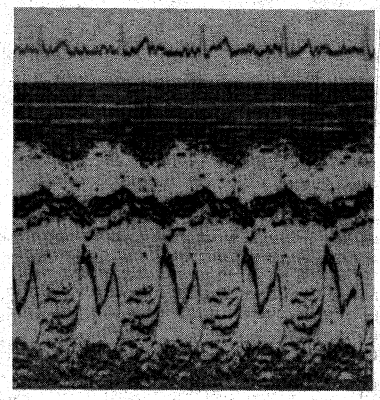
Lou Harrington, M.D.
Lansing, Michigan

Flopped Echocardiogram

I am pleased with the way that PRIMARY CARDIOLOGY is carrying out its mission. The articles in the issues to date are quite readable and relevant. However, I must point out that the echocardiogram is mounted upside down in the February 1976 issue (Mitral Valve Prolapse Clicks: What They Mean, by Robert M. Jeresaty, M.D.).

Lawrence S. Cohen, M.D.
Professor of Medicine and
Chief of Cardiology
Yale University School of
Medicine

Dr. Cohen is, of course, quite correct. The film was accidentally reversed, as was noted by several other readers. Below, the echocardiogram in its proper right-side-up position. The Editor



Care of the

By Warren J. Taylor, M.D.



Dr. Taylor is President of the American College of Cardiology.

The primary physician plays a vital part in the overall management of his cardiac patient undergoing elective surgery. The history, physical, basic laboratory data, and when indicated, testing, provide information that may be critical in the OR. Respiratory function, nutritional status, hepatic and renal function must all be optimal prior to surgery. Consultation with the other physicians providing care during the patient's hospital stay is crucial. Yet one of the most important contributions the primary physician can make is simply "being there" when his apprehensive cardiac patient is anesthetized.

Your cardiac patient is scheduled for an elective or semi-elective procedure. What are the consequences of his cardiac condition for the overall surgical management? The operative stress coupled with the potential hazards of the anesthetic state may have a debilitating effect on an already over-strained heart. Careful attention to the following details will help your cardiac patient have as safe a hospital stay as possible.

Basic Workup

Primary prerequisites are a thorough history and physical exam, basic laboratory data, profile, electrolyte determination, prothrombin time and partial thromboplastin time, chest x-ray, and resting ECG. Stress testing may be helpful in evaluating an individual with occult cardiac disease. It should not be considered as a routine, but when indicated it provides a baseline on cardiac reserve that might prove to be a useful reference when the heart is stressed by the surgical procedure. Often, patients who exhibit a normal resting ECG will, when stressed on a treadmill or bicycle, show ECG changes or manifest chest pain. It is important that all clinicians involved in the management of your patient—anesthesiologists and surgeons—be informed of the results of the stress test and any other pertinent information derived from the history and physical.

Cessation of Beta Blockade?

The cardiac patient who regularly takes beta blockers like propranolol may present a problem. There is recent convincing evidence that sudden cessation of propranolol can precipitate status anginosus or myocardial infarction. Yet having propranolol on

Cardiac Surgical Patient

board during surgery may be hazardous in that the drug may effectively prevent the patient from responding to vasopressors should vasoconstriction be necessary perioperatively.

Clearly a compromise course is indicated. It is advisable to withdraw the drug gradually a week (some say two weeks) prior to surgery. This may be impossible in certain patients, and it may be necessary to accept a degree of beta blockade during surgery, but the drug should be tapered to minimal levels preoperatively.

If emergency surgery must be performed on the cardiac patient who regularly takes propranolol, the hypotension he may experience must be managed in light of the fact that many of the therapeutic agents which would normally be effective simply will not work.

Electrolyte and Fluid Balance

The primary physician should be aware of electrolyte imbalances in the presurgical cardiac patient. A low serum potassium or high sodium, for example, should be corrected with appropriate fluid therapy and judicious diuresis prior to surgery.

Fluid balance is another key consideration. Hypovolemia may result in depletion of metabolic reserves and electrolyte imbalances. The "dried-out" patient may be in relative good health preoperatively, but the stress of surgery and fluid loss may make him hypovolemic very rapidly. This sets the stage for arrhythmias and an operative catastrophe. It is not good clinical practice to overly "dry out" your cardiac patients prior to elective surgery. In fact, many patients do better in the OR when they are a little on the "wet" side.

However, hypervolemia caused by excess fluid replacement with crystalloids or colloids is also to be avoided, especially in the postoperative stage. Similarly, anemia should be corrected.

Assessing Respiratory Function

Baseline pulmonary function values must be ascertained so that any obstructive or restrictive respiratory defect can be identified. Measurement of vital capacity and maximum voluntary ventilation will suffice for initial screening.

Any patient with demonstrated respiratory problems should be prepared for surgery with chest physical therapy and/or respiratory therapy, specifically intermittent positive pressure breathing. Chest physical therapy entails teaching diaphragmatic breathing technics and other ways to optimize pulmonary function, i.e., effective coughing. In addition to the pulmonary function tests previously mentioned, baseline arterial blood gas values should also be obtained in these individuals.

Smoking Absolutely Contraindicated

Many physicians pay lip service to the idea that any patient, particularly a cardiac undergoing elective surgery should abstain from smoking for at least three weeks preoperatively. I feel very strongly that this should be an absolute requirement. If a patient insists on smoking prior to elective surgery, the procedure should be postponed.

Nutritional Status

The importance of nutritional status has recently been stressed by countless clinicians. It is now being recognized that protein calorie malnutrition (PCM) may be responsible for more postop morbidity/mortality than has been previously identified. It may

be advisable to hyperaliment your cardiac cachectic patient prior to an elective procedure. since cachexia may cause defects in the immune response mechanism.

There are several rather simple clues by which you can identify the cardiac cachectic. Many of these patients have albumin deficits and lymphopenia. They are anergic and do not respond to skin testing with tuberculin or *Candida albicans*. If a patient doesn't react to these antigens, it can be assumed his host defenses have been severely compromised.

Anthropometric measurements such as comparing height/weight ratios, arm circumference, or triceps skin fold to established norms can also aid in the identification of these depleted patients.

If the cardiac cachectic is relatively stable, you can afford the luxury of a few weeks of oral or parenteral hyperalimentation prior to surgery. Ideally, one attempts to provide 40 calories and 1.7 grams of protein per kilogram of optimal body weight per day. It is remarkable how well the majority of these patients respond to hyperalimentation. The change from a negative to positive protein balance and adequate caloric intake literally reignites their interest in living. These patients stop being argumentative, paranoid and lethargic.

Preliminary data on one group of patients undergoing multivalve replacement suggest that mortality and morbidity decrease significantly if the patient is properly nourished pre- and postoperatively. Ongoing investigation of this link in many cardiac syndromes will hopefully buttress this observation.

Hyperalimentation is especially useful — even life-saving—in the interim between a major cardiac procedure and elective or semi-elective surgery. Building the post cardiac surgery patient back up to near normal body weight and positive protein balance is imperative before attempting lesser procedures.

Sedation

Cardiac patients are probably the most apprehensive of all. There is a tendency on the part of physicians to combat this fear and its potential cardiovascular ramifications with heavy preop sedation. But this can cause hypoventilation and CO_2 retention, which is much worse for these patients than the anxiety the sedation was supposed to relieve in the first place.

I recommend that the cardiac patient be transported from his room to the OR with oxygen running. If he has angina and nitroglycerin has been effective in controlling it, let him take nitroglycerin before leaving his room and let him carry his bottle of nitroglycerin with him. This can be tremendously reassuring, as well as being therapeutic.

Use preoperative sedatives judiciously. One can always give more sedation, but you can't diminish a heavy dose once it has been administered.

Induction

Certain anesthetic techniques are contraindicated in cardiac patients. It behooves the primary physician, if he is to be a member of the surgical team, and the surgeon to discuss the anesthetic technique with the anesthesiologist well before the operation.

It is well known that certain inhalational agents such as halothane sensitize the myocardium and can precipitate arrhythmias. Hence an anesthetic technique for known cardiacs that seems to be gaining acceptance is I.V. morphine, nitrous oxide, and high-flow oxygen. Though the nitrous oxide does have some cardiodepressive action, this combination seems to present minimal hazards to the diseased myocardium.

Whatever technique the anesthesiologist decides to use, a calm, sedate induction with preoxygenation is certainly to be desired. Too often, the surgical team forgets that the general commotion in the OR becomes amplified for the patient during induction. Soft noises seem to be very loud and disconcerting. There should be no talk and minimal background noise until the patient's level of consciousness descends below the auditory level.

There is one exception to the edict against talking. We should never forget how terribly frightened these cardiac patients are immediately prior to surgery. It is important to stand by them—perhaps pat them on the shoulder—or talk softly to them before they go to sleep. If the primary physician is to be a member of the operative team, he can make a key contribution simply by being near his patient during induction. Seeing a familiar face can make all the difference.

Intraoperative Monitoring

The basic minimal monitoring of a cardiac patient during surgery should include continuous ECG and measurements of arterial and central venous pressures, and urine output. The Swan-Ganz catheter is indicated in the unstable cardiac undergoing surgery. It can be used to measure not only right- but left-sided pressures during surgery and postoperatively. However, since the use of the Swan-Ganz has been associated with certain complications and morbidity, its routine use is not recommended, and when used, the technique should be supervised by someone thoroughly familiar with it.

A gross but helpful assessment of cardiac output is urine output. Low cardiac output will result in in-

adequate renal perfusion and a concomitant drop in urine flow.

Packed Cells Preferred

When replacing blood lost during surgery, the use of packed cells should be considered. We should not be slaves to the notion that whole blood lost must be replaced by whole blood. The use of packed cells can lessen the hazard of hepatitis and other adverse transfusion reactions. In my view, packed cells, supplemented with plasma substitutes meet the patient's requirement for blood replacement with minimal risk. Frozen blood is even better if available in your institution.

Initiating Postop Care

Anesthesia may cause a very labile blood pressure in the immediate postop period. Sudden motion accentuates any instability that may exist. Hence it is critical that anesthetized patients — especially known cardiacs—be transported as gently as possible to the RR, ICU, etc.

Specific postop care is of course geared to the procedure the patient undergoes but two major therapeutic guidelines must be observed at all times.

First, avoid hypervolemia. The cardiac patient is unable to tolerate fluid overload. Secondly, be wary of pharmacologic support of the heart. While cardiotonic drugs such as dopamine or glucagon have a place in the postop armamentarium, excessive inotropic stimulation of the diseased myocardium is very hazardous.

Many patients will require mechanical ventilatory support. Although many clinicians contend that mechanical ventilation should be discontinued as soon as possible, continued use may be indicated in the individual with marginal pulmonary function and labile blood pressure. These patients tend to do better if their work of breathing is reduced, especially if the postop pharmacologic management involves use of agents that will significantly depress respiratory drive.

Analgesia in the postop phase may be associated with significant complications. It should be mentioned that very young or very old patients tolerate potent analgesics — especially morphine — very poorly. Chronologic age, not apparent clinical condition should be the determining factor in morphine dosage. Once again, a good rule of thumb is: give small doses more often rather than a large dose initially. This provides a wider margin of safety.

Early Ambulation

It is advisable to get your cardiac patient on his feet and moving about as soon as he is able. This prevents venous stagnation and lessens the possibility of pulmonary embolism.

An adjunct to early ambulation is low-dose heparin therapy, i.e., 5000 units two times daily. This low dose doesn't cause full heparinization but does retard the thrombotic process. If one elects this therapy, a profile of the patient's bleeding and clotting factors should be available including prothrombin times and partial thromboplastin times.

Prophylactic Antibiotics?

Touching on what continues to be a controversial subject, prophylactic antibiotics, I suggest that the use of antibiotics for this purpose depends entirely on the extent of the procedure, the number of catheters invading the patient and other operative factors. If the potential contamination time for a cardiac patient undergoing a relatively short procedure is minimal, it is probably not necessary to use prophylaxis.

But for longer procedures, prophylactic antibiotics are indicated immediately prior to surgery so that a blood tissue level exists at the time of possible contamination and for three days postoperatively. The drug should be discontinued at this time so that the normal flora are not suppressed and the road to secondary invasion remains closed. If sepsis develops, it should be identified, the organism isolated, the sensitivity determined, and the appropriate antibiotic utilized. □

Practice Procedures

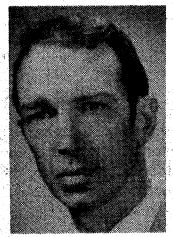
Steps for Managing Cardiac Surgical Patients

Obtain baseline levels for cardiac reserve, pulmonary function and blood gases. Optimalize electrolyte balance, blood volume and fluid. The preoperative nutritional state of the patient may have a dramatic influence on the surgical outcome; hyperaliment when indicated.

- Perioperatively, a morphine, nitrous oxide technique seems to be the least hazardous anesthetic. Minimal monitoring should include ECG, arterial pressure, central venous pressure, and urine output. A Swan-Ganz is indicated in the unstable surgical cardiac.
- Hypervolemia and undue pharmacologic stimulation of the myocardium are to be avoided postoperatively. Ventilatory support may be necessary to reduce work. Early ambulation is a must; heparin therapy is optional to prevent embolism. If prophylactic antibiotics are used, they should be discontinued on the third postoperative day.

Current Status

By Gordon K. Danielson, M.D.



Dr. Danielson is Professor of Surgery, the Mayo Clinic Rochester, Mn.

Although heart valve surgery has come of age, specialists are not yet ready to recommend the procedure for every potential candidate. With any progressive pathologic process, it is fundamentally true that the earlier remedial measures can be instituted the less likely that serious tissue destruction will ensue. Nonetheless, except for some special circumstances, heart valve substitutes — whether of biologic or man-made origin—cannot yet be universally recommended for most patients until the disease process is quite well advanced, because of the remaining inherent risk of such substitutes over a span of years. In many patients for whom this life-saving measure is warranted, there is a remarkable improvement in both the quality and the duration of their life-spans.

After more than a decade and a half of clinical experience with surgical replacement of heart valves, few physicians would question the basic soundness of this approach to the correction of mechanical malfunction of these cardiac structures. Nevertheless, and despite the impressive clinical results of surgical replacement, not all patients with damaged valves are automatically candidates for this type of restorative procedure.

Theoretically, valvular heart disease would seem to be best treated by replacement of the diseased valve. This is an inherently rational concept. But there are at least two major limiting factors: First, in terms of consideration of patient safety, there must be a valid possibility of replacing the diseased valve by a suitable substitute type in circumstances that carry an acceptable operative risk. Second, the prosthetic valve must be free of intrinsic limitations; or, if it lacks this ideal attribute, it must have an established record of performance far better than the prospect that otherwise awaits the patient based on the natural history of the valvular disease in question.

Given these two conditions, and in the hands of a well-trained, experienced team, your patients with valvular disease, in most cases, can be greatly helped to attain an improved quality of life, as well as prolongation of life. This is compared to the clinical deterioration, long-drawn-out progressive disability and attendant emotional impact, or sudden death, that would be the alternative to surgical intervention.

Our group at the Mayo Clinic recently undertook a reappraisal of the long-term results of 1,684 cases of aortic or mitral valve replacement with a Starr-Edwards type of ball valve prosthesis. We confirmed our impression that this measure does appreciably increase life expectancy over what would otherwise be expected in the natural course of valvular heart disease. Various studies have chronicled the natural course of mitral stenosis and have borne out that, without corrective surgery, the median survival time is only 6-7 years. In the last 5 years of our retrospective survey we found that there had been an overall early operative mortality of 7 per cent; and over the entire survey span of 11 years, the operative mortality rate was 14 per cent.

The early mortality rates noted in this survey were 6 per cent for aortic valve replacement and 9 per cent

# of Prosthetic Valves

with the Model 6120 mitral valve procedures. Since then even these rates have been lowered at our institution. What is truly impressive is that the survival rate beyond 8 years after aortic valve replacement was 65 per cent compared with 85 per cent for the general population. Those who survived mitral valve replacement also had an appreciably greater likelihood of surviving 5 years or more postoperatively—a much better prospect than for those who receive only nonsurgical management. Indeed, for patients with mitral impairment, both early and late mortality are likely to be reduced when surgical relief is carried out early in the course of the disease.

Actually, when correction of the aortic or mitral valve malfunction can be achieved early, before advanced disease poses a limit on operative outcome, the surgical risk becomes minimal—no more than about 2 per cent. Furthermore, the long-term results of surgery—even surgery performed years ago with early and less perfected types of substitute valves—are remarkably good in those patients corrected before the onset of devastating secondary effects of the disease process.

Thus, patients who undergo aortic valve replacement before there is significant cardiac enlargement can expect about the same life-span as the general population of the same age and sex. The identical statement applies to mitral valve replacement if the procedure is carried out before the age of 50, and before there is marked enlargement of the left atrium.

When we add up all these pluses and minuses, we can hardly escape the conclusion that the poor results of valve replacement encountered today frequently stem from the fact that surgical correction was delayed beyond the optimal period.

Why is it, then, that few cardiologists recommend the radical step of earlier surgical intervention? For one simple reason: there is still great uncertainty about the long-term function of the substitute valve in comparison to the known long-term function of even a diseased but natural valve at the early stage of the condition.

Although valve replacement is now almost commonplace, we still lack sufficient 5-year-follow-up data on most of the prostheses currently in use. Even those valves based on the traditional models have had some design modification. Unfortunately, as

SCOREBOARD OF RELATIVE MERITS OF VARIOUS SUBSTITUTE VALVES

	Ball valves	Cloth-covered ball valves	Tilting disc valve	Viable homograft	Heterograft	Autogenous graft (stented)
Durability	+++	++*	++	++	+	0
Hemodynamic characteristics	+	+	++	+++	+++	+++
Thromboembolism	+	++	+	+++	+++	+++
Assurance of function	++	++	++	+	+	+
Availability and choice of size	+++	+++	+++	0	++	++
Ease of insertion	+++	+++	+++	+	+	++
Record of survival	++	+	+	++	+	0
TOTAL SCORE	15	14	14	12	12	11

Symbols: 0 = unfavorable; + = fair; ++ = good; +++ = excellent.

*The durability of the metal ball in this type of valve should be excellent, but the noise created by such a prosthesis is significantly objectionable.

experience has warned us, the slightest structural alteration of a valve carries the threat that it might in some unforeseen way contribute to a detrimental outcome, whether immediate or delayed.

Admittedly, we do not now have at hand an ideal or near-ideal substitute for the natural heart valves, but there is every reason to be optimistic about eventual achievement of this goal. Whether the scales will finally be tipped in favor of a mechanically passive or biologically active implant is still a matter of conjecture. Some specialists believe that in the long run the former will win out, because the man-made prosthesis allows greater freedom in the modification of design. With further experience, the design that is most likely to approach the ultimate ideal, at least for aortic valve replacement, will simulate the natural semilunar valve. Our ultimate goal will be attained, however, only with the development of a type of material that will be nonthrombogenic and at the same time as durable as a delicate semi-elastic membrane.

EIGHT YEARS—THREE VALVES

A 48-year-old man underwent aortic homograft replacement for calcific aortic stenosis, with excellent results for six years. He then developed severe shortness of breath on exertion and episodic, paroxysmal, nocturnal dyspnea. Examination revealed severe aortic insufficiency plus gross cardiac failure, necessitating removal of the homograft and insertion of a Braunwald-Cutter valve, but without outpatient anticoagulant medication. About 18 months later, he was well but anemic, and showed schistocytes and burr cells in peripheral blood smears. One month later, hemoglobin count was slightly lower and ferrous sulfate therapy was started. When anemia failed to respond to therapy, patient was readmitted and tests confirmed red cell fragmentation.

Operation revealed that the implanted valve had cloth wear at the distal third of the struts, with retraction of the cloth and with easy displacement of the ball through the remaining struts and the inlet orifice. Substitution of a Bjork-Shiley semicentral flow-tilting valve has given this patient good recovery and an uneventful subsequent course.

What does this all add up to in terms of a rational policy of patient management? We cannot draw up inviolate guidelines to selection of a specific patient as a candidate for a specific type of substitute valve implantation. Each case must be considered individually, and the appropriate valve chosen by the surgeon in charge. Our group, for instance, continues to use predominantly the ball valve for both aortic and mitral positions although trials of the Bjork-Shiley tilting disc and the Hancock heterograft valve are also in progress. Our basic criteria for surgical intervention are to undertake correction of the significantly damaged aortic valve at the time of onset of secondary symptoms, or when the heart has appreciably enlarged (to a cardiothoracic ratio of about 0.57).

We recommend replacement of a significantly damaged mitral valve when progressive disability first carries the patient into the state designated as functional class III (according to New York Heart Association criteria). We also advise an operation if the left atrial or overall heart size is showing progressive enlargement beyond a moderate degree.

For easy reference, here is a greatly simplified thumbnail sketch of the heart valves most commonly used at the present time:

Central Flow-Type Valves

Simulation of flow characteristics of human valves is generally considered desirable in any substitute. Central flow design tends to streamline entrance of blood into the aorta, thereby decreasing energy dissipation and turbulence (a source of trauma to blood components that probably impedes healing). At present this can be achieved only with biologic tissue such as:

Autografts—The pulmonary valve is suitable for use in the aortic position because its configuration is nearly identical with that of the aortic valve. While autografts have the advantage of being fully viable and capable of regeneration and therefore theoretically resistant to late valve failure, follow-up is not yet adequate to confirm this. Criteria for patient selection for this type of replacement are highly selective: it is restricted to individuals with isolated aortic valve disease and life expectancy of 30 years or more.

Viable Homografts—Fresh homograft valves are collected under sanitary conditions and then stored in antibiotic-containing physiologic solutions. Viable cells have been detected in such valves 5 years after implantation—though the total number begins to decrease sharply within 2-4 years. Whether these substitutes will remain viable and continue to function without subsequent degeneration remains to be seen. Homografts mounted on struts have simplified surgical procedures, and allowed use of such implants in sites other than just the aortic position.

An important advantage of the homograft is that anticoagulants are not needed for prevention of

thrombi.

Nonviable Homografts—Freeze-drying, or potent sterilizing solutions, improve availability and storage of homograft valves, although they do destroy viable cells. Initial results were encouraging despite murmurs of aortic incompetence in nearly 45 per cent at the end of the first year postoperatively, but late results show a high failure rate.

Heterografts (porcine)—Preservation is aimed at eliminating antigenic components of the valves which, therefore, become nonviable, with function dependent on their inherent elasticity and flexibility. The preservation methods appears critical to the long-term performance of these valves. Several recent reports indicate favorable results when glutaraldehyde-preserved porcine valves (Hancock type) have been employed, with generally excellent function, low rate of thromboembolus formation, and good durability (thus far up to 5-6 years). They are particularly suitable in patients for whom anticoagulant therapy is contraindicated or highly problematical. Since heterograft inner-to-outer-diameter ratio is less favorable than for prosthetic valves in the smaller aortic valve diameters, enlargement of the aortic root is advised for small aortic valve annuli, or use of a central flow prosthetic valve should be considered.

Tissue Valves—Autologous, homologous, and heterologous grafts of fascia lata, pericardium, rectus sheath, and dura mater have been used. As with a natural valve, preservation and storage must be appropriately employed, except for autologous grafts. In the aortic position these various types have functioned satisfactorily for 4-5 years.

Synthetic Central Flow Substitute Valves—Synthetic substitutes for duplicating the central flow orifice valve have been unsuccessful; relentless growth of fibroblasts soon produces a stiff, nonpliable valve with subsequent valve dysfunction.

Lateral Flow Valves

Ball Valve (simple)—Of all valves based on a lateral flow design, the ball valve has had the greatest clinical success. Originally constructed of a heavy acrylic cage with a metal poppet, this type was later refined to a lightweight metal cage with a Silastic poppet and with many subsequent significant modifications. The Starr-Edwards type, having gained greatest acceptance by surgeons, became a standard for comparison with other models. It, too, had undergone many modifications, including a change in the method of cure of the Silastic poppet which also affected all ball valves using such a poppet. Earlier Silastic produced swelling of the ball with consequent valve dysfunction or poppet fragmentation. Although ball variance has not been completely eliminated, its incidence is now nearly negligible in follow-ups of up to 7 years.

The major problem with this type of prosthesis has been systemic thromboembolism (even with chronic anticoagulant therapy). This is a drawback with all prosthetic valves. Fortunately, the multiple modifications in fabrication of this valve have had a salutary effect on thromboembolism incidence. The current model (1260 aortic valve) appears to be associated with only a 5 per cent, 3-year incidence, compared with a 20 per cent, 3-year incidence a decade ago.

Cloth-Covered Prostheses—Since its introduction in 1967, the Starr-Edwards cloth-covered type of prosthesis has appeared to decrease thromboembolic episodes. But problems with cloth wear along the struts and at the seating ring necessitated many changes, and there is a paucity of follow-up data on the current types. (A Braunwald-Cutter model, introduced in 1970, has recently been withdrawn from the market because of hemolysis and accelerated poppet wear associated with poppet embolism.)

Track Valves—A modified Starr-Edwards fabric-covered model designed to prevent cloth wear is now being assessed clinically. The new design protects the cloth at the critical areas in contact with the ball.

Disc Valves—Integral to the design of a ball valve is a cage of sufficient length to permit adequate excursion of the ball from the inlet orifice. Although easily accommodated in the aortic position, it has been considered unsatisfactory for some atrioventricular valve replacements. To obviate its drawbacks, a low-profile disc valve with a flat lens suspended in an abbreviated cage was devised, but initial enthusiasm waned as the disc design failed to demonstrate any superiority in the atrioventricular position.

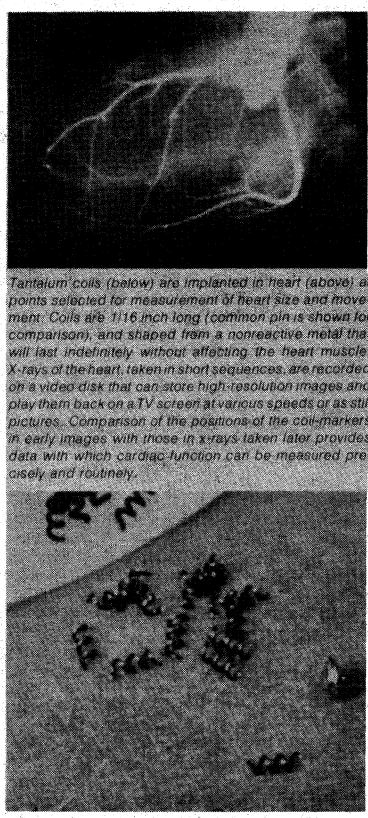
Semicentral Flow-Tilting Disc Valve—This type retains the low-profile contour but approaches the central flow concept, and consists of a free-floating lens in a partial Satellite cage. In the open position, it tilts 50° to 60° in the direction of blood flow. With an ultralight disc, this valve has an extremely favorable orifice-to-mounting ring ratio and thus appears to be well suited for pediatric patients or those with a diminutive aortic valve annulus.

The semicentral flow-tilting valve (Bjork-Shiley type) is also popular for use in adults and, to date, general problems have been few so far (except for thrombosis of the aortic valve prosthesis, which has occurred nearly exclusively in those with no or inadequate anticoagulant prophylaxis). Good control of long-term anticoagulation is clearly essential in patients with this type of valve implant, and although the wear characteristics thus far seem good, it remains to be seen if its performance will equal that of the ball-and-cage type. □

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Internal Heart Functions Are Mon

Precise and continuous monitoring of the heart's internal functioning, without discomfort to the patient, has been made possible by a technique that establishes mapping points in the heart muscle, then records their subsequent changes of position by x-rays shown via "instant replay" on a TV screen.



Tantalum coils (below) are implanted in heart (above) at points selected for measurement of heart size and movement. Coils are 1/16 inch long (common pin is shown for comparison), and shaped from a nonreactive metal that will last indefinitely without affecting the heart muscle. X-rays of the heart, taken in short sequences, are recorded on a video disk that can store high-resolution images and play them back on a TV screen at various speeds or as still pictures. Comparison of the positions of the coil-markers in early images with those in x-rays taken later provides data with which cardiac function can be measured precisely and routinely.

The new technique is being used at the Stanford University Medical Center to follow the progress of patients after they have undergone surgery for heart transplants, coronary artery bypass graft, or valve replacement. Within an hour after x-rays are taken, physicians can determine how well each segment of the heart is working, how much blood the heart contains, how much blood it is pumping, and the length of contraction time.

The first step in the system is taken while the patient is on the operating table: the surgeon implants minute metal coils at appropriate places in the muscle fiber. The coils are made of tantalum, a nonreactive substance that can last a lifetime.

Later, x-rays taken in 3-second sequences are recorded on a video disk—a magnetized, coated, 14-inch aluminum disk that can store high-resolution images and replay them at normal speed, in slow-motion, in reverse, or one frame at a time. Pulsed signals from angiograms also can be played repeatedly, making them available for prolonged analysis when a computer is linked to the disk.

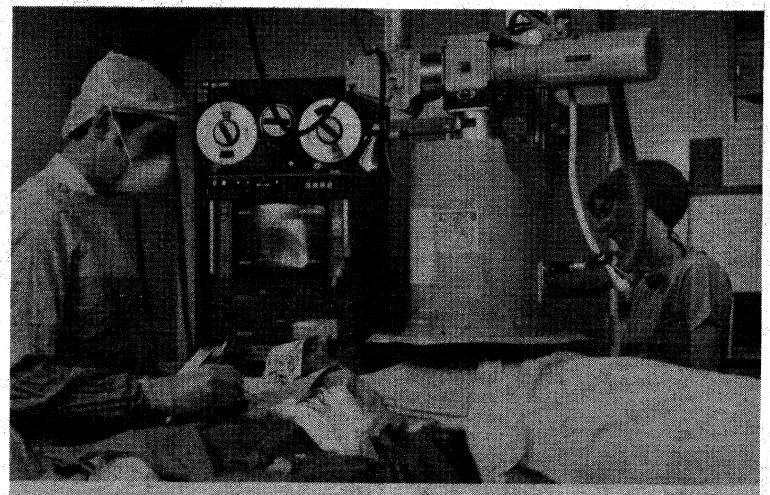
By comparing sets of x-rays with those taken later, researchers can determine if the heart is getting larger or smaller. They can also discover whether a transplanted heart is undergoing rejection, and how fast the heart is deteriorating or improving.

In addition, the technique will be used to study the short-term recovery process after surgery, to investigate the effects of drugs and exercise on the hearts of patients, and to examine the mechanisms by which the rejection process and anti-rejection drugs affect the transplanted heart.

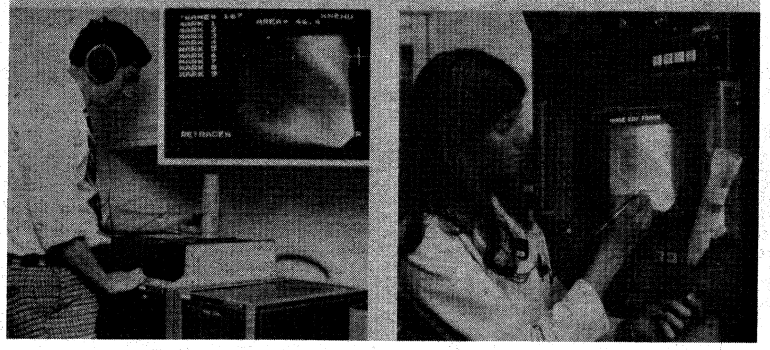
The system has been employed with considerable success for about two years on more than 50 heart-transplant, coronary artery-bypass, and valve-replacement patients at Stanford University Hospital, by a research team composed of Dr. Neil Ingels and engineer George Daughters of the Palo Alto Medical Research Foundation, and Drs. Edward Stinson, Edwin Alderman, Lewis Wexler, Philip Oyer, Randall Griepp, Jack Copeland, and Norman Shumway of Stanford.

The video disk, but not necessarily the metal coils, are also used to analyze the blood-pumping efficiency of hearts of patients in the Mayo Clinic's cardiovascular research ward. □

itored by Noninvasive Technique



"Instant replay" of heart's action appears (above) on TV monitor as team at Stanford University Medical Center performs a cardiac catheterization. X-ray image had been stored on Ampex magnetic video disk. At left, below, Dr. Edwin Alderman uses ultrasonic listening device to check the positioning of the magnetic recording heads; inset shows TV display of x-ray image of heart and coil-markers. Lab technician Alison Ferris (below, right) traces with a light pen the borders of a heart's pumping chamber.



Diagnosis and Treatment

By H.J.C. Swan, M.D. and William Ganz, M.D.



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Dr. Ganz is Senior Research Scientist, Department of Cardiology, Cedars-Sinai Medical Center, and Professor of Medicine, University of California, Los Angeles.

The balloon flotation catheter, developed in recent years, has greatly facilitated right heart and pulmonary artery catheterization. It provides important physiologic data for diagnosis and treatment of patients who are seriously ill, not only from myocardial infarction, but from other diseases as well.

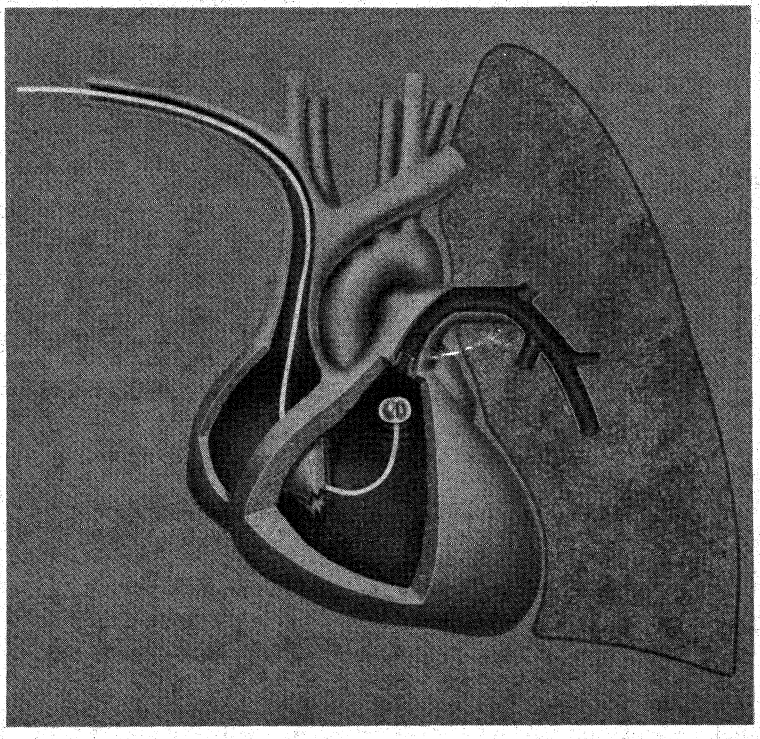
Balloon flotation catheters—now widely accepted as reliable and relatively safe—are finding extended use for diagnosis and treatment of the critically ill, not only in larger institutions but also in the community hospital.

They can be used at the bedside without fluoroscopy to provide highly relevant circulatory data, and to permit a rational selection of therapy with, at the same time, assessment of patient response. The applications of balloon flotation catheters have come to include the accurate management of fluid volume control in patients with bacteremia, acute pancreatitis, and severe blood loss.

These catheters have found use also in the coronary care unit, the medical intensive care unit, the postsurgical recovery unit, the intraoperative cardiovascular surgical suite, the neurosurgical and obstetric areas, and in anesthesiology. They are of importance as well in the diagnostic cardiac catheterization laboratory, the pulmonary intensive care unit, and the pulmonary function laboratory. They have been of value in outpatient facilities including exercise laboratories and "noninvasive" laboratories.

The Swan-Ganz catheter is relatively simple and does not require the special technical skill usually needed for manipulation of the traditional catheter. Further, it is a procedure of short duration and involves a low incidence of ventricular arrhythmias, as

With the Balloon Catheter

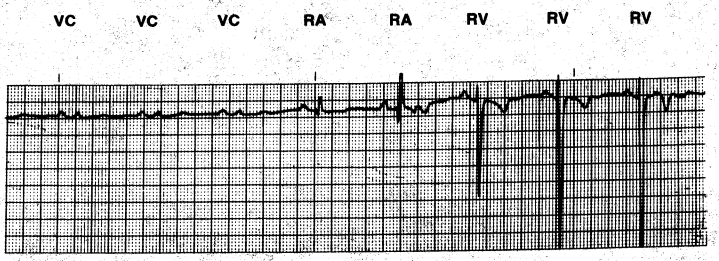


compared to traditional catheterization. Nevertheless, its use does require training and practice. We recommend that every hospital prepare a small group of physicians, nurses, and technical personnel who will be truly familiar with use of the apparatus and ready to offer their services to other physicians on request. The results in information gained could help advance diagnosis and treatment on a broad front.

We developed the flow-directed catheter in the late 1960s in connection with myocardial infarction studies in seriously ill patients. We sought a method for placement of catheters within the pulmonary artery which would provide prompt and reliable passage; would permit manipulation without use of the fluoroscope; and would not cause ventricular arrhythmias. A small inflatable balloon, mounted at the tip of a highly flexible cardiac catheter, was developed and tested, first in animal experiments and later in the clinical area. By 1970 we had reported on its use in 100 patients.

The device consists of a double lumen catheter of about 1.5 mm outside diameter. The smaller lumen is approximately 0.4 mm in diameter and is used to inflate a latex balloon at the catheter tip with either carbon dioxide or air. After the catheter has been introduced into the circulation by cutdown or percutaneous technique, the tip is positioned in the

ECG LEAD FROM CATHETER TIP DURING INSERTION



VC *Catheter tip in vena cava (note: low signal amplitude with "P" and "QRS" approximately equal)*

RA *Catheter tip in right atrium (note: larger amplitude and* positive *direction of "QRS"; "P" may be larger than shown here, depending on position of catheter within atrium).*

RV *Catheter tip in right ventricle (note: small "P" with large* negative *direction "QRS").*

Caution: For electrical safety, it is mandatory that these intracavity ECG's be monitored with suitably isolated, preferably battery powered, equipment.

superior or inferior vena cava or high right atrium. The balloon is then inflated, providing a relatively solid (nonliquid) element which is directed by the flowing stream of blood. The inflated balloon, with a diameter of about 11 mm, is flow-guided through the right atrium and tricuspid valve into the right ventricle. From there it finds its way into the main pulmonary artery and into a branch of the pulmonary artery. When it reaches a pulmonary vessel slightly smaller in diameter than the inflated balloon it stops, much like a pulmonary embolus. When fluoroscopically controlled, the balloon guidance system will flow from the right atrium to the pulmonary artery in about 10 to 20 seconds.

When the balloon is deflated, the catheter shaft will recoil slightly into a larger pulmonary artery since the flowing blood now does not have the "mass" on which to act. When it is slowly and briefly inflated again, it has impact once more on a smaller artery and permits a wedge measurement—one of the most significant readings provided by the catheter.

The balloon is important in avoiding endocardial irritation and arrhythmias: When it is inflated for passage through the right ventricle, the balloon protrudes over the catheter tip, which maintains its place in "the hole in the balloon doughnut."

Pressure Tracings Needed

The apparatus requires manometers and display units as well as electrocardiographic monitoring. A fluoroscope, although it can be dispensed with at the bedside, facilitates effective placement in some cases. Pressure tracings are needed, not only to assist in positioning the catheter tip, but to provide data useful in diagnosis. An increase in respiratory cycle pressure variations on the tracings indicates that the tip has reached the thorax. The balloon is then inflated and a recording made of the maximum and minimum pressures in the atrium. As it enters the tricuspid valve and passes into the right ventricle a similar recording is made. Pressure tracings then show the pulmonary artery pressure contour, and advancement is continued until a pressure is identified which approximates pulmonary artery diastolic levels. The balloon is then deflated and phasic pulmonary artery pressure should reappear. The pressure levels in the pulmonary artery are then measured again.

The balloon flotation catheter may be left in place up to a maximum of 48 hours in management of the critically ill, but re-use is questionable because cleaning and sterilization may cause deterioration of the latex balloon. The balloon must always be deflated for withdrawal of the catheter.

The flow-directed catheter is particularly useful in measuring the pressure in the pulmonary veins when wedged with its balloon. This reading is of critical importance in clinical practice in that it provides information on the level of pulmonary congestion and on the transfer of fluid at the pulmonary capillary level. In addition, pulmonary venous pressure relates closely to left atrial pressure and, in the absence of mitral valve disease, to left ventricular diastolic pressure. For clinical purposes the mean wedge pressure provides highly relevent data of practical significance.

Most Valuable Parameter

The principal data obtained from using the catheter include the filling pressures of the right and left ventricle, $\Delta P/\Delta T$, and the cardiac output. Of these, left ventricular filling pressure appears to be a most valuable parameter of cardiovascular function on which to base therapeutic decisions and on which to evaluate their effectiveness.

A knowledge of cardiac output as a basic component of cardiovascular function has always been accepted as important by the cardiovascular physiologist. However, many clinicians have doubted the value of precise data on cardiac output levels and changes. Now that this variable can be measured with the balloon flotation catheter without particular difficulty or increased hazard to the patient, the significance of such measurements in clinical practice is becoming recognized more widely.

Several definable states of cardiovascular function can now be identified in pathophysiologic terms, and specific therapies can be indicated on a hemodynamic basis. For instance, normal cardiac output associated with a normal left ventricular filling pressure does not require specific cardiovascular therapy, and indicates that abnormal symptoms or signs suggestive of cardiovascular dysfunction may have another basis. Changes may occur later, and the comparison of data can be significant.

From the basic balloon flotation catheter have come other developments permitting more complex measurements. The thermodilution catheter incorporates a thermistor which allows measurement of cardiac output by the thermodilution principle.

Pediatric catheters have permitted catheterization of the heart with less risk of perforation or major arrhythmias in critically ill infants with congenital heart disease. And special balloon-tipped catheters are used for rapid bedside temporary pacing. Some surgeons have predicted that the balloon flotation catheter ushers in an era in which monitoring of critically ill surgical patients will become accepted as being indispensable. □

Diagnostic Sequence

By Arthur A. Sasahara, M.D.

Dr. Sasahara is Chief, Department of Medicine, Veterans Administration Hospital, West Roxbury, Mass.



Embolism is the most common cause of pulmonary death among hospitalized patients; and when its secondary effects are also taken into account, it may well be the most common cause of all deaths in the hospital.

Untreated, pulmonary embolism is fatal in 25 to 35 per cent of cases. But the mortality rate plummets to between eight and ten percent with prompt diagnosis and effective treatment. Unfortunately, the diagnosis is not immediately apparent in many instances, because the clinical picture of pulmonary emoblism is largely nonspecific.

A practical diagnostic sequence can be formulated to assess patients with suspected emboli, even in situations where definitive, sophisticated diagnostic techniques are not readily available.

It is a paradox that while medicine and surgery are taking great, if not giant steps forward, the incidence of thromboembolism is on the rise. Undoubtedly, the increase is related to that progress, in that older and sicker patients are undergoing more complex operations and are confined to bed for longer periods. Thus, the stage is set for the formation of thrombi and their release as emboli.

Embolism is certainly regarded as the most common cause of pulmonary deaths among hospital inpatients. In fact, if its secondary effects are taken into account, embolism may well be the most frequent cause of all in-hospital deaths. Each year, about 142,00 patients (four to five per 1,000 inpatients) die of pulmonary embolism in American hospitals. And another 568,000 (20 per 1,000 inpatients) suffer nonfatal embolic episodes.

To some extent, the grim mortality figures are the result of slow or missed diagnosis. Without treatment, pulmonary embolism is fatal in 25 to 35 per cent of cases. But with prompt diagnosis and effective treatment, the mortality is considerably lower—eight to ten per cent.

The obvious implication is that early diagnosis and immediate treatment greatly improve the prognosis. Early recognition is not always easy, however. Even in patients with segmental and larger vessel occlusion, pulmonary embolism is diagnosed infrequently.

One reason is that the symptoms and signs are usually nonspecific—attributable to any other cardiopulmonary problem. Another is that laboratory tests contribute little to diagnosis because they, too, are nonspecific for pulmonary embolism. Determinations that once seemed promising—serum LDH, GOT, and bilirubin—have been found to be of little use in diagnosing pulmonary embolism. Since neither clinical nor laboratory evidence points specifically to thromboembolic disease, appropriate diagnostic studies often may be delayed until after another embolism occurs.

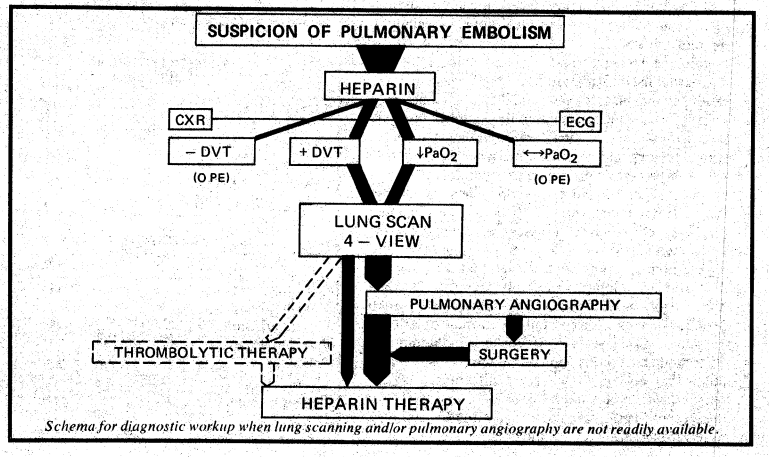
As in so many clinical situations, the essential first

for Pulmonary Embolism

step toward diagnosis is the physician's index of suspicion. Once he suspects that his patient has suffered pulmonary embolism, he can carry out a number of relatively simple tests to support the presumptive diagnosis or rule it out. At the same time, he can begin prophylactic treatment with low-dose heparin.

One routine measure that yields valuable information is the plain chest x-ray. In a cooperative study conducted by the National Heart and Lung Institute (NHLI), the two most common features of chest films in patients with pulmonary embolism were high diaphragm on the side where the embolus was lodged, and pulmonary consolidation. Many of the patients had both. In the clinical context of cardiopulmonary distress, then, either or both of these features on the plain film should suggest the strong possibility of pulmonary embolism, infarction, or both. To confirm the diagnosis, perfusion lung scanning should be performed immediately.

In the NHLI study, data were obtained from 14 institutions that followed a uniform study protocol, in which the presence of pulmonary embolism was established by pulmonary angiography. Patients were grouped according to the extent of occlusion: Those who had two or more lobar arterial occlusions were classified as having suffered massive embolism; where there was lesser occlusion, the em-



Schema for diagnostic workup when lung scanning and/or pulmonary angiography are not readily available.

bolism was classified as sub-massive. It must be pointed out that all the patients in the NHLI study had serious pulmonary vascular obstruction (average, half of one lung), since the criteria for eligibility excluded those with small pulmonary embolism.

Massive or Submassive Embolism

Symptoms such as pleural pain and syncope, and physical findings of tachypnea, rales, loud pulmonary second sound, and cyanosis were considered in relation to the massivity of the embolism. The only symptoms that could be used to distinguish between massive and sub-massive embolism were pleural pain (associated with sub-massive embolic occlusion) and syncope (indicating massive embolism). As for physical signs, only loud pulmonary second sound, S_3 or S_4 gallop sounds, and cyanosis were pertinent. These findings more often reflected massive than sub-massive embolism. Only one-third of the patients had clinical evidence of thrombophlebitis.

The two definitive diagnostic procedures — perfusion lung scanning and selective pulmonary angiography—often cannot be performed at the critical time because most hospitals are not equipped to provide these services 24 hours a day, seven days a week. However, diagnostic evaluation for pulmonary embolism is incomplete without lung scanning, unless angiography has been performed.

If the lung scan is abnormal, confirmation by pulmonary angiography is very desirable, especially in patients with prior cardiopulmonary disease. But in certain patients, treatment may be started without angiographic confirmation when the lung scan shows lesions that have a "high probability" of being due to pulmonary embolism. Such patients are young persons without pre-existing cardiopulmonary disease, whose history and physical findings are compatible with the presence of pulmonary embolism.

When scanning and angiography have to be delayed, simpler tests may suffice for a provisional diagnosis. One such test is measurement of the arterial oxygen tension, which can be of value in excluding pulmonary embolism provided that certain modifications are adopted. First, the oxygen electrode must be calibrated with standard gas before each use. Second, the lower limit of normal for arterial oxygen tension should be established at 90 mm Hg, since higher tensions are rare among patients with pulmonary embolism. Therefore, if arterial oxygen tensions are to be used for diagnosis, calibration of the electrode prior to each measurement is essential to assure accuracy.

NHLI UROKINASE PULMONARY EMBOLISM TRIAL

Presenting Symptoms

Symptom	Prevalence (%) All	Massive	Submassive
Dyspnea	81	79	83
Pleural pain	72	62	84*
Apprehension	59	61	56
Cough	54	50	60
Hemoptysis	34	27	44
Sweats	26	27	24
Syncope	14	22*	4

*Differences are significant.

UPET: Presenting Signs

Signs	Prevalence (%) All	Massive	Submassive
Rales	53	50	57
↑P_2	53	60*	44
Phlebitis	33	42	21
S_3, S_4	34	47*	17
Sweating	34	41	24
Cyanosis	18	28*	6
↑Respiration (>16)	87		
↑Pulse (>100)	44		
Fever (≥37.8)	42		

*Differences are significant.

Laboratory Findings

Test	Per cent Patients
LDH ↑	37
GOT ↑	26
HCT <35 per cent	23
BUN ≥20	18
Platelets (<200,000)	18
Bilirubin (>1mg/100 ml)	10

Frequency of CXR Abnormalities

Abnormality	Per cent Patients
Consolidation	41
High diaphragm	41
Pleural effusion	28
Plump pulmonary arteries	23
Atelectasis	20
LV ↑	16
Focal oligemia	15
RV ↑	5

Also useful as aids to the diagnosis of pulmonary embolism are various procedures for assessing the patency of the deep veins of the legs. A strong association between pulmonary embolism and deep vein thrombosis was documented recently by Sevitt, who found deep vein thrombosis in virtually all his patients who suffered fatal pulmonary embolism. Similarly, another physician, who assessed thrombosis using the radioactive fibrinogen test, found pulmonary embolism to be present only when radioactive clots were detected in the deep veins.

In the author's own experience, deep vein thrombosis was detected in 95 per cent of patients with pulmonary embolism confirmed by selective pulmonary angiography. Contrast ascending phlebography is more definitive for detecting venous thrombosis than the simpler electrical impedance phlebography. However, its usefulness is limited because it is not readily available. Another limitation is that contrast ascending phlebography is an invasive test. Therefore, non-invasive procedures— electrical impedance phlebography and Doppler ultrasound—have been used with increasing frequency. For detecting deep vein thrombosis, impedance phlebography appears to be more sensitive and more specific than Doppler ultrasound.

Based on these non-invasive, readily available tests, a practical diagnostic sequence can be formulated to assess the many clinical situations in which perfusion lung scans and selective pulmonary angiograms cannot be obtained immediately.

If Pulmonary Embolism Is Suspected

When pulmonary embolism is suspected, an immediate "covering dose" of 7,500 to 10,000 units of heparin should be given intravenously; a routine chest x-ray and an electrocardiogram should be obtained; arterial oxygen tension should be measured; and patency of the deep veins should be assessed.

If an accurately performed arterial oxygen tension measurement is normal (above 90 mm Hg), it is unlikely that pulmonary embolism is present. If phlebography shows the deep veins of the legs to be of normal patency, pulmonary embolism is also unlikely. Indeed, if the impedance measurement is normal in these suspected patients, our experience indicates a 90 per cent probability that pulmonary embolism does not exist. However, if the arterial oxygen tension is low or the deep veins are obstructed, perfusion lung scanning should be performed to confirm the presence of an embolism.

In this clinical setting, the impedance measurement is specific for detecting thrombosis in the deep veins, where pulmonary emboli originate. Therefore, this test has far greater diagnostic value than the arterial oxygen tension measurement. In our group of patients, angiography confirmed the presence of pulmonary embolism in 90 per cent of those whose impedance phlebograms indicated deep vein obstruction.

Most patients should have angiographic confirmation of pulmonary emboli, particularly if some form of surgical intervention is contemplated. And in the vast majority of patients, intravenous heparin therapy should be continued, provided that anticoagulation is not contraindicated.

In the future, heparin will probably be replaced by thrombolytic agents. Because such compounds are superior to heparin in rapidity of action and completeness of clot resolution, they are likely to become the preferred drugs for treatment of pulmonary embolism. However, since the thrombolytic agents are not yet available for general use, heparin remains the drug of choice. □

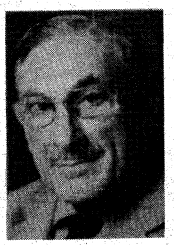
Practice Procedures

Detecting Pulmonary Embolism

Perfusion lung scanning and selective pulmonary angiography are the definitive diagnostic procedures to confirm or rule out pulmonary embolism, but in many situations, they are not immediately available. When pulmonary embolism is suspected, the following diagnostic sequence is a practical means of prompt diagnosis and successful management:

- Give a "covering dose" of 7,500 to 10,000 units of heparin intravenously.
- Obtain routine chest x-ray and ECG.
- Measure arterial oxygen tension. When tension is normal (90 mm Hg or higher), pulmonary embolism is unlikely.
- Assess deep veins for thrombi by contrast phlebography or impedance phlebography. If the impedance measurement is normal, the probability is 90 percent that there is no pulmonary embolism.
- Perform perfusion lung scanning if arterial tension is reduced or deep veins are obstructed.
- Confirm the presence of pulmonary emboli by angiography.
- Continue intravenous heparin therapy in patients with confirmed pulmonary emboli, unless anticoagulation is contraindicated.

Minor Infarctions:



Dr. Sampson is Clinical Professor of Medicine, University of California, San Francisco; and past president of the American Heart Association

The intermediate syndrome of acute coronary insufficiency has been described in ill-defined, imprecise terms. It encompasses a broad spectrum of signs and symptoms, with wide variations in prognosis. Distinction between the benign and more serious forms of this syndrome is important in charting the course of therapy.

Almost 40 years ago, two papers (one by Feil and the other by Eliaser and myself) were published describing the clinical patterns of patients who had experienced recurrences of angina pectoris that deviated from prior attacks. Importance was attached to these new episodes because about half of the patients developed typical myocardial infarction within a half-day to a month, despite restriction of their physical activity. At that time, it had already been well established that a number of anginal attacks may precede myocardial infarction.

Many of the patients recovered spontaneously without ill effects, while others developed serious myocardial infarctions with a relatively high fatality rate. Still, the tendency has been to label all such patients with one diagnosis: intermediate coronary insufficiency syndrome.

Since myocardial revascularization, largely by aorto-coronary bypass, has come into widespread use, the challenge to the physician is to distinguish between the benign and the more serious forms of such deviant anginal attacks. This distinction may indicate which patients might best benefit from surgical revascularization, and which run the highest operative risks.

Criteria Are Limited

Unfortunately, prognostic criteria are few and diagnostic criteria are inadequately defined. Various degrees of coronary occlusion can develop asymptomatically. Even severe narrowing of all three coronary vessels may give no warning or may produce only minor, stable angina until a serious myocardial infarction suddenly occurs. Conversely, coronary angiography may reveal relatively minor occlusive disease or no evident arterial narrowing in patients who have repeatedly required protective hospital admissions for severe "crescendo" spontaneous angina or angina of effort. Patients with Prinzmetal's variant of angina often present such deviations from the expected occlusive disease, and this type of attack has been attributed to coronary arterial spasm.

Different authors have offered different diagnostic criteria. To a large extent, these criteria depend on what statistical plan is to be used for analysis of a specific collection of cases. Another factor that influences the specified diagnostic criteria is the physician's sphere of interest. For example, a cardiologist who is interested in the natural history of the disease, in noninvasive diagnostic study, and in "medical" care (i.e., rest during a presumptive criti-

What to Look For

By John J. Sampson, M.D.

cal period and treatment with anticoagulant drugs and beta-adrenergic-blocking agents) will not set the same criteria as a cardiovascular surgeon. The surgeon who is interested in revascularizing the myocardium by performing an aorto-coronary bypass will consider surgical mortality and postoperative benefits and complications. Still other criteria would be set by investigators who are studying the pathogenesis of the syndrome, or the pathology that can be found at autopsy. The latter is especially valuable when it is correlated with the associated clinical patterns.

The "intermediate syndrome" is generally recognized as coronary insufficiency of a severity intermediate between myocardial infarction and chronic angina pectoris with a consistent pattern. And there has been general agreement that the coronary circulation is insufficient to consistently meet the metabolic needs of the myocardium at rest, although it is generally sufficient so that myocardial infarction does not occur.

However, evidence has accumulated that these neat definitions are not always entirely valid. For example, 20 years ago an autopsy study indicated that when the onset of prolonged or new pain points to an important new occlusion, the development of necrosis depends on both the degree and the duration of myocardial ischemia. The intermediate syndrome, then, may reflect a state of jeopardy that culminates in small or large areas of necrosis when the compromised blood supply can no longer support the vitality of the myocardium.

Syndrome Subgroup Sought

Such necrosis, sometimes called a "silent" infarction, may not cause pain. However, it does produce the delayed rise of serum enzymes in 2 to 11 days, the electrocardiogram changes, the altered cardiodynamics, and other signs that are characteristic of myocardial infarction. Because these changes occur with some frequency, it is necessary to account for them by defining a more serious subgroup of the intermediate syndrome.

One recent report indicated that among 84 patients who had typical prolonged infarction pain, only 26 had moderately elevated serum CPK-MB levels, which presumably were detected because of fortuitous timing of the determinations. However, their ECG patterns showed only S-T depressions or elevations and inverted T-waves. Another team of investigators had previously reported similar elevations of SGOT.

Myocardial Scintigrams as Evidence

The most recent evidence of minor infarctions, presented in several papers published over the past year, came from myocardial scintigrams taken on patients with the intermediate syndrome. In these studies, radioactive agents such as 99M-technetium stannous pyrophosphate, 201-thallium, 43-potassium, 81-rubidium, and 129-cesium were either relatively concentrated or reduced in the infarcted areas and appeared on the scintigraphic image.

Definition and identification of a more seriously threatened subgroup can pay off with a reduced surgical mortality rate. For example, in a study of 111 intermediate syndrome patients who had the same pattern of pain and ECG changes in the S-T segment and T-wave, 16 patients had elevated serum levels of CPK-MB. These 16 patients did not undergo bypass surgery, with the result that operative mortality among the total study group was reduced from the expected figure of more than 10 per cent to an actual death rate of only four per cent. In contrast, in another report, there was 80 per cent mortality in a small series of patients with impending or extending myocardial infarction who were subjected to emergency aorto-coronary bypass without exclusion of those at high risk.

In patients who were followed for long periods, several reports have indicated that sudden death is not infrequent. In one group of 100 patients, 22 died suddenly in one year.

It may be erroneous, then, to assume that "ischemic" ECG changes in T-waves and the S-T segment are benign patterns. Indeed, a number of investigators have noted a tendency toward failure to recognize that these were the only ECG changes in autopsy-proven cases of myocardial infarction with one or more arteries occluded.

Basically, the intermediate syndrome can be accepted as a form of acute coronary insufficiency that does not meet the criteria of a major myocardial infarction; but it is important to recognize the existence of this syndrome as a subgroup with evidence of minor myocardial necrosis and a hazardous prognosis.

nation encompasses the zone between chronic angina or no chest pain, through noninfarcted ischemic myocardium, to minor infarction as discussed earlier. Those patients with minor infarction must be classed as a subgroup with more serious prognosis.

• *Unstable angina* has recently become a popular term. However, it has the shortcoming that many

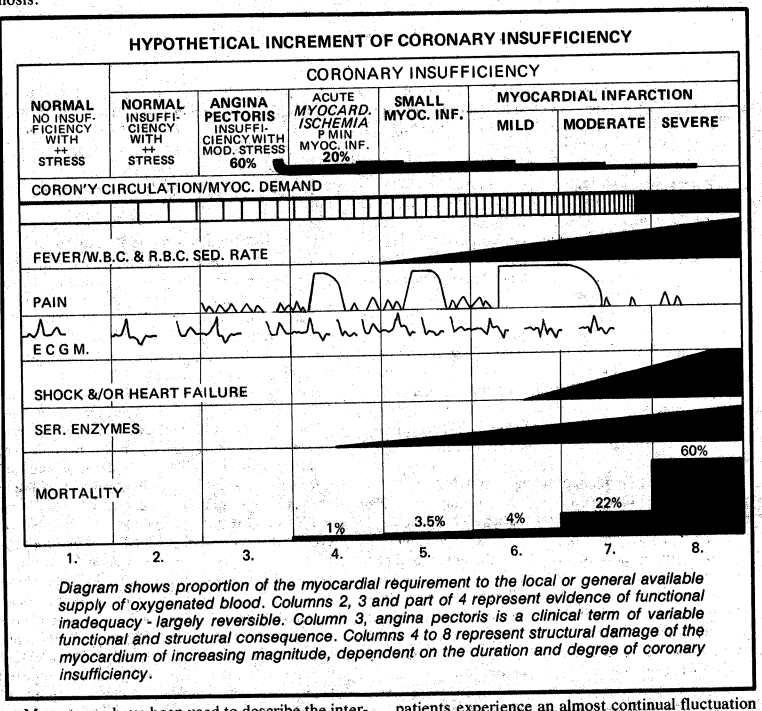


Diagram shows proportion of the myocardial requirement to the local or general available supply of oxygenated blood. Columns 2, 3 and part of 4 represent evidence of functional inadequacy - largely reversible. Column 3, angina pectoris is a clinical term of variable functional and structural consequence. Columns 4 to 8 represent structural damage of the myocardium of increasing magnitude, dependent on the duration and degree of coronary insufficiency.

Many terms have been used to describe the intermediate syndrome, and some of them require comment:

• *Acute coronary insufficiency* (or acute myocardial ischemia and acute coronary heart attack). This term is probably accurate, but it requires further definition because it is too broad. It could include a spectrum of disease ranging from relatively new ischemia without myocardial damage, through minor infarctions, to major infarctions.

• *Intermediate coronary syndrome.* This designation encompasses the zone between chronic angina or no chest pain, through noninfarcted ischemic myocardium, to minor infarction as discussed earlier. Those patients with minor infarction must be classed as a subgroup with more serious prognosis.

• *Unstable angina* has recently become a popular term. However, it has the shortcoming that many patients experience an almost continual fluctuation in the frequency, precipitating factors, and intensity of recurring pain, but they show no objective evidence of myocardial damage or myocardial infarction over periods of many years.

An example is a 71-year-old man who has suffered four mild myocardial infarctions. Over the past four years, he has had periods of one to three weeks with angina only on effort (e.g., rapid walking), interspersed with isolated spontaneous attacks lasting 30 to 45 minutes, recurring for four to five days, and

relieved only with repeated doses of nitroglycerine. Another patient—a 63-year-old woman—had three minor myocardial infarctions in six years. She has spontaneous episodes of chest pain in the evening or night, lasting a half-hour to an hour despite the use of two to three sublingual nitroglycerine tablets. These attacks recur six to ten times a year. No changes are found in the ECG or serum enzymes, and she returns to moderate physical activity (e.g., climbing low hills) without discomfort.

• *Preinfarction angina* is an inaccurate term in two respects: many of the attacks prove to be minor infarcts; and 50 to 80 per cent of patients never develop myocardial infarction.

• *Accelerated angina pectoris* (or prolonged anginal pain) is a generally descriptive term, but it does not cover the variations in clinical patterns.

• *Prodromata in acute myocardial infarction; premonitory period of myocardial infarction; impending myocardial infarction; impending coronary artery occlusion; and preliminary pain in coronary thrombosis.* These five terms emphasize the hazard of an impending myocardial infarction, but they do not account for the many cases where immediate small infarctions have already occurred, and the many more in which infarction never develops.

The duration of this premonitory, intermediate coronary syndrome is not given detailed attention in many reports. Four weeks would seem to be a rational limit of duration of the acute episode, but the physician must take into account the dynamic modifications that result from serial angiographic and scintigraphic studies, as well as the concentration and clearance of radioactive indicators in areas of infarction.

A four-week duration of the syndrome is evident and expected after aorto-coronary bypass surgery, because of the alteration of collateral flow, and because of the possibility of acute thrombotic and presumably atherosclerotic advance of the arterial occlusion. A surprisingly rapid advance of atherosclerosis has been observed on serial coronary angiograms; and such progression is seen more frequently after aorto-coronary bypass than during the course of medical therapy. Although many authors suggest a four-week duration of the intermediate syndrome, others believe the limit may be six weeks to two months.

It is important to recognize that the acute coronary insufficiency is a labile process which may or may not advance to serious infarction. The clinical evidence may not indicate that a myocardial infarct, generally minor, often exists at the onset of this syndrome. Therefore, it is the physician's responsibility to carry out without delay, diagnostic procedures (e.g. radioactive scintigrams) that will reveal the myocardial lesion.

In identifying these patients with suspected or proven minor infarct, the same care should be applied as if they presented with the clinical and laboratory evidence of a major infarction.

The importance of accurate diagnosis is that the presence of minor infarcts may modify the decision to perform an aorto-coronary bypass because of increased operative risk. □

Practice Procedures

Criteria for Diagnosis

The criteria for diagnosis of intermediate coronary insufficiency have varied in extent, and to some degree in content, from one author to another. But all definitions include the elements listed below:

1. *New cardiac symptoms, or recurrence of symptoms after freedom from pain.* The symptoms may be anginal pain precipitated by effort or occurring spontaneously; episodes of spontaneous dyspnea; fatigability; giddiness; or faintness.
2. *Worsening of symptoms:* pain with increased severity, duration (over 15 or 30 minutes), frequency, or spontaneity (nocturnal or during rest); or pain precipitated by less stress than in previous attacks. Some authors classify patients within a more serious subgroup if they have spontaneous pain recurring persistently after a day of bedrest.
3. *Nitroglycerine or other nitrites have less effect or give no relief from chest pain.*

In most reports, the following objective findings are considered pertinent:

1. Electrocardiogram showing depressed or elevated S-T segment; flattened or inverted T-wave (likely to be transient for 1-24 hours); no new Q deflections.
2. Serum enzymes not rising to "diagnostic" levels. The fallacy of this criterion—especially with respect to CPK-MB isoenzyme — is the transiency of the elevation, so that if blood is not taken at the critical time, the peak level may be missed. CPK, SGOT, LDH (and LDH isoenzymes 1 and 2) may rise from base levels but remain within the customary maximum limits (40 units). However, on daily testing for three to five days, these enzymes may present a curve with over 10 per cent variation to the peak level.
3. Coronary angiogram revealing greater than 50 per cent occlusion in one, two, or all three major coronary arteries, with rare exception.

Cardiac Case History—

Tachyarrhythmia and the Wolff-Parkinson-White Syndrome

By Sanford S. Zevon, M.D.

A 22-year-old white male presented to the Emergency Room following the abrupt onset of palpitation. During the preceding three years, he had experienced two or three episodes which subsided spontaneously. There was no history of heart disease, murmurs, or significant cardio-respiratory symptoms. He had had no previous hospitalizations or operations. His parents and two siblings, ages 24 and 13, were alive and well, with no record of heart disease or palpitations.

Review of systems was noncontributory.

He did admit to using drugs (intravenous heroin and barbiturates) a few months prior to admission.

On physical examination, he appeared to be a well-developed, well-nourished, thin white male in no distress. Blood pressure 110/80, pulse 215 per minute, temperature 100°, respirations 20 per minute. The only pertinent finding involved the heart, which had a regular apical rate of 215 per minute. There were no murmurs, gallops, or rubs, and the lungs were clear.

An electrocardiogram revealed a regular, supraventricular tachycardia with a ventricular rate of 215 per minute. Intravenous metaraminol raised the blood pressure to 210/100 but failed to convert the rhythm. Deslanoside (0.8 mg.) was then given intravenously, and within 30 minutes he converted to normal sinus rhythm.

A 12-lead electrocardiogram was being taken at the time of conversion, and normal sinus rhythm was recorded in leads V3-V6 (Fig.1). The postconversion 12-lead electrocardiogram revealed a short P-R interval (0.11 sec. -0.12 sec.) and an inconspicuous but definite delta wave (Fig. 2). In addition, a change in the direction of the initial QRS vector was noted with the rhythm change. These findings supported the diagnosis of a pre-excitation phenomenon.

Conversion to normal sinus rhythm was short-lived, and within 30 minutes he reverted to another tachyarrhythmia, which had strikingly different electrocardiographic features (Fig. 3). The ventricular rate was 240 to 300 per minute and irregular, and wide bizarre QRS complexes were observed. Blood pressure at this time was 95/60, and he was diaphoretic. Although the rhythm resembled a ventricular tachycardia, the awareness of the existence of a Wolff-Parkinson-White syndrome led to the diagnosis of atrial fibrillation with aberrant conduction.

Intravenous propranolol (3 mg.) and edrophonium (10 mg.) were tried unsuccessfully, and he was finally cardioverted with a DC electric shock of 60 watts-seconds. The post-conversion 12-lead electrocardiogram was identical to the one taken after the initial conversion to sinus rhythm (Fig. 2).

The increased frequency of tachyarrhythmia in the WPW syndrome is well known and is related to the presence of anomalous atrioventricular connections. Paroxysmal atrial tachycardia is the arrhythmia most often observed. Less frequently, atrial fibrillation takes place.

The occurrence of both tachyarrhythmias in the same patient illustrates the function of the anomalous pathway in this syndrome. During the atrial tachycardia, activation of the ventricle occurs exclusively via the normal A-V conduction system and in a retrograde direction via the anomalous pathway to complete a re-entrant circuit. During atrial fibrillation, the ventricles are activated exclusively via the anomalous pathway resulting in a very rapid ventricular rate with wide QRS complexes that simulate ventricular tachycardia.

The likelihood of misdiagnosis should be em-

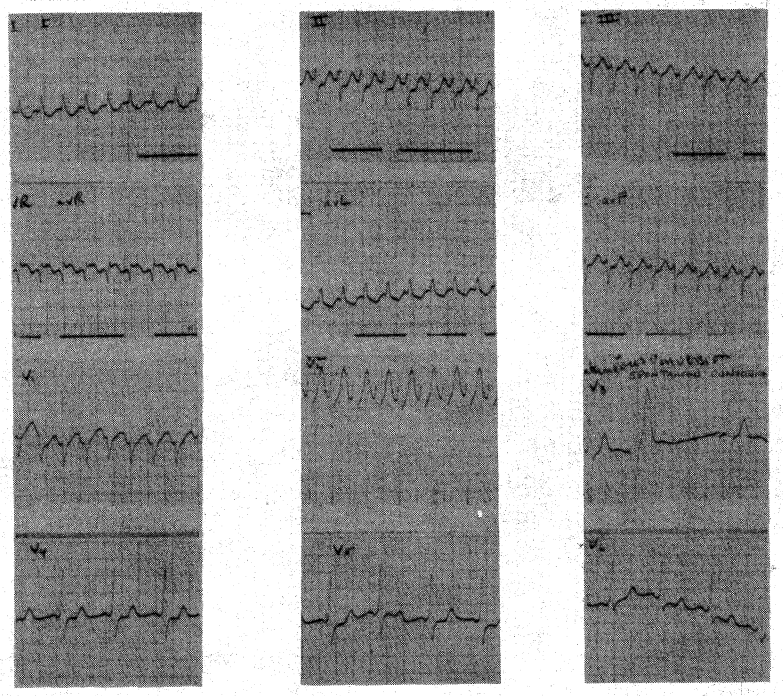
phasized. However, the combination of a very rapid (more than 240/min), irregular ventricular arrhythmia, particularly in younger individuals, that is unexpectedly well tolerated should suggest the correct diagnosis. Although this arrhythmia is usually well tolerated, prolonged attacks may lead to adverse hemodynamic consequences, and ventricular fibrillation and death may occur.

Pharmacologic therapy to slow the ventricular rate using intravenous procainamide or lidocaine may be tried; however, the treatment of choice for atrial fibrillation in the WPW syndrome that presents with a "pseudoventricular tachycardia" is cardioversion with DC countershock.

Digitalis should not be used in the presence of atrial fibrillation since it decreases conduction in the A-V node and enhances conduction through the accessory pathway. It may also shorten the refractory period of the accessory pathway and result in an increase in the ventricular rate.

For the treatment of atrial tachycardia, intravenous propranolol is very useful, and procainamide or lidocaine may also be tried. To prevent recurrent atrial tachycardia, quinidine or propranolol, or the combination of these two agents, has been most useful. □

FIGURE 1



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Cardiac Case History

FIGURE 2

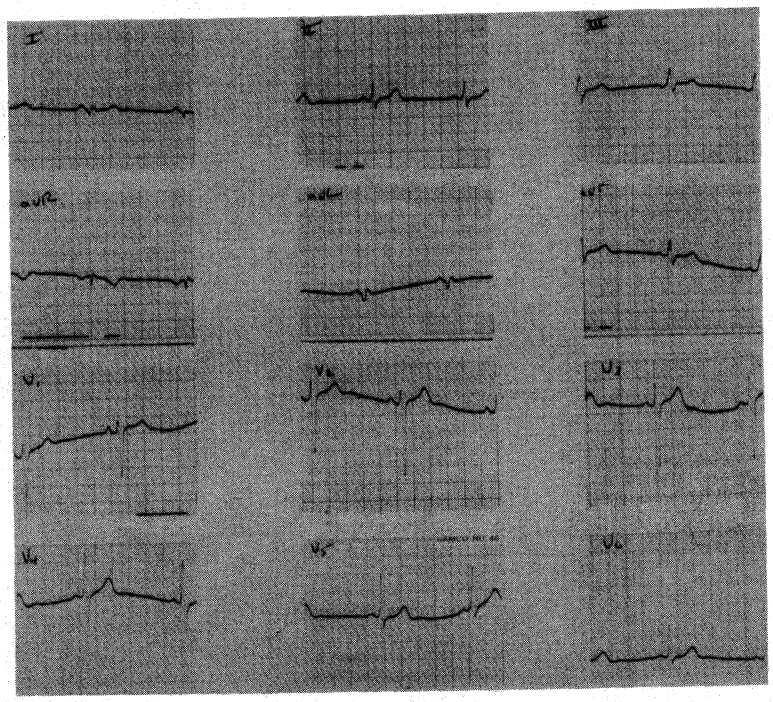
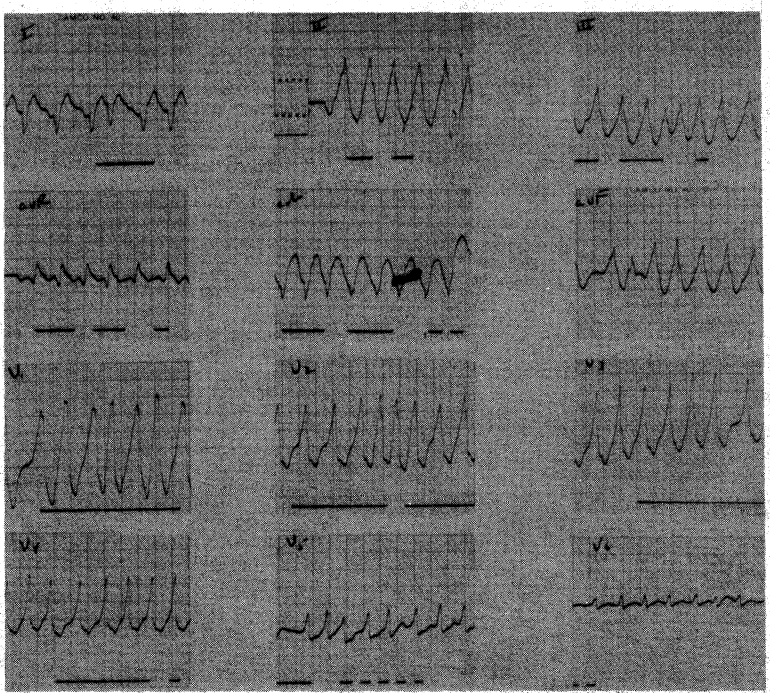
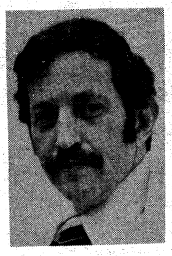


FIGURE 3



Anticoagulant Drugs:

By Louis M. Aledort, M.D.



Dr. Aledort is Associate Professor of Medicine, Mount Sinai School of Medicine.

This overview of the complex subject of anticoagulant therapy introduces a series of articles that will explore the roles of various drugs in the treatment and possible prevention of thrombosis. The modes of action of heparin, warfarin derivatives, and the new antiplatelet compounds will be discussed in relation to their effect on clotting mechanisms in the veins and arteries and also in relation to their use against a condition that is receiving considerable attention — diffuse, intravascular coagulation. The clinical indications and contraindications for each drug will be delineated, and the results of some of the latest research will be explained.

Thrombosis of arteries and veins is one of the foremost causes of disability and death. Perhaps the most effective way of treating and preventing these conditions is by the use of anticoagulants. And although heparin is effective and widely used, other drugs, such as the warfarin derivatives and compounds that act on the blood platelets, are coming into favor as more is learned about the seemingly endless complexities of blood coagulation.

Highly important has been the discovery that thrombotic disease is in reality two diseases, or perhaps three. One must separately consider venous

When to Use—and How

thrombotic disease and arterial thrombotic disease, because the pathogenesis of the two may be quite dissimilar. This is a critical distinction therapeutically because they require different modes of treatment and prevention.

Clotting Leads to Bleeding

There is, in addition, a relatively recent concept suggesting a third, basic type of coagulopathy, which is neither classical arterial disease nor venous disease, but one that affects primarily the large vessels, small vessels, and capillaries. It is diffuse, intravascular coagulation, which is a complication attendant to many acute, subacute, and chronic diseases that lead to either overt thrombosis and/or what we call paradoxical hemorrhage in the face of thrombosis. Whatever triggers the coagulation uses up clotting factors in the blood, and bleeding results from the "consumption" of those factors, hence the term, consumption coagulopathy. Thus, paradoxically, a primary clotting problem leads to a bleeding problem.

Now as far as we know there are three elements that keep that body from bleeding, or blood from clotting. One is the integrity of the blood vessels, and this has been much underrated in the past. Very little has been written, until the last decade, about the role of the vessel itself in preventing either hemorrhage or thrombosis.

The second element is the function of the platelet, a process that is being given more and more attention. If the blood vessel is the first line of defense against coagulopathy, then the platelet is the second line. Platelets course through the blood, and when a vessel is injured they leave the mainstream and, through a series of complicated physical, biochemical, and structural changes, form the primary hemostatic plug, which blocks the hole in the vessel.

The third element in the control of hemorrhaging and clotting is a group of various coagulation factors. For example, in order to maintain large blood vessel integrity during a surgical procedure, it is very important to the clotting process that an entire chain of plasmatic factors are present, and that there is a working system of the checks and balances that aid or inhibit coagulation.

Roles of Heparin and Warfarin

For some time we have been aware that hemorrhage is caused by lack of platelets or clotting factors, or by abnormalities in blood vessels. We have not been so aware of how these same elements may be leading to clotting, even in the best-known type of thrombosis—that is, thrombophlebitis. It has been thought that this is a classical disorder in which the blood-clotting factors are in some way activated and the clots formed, and that the only way to treat the acute condition is with heparin. For long-term prevention, warfarin derivatives were the choice.

We are now finding that there are people who have recurrent phlebitis or recurrent venous disease that may be resistant to those agents. We are beginning to

look at the elements of these clotting systems and at the platelets, and one can begin to dissect out the etiology of clotting in these vessels. In cases that are resistant, it may be that the initiating event is not the reaction of the blood coagulation factors per se but the response of the platelets, and that the heparins and warfarin derivatives do not inhibit this response.

It is important, too, to keep in mind the role of antithrombin III, a co-factor for heparin. Without it there cannot be a balanced clotting mechanism. The recently recognized heparin co-factor may be deficient in some people, leaving them with a predisposition to clotting. Such situations, if not identified, will lead to an inadequate response to heparin when it ordinarily would provide effective therapy.

One must also consider the difficulties with arterial diseases. We recognize arterial thrombosis as either a blood clot in an artery that seems to have a normal surface, or a blood clot in a vessel in which the surface has been significantly altered by atherosclerosis.

In the past there have been fads in the treatment and prevention of thrombotic diseases with the warfarin derivatives, which have been in and out of favor for use in long-term prevention. Recent information suggests that some people who have changes in vessels may very well not have adequate coagulation mechanisms, and that their clots are not alone due to the conversion of fibrinogen to fibrin. Their thrombi may in some way be related to the alteration of fats, lipids, triglycerides, and other substances that may help initiate or perpetuate vessel-wall abnormalities.

Platelets Alter Vessel Wall

There seems to be a very intimate relationship between the platelets sticking to the blood vessel wall and an alteration in the vessel wall, all of which may perpetuate the system. There is even some very suggestive laboratory evidence that the platelets being sequestered near the surface walls of other platelets may actually promote changes in the vessel wall, and that this in turn makes it easier for the platelets to adhere to it.

And remember, not all arteries are closed by clots. Some are blocked by pure atherosclerotic plaque. Evidence has been found that alteration of blood vessel walls in classical atherogenesis can be produced by platelets releasing substances that act on the walls. Consequently, in the search for ways to prevent arterial vascular disease, one now may begin to investigate agents that eliminate or interfere with the platelet attaching itself to the vessel wall.

We are beginning to get a handle on mechanisms that produce coagulation problems, by studying the causes. Is it the coagulation mechanism, together with various other interactions, that set off the cascade of clotting factors that actively leads to the final deposition of fibrinogen as the main cause of the clot? Or is it the platelet-vessel wall interaction that initiates the formation of what we call "platelet plug," which is as effective a clot as the fibrinogen plug in stopping blood flow? If one can answer these questions, one can give more rational therapy, and that is the key to this whole question.

Key to Thrombus Prevention

The heparin-like drugs are basically compounds that attack the final depositions and the conversion of fibrinogen to fibrin. If the clot was not formed by this pathway, those drugs will not work. On the other hand, if the platelet-vessel wall interaction is the initiating factor, we now have drugs that in vitro or in vivo can alter platelet responsiveness, and may be the key to the prevention of arterial and/or venous thrombus formation.

Warfarin derivatives have been used for major prevention of venous and arterial clots because of their ease of administration and relatively low rate of complications. They slow the coagulation cascade to retard the formation of fibrin. They do not, however, affect platelet function, and cannot prevent fibrinogen conversion.

In addition, in an age of multiple drug use, we are recognizing more and more drug-drug interactions that affect warfarin derivative metabolism, making the simple monitoring of these drugs by the familar prothrombin-time more difficult. An ideal, easy-to-administer, appropriate inhibitor of fibrinogen conversion is not available. However, more reports are appearing, suggesting that self-administered, low-dose, subcutaneous heparin may be an effective measure for the prevention of venous thromboembolic disease.

There are arterial diseases and there are venous diseases. Major ones are caused by the coagulation mechanism; some are possibly caused by platelets. The arterial diseases look more and more as though they are influenced by the platelets. It is possible, in a highly sophisticated medical center, to do complex laboratory tests that might give an indication, but as yet we do not have appropriate methods widely available for the primary care physician. Investigations are continuing, however, and one hopes they will lead to rational use of the heparin drugs and the anti-platelet drugs. □

NEXT IN THE SERIES: Heparin—when and how it should be used, and when it should probably not be employed, based on what is proved, what appears probable, and what is indicated by recent research.

Pulmonary Edema—Treatment

By Max Harry Weil, M.D.

Dr. Weil is Director and Clinical Professor of Medicine and Biomedical Engineering, University of Southern California Center for the Critically Ill, Los Angeles.

Pulmonary edema may follow shock, respiratory distress, lung injuries and infections, and a wide range of heart disorders. Although a common condition, it is not completely understood and should not be regarded as simply a result of pressure backed up from the left ventricle. Recent treatment of a specific group of patients shows that plasma colloid osmotic pressure and its relationship to pulmonary capillary pressure is intimately involved in the development of pulmonary edema. The difference between the two pressures, determined by measuring each, may indicate a leakage of plasma fluid into the pulmonary interstitium. Following massive fluid loss, replenishing of plasma volume with albumin-containing fluids may restore colloid osmotic pressure to normal and thereby reduce the risk of pulmonary edema.

Pulmonary edema may seem simple enough to diagnose and perhaps equally simple to treat, but, in fact, it presents a dire threat to the patient and a considerable challenge to the clinician.

Multiple factors contribute to its cause, and they bear directly on the choice of therapy. Signs and symptoms don't always mean what they seem to mean, and can lead the physician astray. Mortality is high—a survival rate of less than 50 per cent for patients who have pulmonary edema after active myocardial infarction. To successfully meet this combination of problems, the workup must be meticulous and management extremely selective.

Pulmonary edema often develops in the seriously ill patient, particularly after acute myocardial infarction. It can appear, too, after any type of left ventricular failure, aortic stenosis, myocardiopathy, mitral insufficiency, and congenital heart diseases. It may be related to lung injury (e.g., from aspiration), shock, or adult respiratory distress syndrome; or it may be superimposed on pulmonary infections. The so-called hydrostatic pulmonary edema is caused by a rise in the left ventricular filling pressure, which in turn results from a decrease in the ability of the left ventricle to eject a sufficient volume of blood. The pressure is retroconducted to the left atrium and then to the pulmonary veins, thereby increasing capillary hydrostatic pressure.

Measure the Pressures

In some patients, however, the problem stems from a reduction in the plasma colloids and therefore a lowering of colloid osmotic pressure. Since it is the connection between colloid osmotic pressure and hydrostatic pressure which has recently been related to the appearance of pulmonary edema, colloid osmotic pressure should be measured. At the same time wedge pulmonary artery pressure or pulmonary diastolic pressure should be measured as an indication of hydrostatic forces. In many cases, both decreased colloid osmotic pressure and increased hydrostatic pulmonary capillary pressure are involved.

The chief sign of pulmonary edema may be the early appearance of moist rales or evidence of pulmonary interstitial and alveolar fluid accumulation on the chest radiograph. To this extent the chest x-ray is a very useful qualitative guide. Quantitative determination of conditions in which pulmonary edema develops, however, is best gauged by hemodynamic measurements — of left ventricular filling pressure and colloid osmotic pressure. The left ventricular pressure can be recorded in a hospital, by putting a catheter directly into the left ventricle retrograde, from the axillary artery or from the femoral artery. The colloid osmotic pressure can be mea-

Depends on Cause

sured on a sample of plasma by a commercially available oncometer. Physical signs include distention of the neck veins—which suggests an increase in right ventricular filling pressure — and alveolar edema, indicated by rales and generally confirmed by auscultation of the lungs. The clinician must, however, regard these signs with some caution, for several reasons. An increase in central venous pressure is contingent on right-sided failure, which is usually delayed following acute myocardial infarction. This is the very reason why the Swan-Ganz catheter—the flotation pulmonary artery catheter by which wedge pressure is measured — has been a boon.

Failure Without the Signs

The clinician should remember that right heart failure, although very common following left heart failure, follows only after substantial lag. Therefore, central venous pressure and neck veins may sometimes be normal during the earlier stages of left heart failure. Thus, pulmonary edema may develop as a result of left heart failure without the patient having signs of left heart failure.

Secondly, pulmonary edema in its earlier stages is interstitial edema, which doesn't involve the alveoli of the lungs. Consequently, the assumption that rales, heard in patients after onset of alveolar edema, would be reliable as a clear indication has to be rejected. The clinician must use more sophisticated measurement.

Now it may be that we have to accept the lesser efficiency of clinical signs and clinical measurements, under circumstances in which cost and volume of patients present obstacles to the more sophisticated techniques. But it would be really quite wrong to even remotely imply that this is sufficient, if the physician wants to do the optimal job.

For the management of these patients, the most effective drugs we have are a loop diuretic, furosemide, and ethacrynic acid. Occasionally, restoring of fluid volume is indicated. It is important, under those circumstances, to be aware of the colloid osmotic pressure in order not to dilute the volume with noncolloid-containing fluids.

Very often, volume buildup is necessary for a patient who is in cardiogenic shock, but that volume probably should not be entirely made up of noncolloid-containing fluids, but rather of albumin-containing fluids.

Pulmonary edema is the end result of multiple factors. In earlier years, it was thought that the condition was caused by failure of the left ventricle, and the hydrostatic forces, which raise the pressure in the pulmonary capillaries, resulting in leakage of fluid. We now know that the colloid contained in the plasma is also an important variable, and may, in fact, greatly modify the extent to which fluid would or would not leak out in the presence or absence of left ventricular failure.

The physician should keep in mind that in some patients following acute myocardial infarction, the colloid osmotic pressure is reduced and the left ventricular filling pressure may in fact be not very high. Under these conditions, the decrease in colloid osmotic pressure may be the primary cause of pulmonary edema. In these patients, not only the hydrostatic pressure but also the colloid osmotic pressure must be considered in efforts to reverse the edema. □

Practice Procedures

Pulmonary Edema—Osmotic Pressure—Hydrostatic Pressure

Chief Signs: (Must be confirmed by more sophisticated measures)

- Moist rales.
- Pulmonary overload evident on chest x-ray.
- Distention of neck veins.

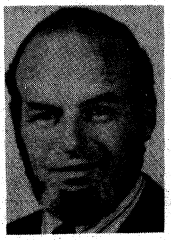
Measure:

- Colloid osmotic pressure.
- Pulmonary artery wedge pressure.

Management:

- If colloid osmotic pressure is critically reduced in a volume-depleted patient, consider fluid volume buildup with 5 per cent human serum albumin. Be cautious in use of noncolloid-containing fluids. Loop diuretics increase colloid osmotic pressure.

Should Digitalis Be Used In Treating Acute



Dr. Haber is Professor of Medicine Department of Medicine, Harvard Medical School; Chief, Cardiac Unit Massachusetts General Hospital, Boston

Conflicting opinions and practices raise questions about digitalis therapy in acute myocardial infarction. Some clinicians feel its use is contraindicated; even when there is accompanying heart failure, the use of digitalis is not universally accepted. Opinions also differ on its use in cardiogenic shock. Most agree, however, that it should be employed when acute myocardial infarction is accompanied by atrial fibrillation. There are still many uncertainties about digitalis therapy, but so intensive is current scientific investigation that helpful data can be expected to appear in the near future.

The role of digitalis therapy after acute myocardial infarction is still uncertain and, despite the magnitude of efforts by biomedical investigators in the study of digitalis glycosides, there are important unanswered questions in this clinical situation.

Some workers emphasize that there is little to be gained by administration of digitalis glycosides to patients who have uncomplicated infarction without cardiomegaly. There is also little clinical documentation of the drug's value in cardiogenic shock, a syndrome in which no pharmacologic agent has as yet been demonstrated to be very effective. Indeed, rapid digitalization may, on occasion, be harmful due to the vasoconstrictor properties of the drug.

Yet Karliner and Braunwald, for instance, wrote in 1972 that until evidence to the contrary appears, digitalis should be continued in cardiogenic shock. They took this position on the basis of experience with experimental work and the presumption that this state, when observed clinically, is a form of extreme left ventricular failure.

Typical of the findings that still project a confused picture to the clinician are results published in the Abstracts of the American Heart Association 48th (1975) Scientific Sessions. One group at North Shore and Cornell gathered data which "suggest that IV digoxin in certain patients with large predicted acute myocardial infarctions and high pulmonary wedge pressures results in salvage of ischemic myocardium." On the other hand, a joint Russian and American group, analyzing serum CPK in patients, concluded that digitalis appears to increase infarct size in patients with evolving myocardial infarction.

Increases Infarct Size

This is an important question; and the finding that digitalis increased infarct size was supported by the 1971 observations of Maroko et al, that a number of inotropic agents, including ouabain, increased the severity and extent of ischemic injury in experimental coronary occlusion in the non-failing heart.

When congestive heart failure complicates myocardial infarction, the use of digitalis is widely advocated. Yet, there are few studies in man that provide a solid basis for this recommendation. Karliner and Braunwald, noting the paucity of experimental and clinical evidence for such therapy, called for more clinical investigation into the question of whether the digitalis glycosides should continue to be used routinely in such cases.

More recently, some benefits have been noted for

Myocardial Infarction?

By Edgar Haber, M.D.

patients after digitalization to treat left ventricular failure following myocardial infarction. Others have found evidence of significant improvement from ouabain therapy in some indexes of left ventricular performance even though cardiac output was not affected. Nevertheless, it was felt that more study was needed of how digitalis acts on ventricle size and myocardial oxygen consumption.

Indication for Atrial Fibrillation

The clearest indication for digitalis after acute myocardial infarction is in the treatment of atrial fibrillation with a rapid ventricular rate. Electrical cardioversion may be preferred in the treatment of other supraventricular tachyarrhythmias. Animal experiments suggest that digitalis may induce arrhythmias at lower doses in cases of acute myocardial infarction, and that the toxic dose of digitalis glycosides is reduced as a result of rhythm disturbances. Clinical evidence on this is not clear.

The questions raised will certainly be studied intensively. Since 1912, when Herrick advocated use of digitalis in all patients with acute myocardial infarction, practice and techniques have changed. The magnitude of effort being expended by biomedical investigators in the study of digitalis can be judged from the listing of more than 600 citations in Index Medicus in 1972 alone. More than 300 compounds in the digitalis classification are known to exist, and the sources and structure-activity relations have been known in depth for more than ten years. But no compound has as yet been indentified with an improved therapeutic-toxic ratio.

It is now considered that the major action of digitalis is the augmentation of the force of myocardial contraction, although this appeared to have been generally overlooked by 19th century investigators, who stressed slowing of the heart as its major effect. Another myth that investigation has dispelled in the past 25 years has been the belief that the inotropic action of digitalis was confined to the decompensated heart. It is now clear that the drug has this action in the normal, as well as failing, heart muscle.

Yet the administration of cardiac glycosides results in no change, or a slight decline, in cardiac output in normal subjects. Cardiac output is determined not only by the cardiac contractile state, but also by ventricular filling pressure, peripheral arterial resistance, and heart rate. It now appears that digitalis augments the contractile state of the normal myocardium in intact man, but that reflex adjustments in the other determinants of cardiac output prevent a ready appreciation of this inotropic effect.

Is there such a thing as too much myocardial contractility? Experiments have shown that the inotropic action of digitalis increases progressively until toxic arrhythmias appear. The clinician's task is to determine the maximum dose consistent with an adequate margin of safety. □

Practice Procedures

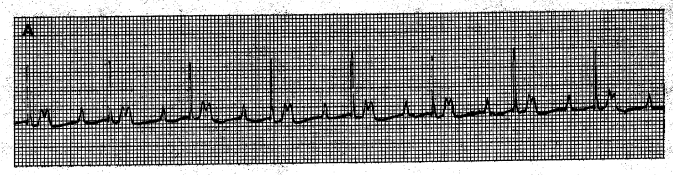
Debate Over Digitalis

The Questions:

- Is the drug useful for cardiogenic shock?
- Does it increase the size of a myocardial infarct?
- Should digitalis be used routinely when congestive heart failure complicates myocardial infarction?
- Should it be used for left ventricular failure following myocardial infarction?
- Does digitalis induce arrhythmias in acute myocardial infarction?
- Is there a possibility of too much myocardial contractility?

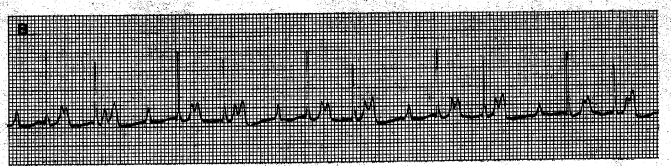
There are few, if any, clear-cut answers at present, but intensive investigation is continuing. In the meantime, the physician must exert extreme care to determine the maximum dose of digitalis that is consistent with an adequate margin of safety.

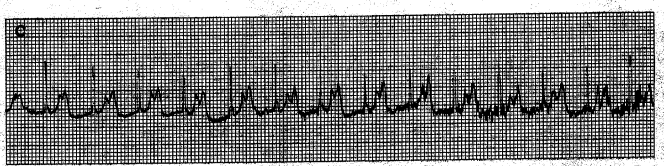
ECG Of The Month



Strip A is a monitor lead from a 62-year-old male admitted to a coronary care unit with retrosternal discomfort and an ECG showing an acute diaphragmatic myocardial infarction.

The rhythm in Strip A is 2:1 AV block, a form of second degree AV block. The atrial rate is 94 and the ventricular rate is 47. Every other P wave is conducted at the ventricle with a PR interval of 0.46 seconds. The blocked P waves occur during the T wave, giving a notched appearance to the T wave.





2:1 AV block may represent either Wenchebach (Type I) second degree AV block, or Mobitz (Type II) second degree AV block. It is not possible to distinguish the two possibilities on Strip A alone, although Wenchebach variety or block in the AV node above the bundle of His can be favored on the basis of the marked prolongation of the PR interval and the normal QRS width. The mechanism is clarified in Strips B and C. Strip B shows 3:2 AV block. The PR interval of the first of the conducted beats is 0.46 seconds and the PR interval of the second of the conducted beats is 0.50 seconds. The third P wave is blocked. Thus, this represents Wenchebach periodicity with progressive PR prolongation prior to the dropped beat. Strip C shows further improvement of conduction with only first degree AV block and with a PR interval of 0.48 seconds.

These strips represent various manifestations of AV block at the level of the AV node which can be seen in patients with diaphragmatic or inferior myocardial infarction. In this case, there was progressively improved conduction, although in some patients there may be progression to complete AV block and either drug or pacemaker therapy may be necessary. In inferior myocardial infarction, however, complete heart block is usually transitory. Mobitz (Type II) second degree AV block with constant and usually normal PR interval prior to the dropped beat(s) is usually seen in anterior wall infarctions, and complete heart block, if it develops, is a more ominous event. □

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
FOOD AND DRUG ADMINISTRATION
ROCKVILLE, MARYLAND 20852

MAY 27 1976

Honorable Gaylord Nelson
Chairman, Subcommittee on Monopoly
Select Committee on Small Business
United States Senate
Washington, D.C. 20510

Dear Senator Nelson:

I wish to correct two errors in our testimony before your subcommittee on April 28, 1976.

In discussing scientific exhibits, I referred to the written programs for scientific exhibits at two recent meetings of the American Academy of Family Practice and the American College of Physicians and pointed out in each a listed exhibit on prazosin hydrochloride, an antihypertensive drug. I indicated that the program did not note that the physician-exhibitor was a full-time employee of the manufacturer which had developed the drug. In addition, I said that prazosin hydrochloride had not been approved for marketing at the time of either meeting, and that regulations did not permit pharmaceutical manufacturers to promote drugs prior to their approval for marketing. When asked about this by Mr. Gordon, I said that "this type of thing is a violation of the current regulations on drug labeling."

I have subsequently learned that the manufacturer of prazosin hydrochloride (Pfizer) prepared the exhibits in anticipation of the approval of the drug but, when this approval was delayed, did not in fact display them at either meeting although they remained listed in the printed programs. I did not know this at the time of my testimony, and I regret that we did not check this point before preparing our testimony. Thus, no violation of FDA regulations occurred, and I extend my apologies to you and to Pfizer, Inc. for suggesting otherwise.

Let me add, however, that this type of presentation remains a valid illustration of our concerns regarding the promotional aspects of scientific exhibits. As I testified, it is FDA's view, which we intend to incorporate into proposed regulations, that a scientific exhibit on a drug must be prepared independently of a drug manufacturer's control if it is to be exempt from drug labeling requirements. I believe the

planned Pfizer exhibit, if actually presented, would have properly been termed "drug labeling," by any reasonable description, and thus subject to the full disclosure and fair balance standards applicable to such labeling.

The second error occurred during Dr. Rheinstein's testimony. In response to your question regarding the selection of the physician panels for the Pfizer-sponsored closed circuit television symposium on diabetes, Dr. Rheinstein replied that "Pfizer contracted with another company called Health Learning Systems to do the entire production." (Testimony at page 22.) Also in response to another question Dr. Rheinstein indicated "And previous seminars sponsored by Health Learning Systems have said that they had up to 8,000 physicians seeing closed circuit broadcasts at one time." (Testimony at page 24.) Subsequent to this hearing, it was learned that Pfizer did not contract with any outside company but made all arrangements including the selection of physician panels staff itself. Health Learning Systems produces multi-city closed circuit TV programs on medical subjects, but was not involved in this program.

Sincerely yours,

J. Richard Crout, M.D.
Director
Bureau of Drugs

cc: Pfizer, Inc.
Health Learning Systems

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
FOOD AND DRUG ADMINISTRATION
WASHINGTON, D.C. 20204

JUN 18 1976

Paul Cutler, M.D.
Professor of Medicine
The University of Texas
Health Science Center at San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78284

Dear Dr. Cutler:

Thank you for your letter of May 17 commenting on the text of my statement before the Subcommittee on Monopoly of the Senate Select Committee on Small Business.

I am pleased that you share my concern regarding the influence of the pharmaceutical industry on the continuing education of physicians. My concern is with our system of continuing education, a system that is supported in growing measure by an industry with a particular interest. It is not with specific articles or their authors. I tried to point out in my testimony that the quality of the articles in controlled-circulation journals is often high and that these journals emphasize practical patient care information often neglected by the "scholarly" journals. Further, I certainly agree with your feeling that investigational therapies should be considered in the medical literature. I have never argued that the FDA-approved package insert should in any way limit discussions about drugs by independent physicians.

The migraine article was used simply as an example of material presented in an industry-supported publication which a drug company would be prohibited from saying in its own labeling or advertisements. Regardless of the merit of the article and degree of review, this situation is properly open to discussion by reasonable men without such discussion being taken as reflecting on the integrity of individuals. If you feel that Current Prescribing has methods of subject selection and internal review currently unknown to the public which protect the independence of scientific content and yet permit industry funding, please let us know of these in greater detail.

In preparing testimony for Senator Nelson, I felt my broad discussion of post-graduate medical education had to include a fair number of examples of the educational materials I was talking about. A number of the editors and authors of these examples have felt that their motives and integrity were questioned. I specifically tried to avoid any such

suggestion but obviously did not convey my feeling fully. My examples were intended to show only that controlled-circulation journals (or educational cassettes, or seminars) were regularly concerned with subject matter that is of crucial interest to the drug industry.

If I understand your letter correctly, you would not disagree with me in feeling that control over the content of educational materials should be in the hands of independent physicians and educators. At the present time, with medical educational materials of all kinds increasingly funded by drug manufacturers, there is, I believe, a growing burden on controlled circulation journals to show this is the case. Please take this as a challenge to our current system and certain policies, not as a comment on anyone's personal honesty.

According to information supplied by the publishers of Current Prescribing and appearing in the May 24, 1976 issue of Standard Rate and Data Service, the circulation of Current Prescribing is entirely non-paid. Although many physicians may have returned to the publishers business reply cards supplied by the publisher and stating that these physicians would like to receive or continue to receive Current Prescribing, this does not change the publication's status as a controlled circulation journal or its financial support.

Once again I appreciate your letter and the opportunity to exchange views.

Sincerely yours,

J. Richard Crout, M.D.
Director
Bureau of Drugs

cc: Senator Gaylord Nelson

STATEMENT OF
THE AMERICAN ACADEMY OF FAMILY PHYSICIANS
BY
JOHN C. KELLY, M.D.
SUBCOMMITTEE ON MONOPOLY
SELECT COMMITTEE ON SMALL BUSINESS
UNITED STATES SENATE
MAY 24, 1976

MR. CHAIRMAN AND MEMBERS OF THE SUBCOMMITTEE, I AM JOHN C. KELLY, A PRACTICING FAMILY PHYSICIAN FROM RENO, NEVADA. I CURRENTLY SERVE AS CHAIRMAN OF THE BOARD OF DIRECTORS OF THE AMERICAN ACADEMY OF FAMILY PHYSICIANS, WHICH IS THE LARGEST MEDICAL SPECIALTY ORGANIZATION IN THE UNITED STATES, WITH OVER 37,000 MEMBERS. SEATED WITH ME IS LESLIE HUFFMAN, WHO IS A PRACTICING FAMILY PHYSICIAN FROM GRAND RAPIDS, OHIO. DR. HUFFMAN IS THE SPEAKER OF THE ACADEMY'S CONGRESS OF DELEGATES AND FORMERLY SERVED AS CHAIRMAN OF OUR COMMITTEE ON SCIENTIFIC PROGRAM.

WE ARE PLEASED TO BE PRESENT TODAY TO PROVIDE INFORMATION TO THE SUBCOMMITTEE REGARDING COMMERCIAL AND SCIENTIFIC EXHIBITS AS THEY RELATE TO THE ACADEMY'S LONG-STANDING CONCERN FOR INSURING THE CONTINUING EDUCATION OF PHYSICIANS. I WOULD LIKE TO POINT OUT THAT THE ACADEMY IS RECOGNIZED AS A PIONEER IN THE AREA OF CONTINUING EDUCATION. WE ARE PROUD TO SAY THAT, AMONG ALL ORGANIZATIONS REPRESENTING PHYSICIANS, WE WERE THE FIRST TO REQUIRE A SPECIFIC NUMBER OF HOURS OF CONTINUING EDUCATION IN ORDER TO RETAIN MEMBERSHIP.

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THE ACADEMY'S ANNUAL SCIENTIFIC ASSEMBLY PROVIDES A WIDE CHOICE OF CONTINUING EDUCATION OPPORTUNITIES FOR FAMILY PHYSICIANS. PRACTICAL DIDACTIC LECTURES BY NATIONALLY KNOWN SPEAKERS ARE AN ASSEMBLY HIGHLIGHT. OTHER PROGRAM ELEMENTS ALSO EMPHASIZE THE PRACTICAL: COURSES COMPLETE WITH SYLLABI; SMALL GROUP DISCUSSIONS; DEMONSTRATIONS OF TECHNIQUES AND PROCEDURES WHICH FAMILY PHYSICIANS CAN USE IN THEIR DAY-TO-DAY PRACTICES. SCIENTIFIC AND TECHNICAL EXHIBITS PROVIDE A GREAT SOURCE OF INFORMATION ON RESEARCH PROJECTS, NEW EQUIPMENT, SERVICES, AND PHARMACEUTICALS.

PHYSICIANS EARN ONE HOUR OF PRESCRIBED AAFP CONTINUING EDUCATION CREDIT FOR EACH HOUR OF PARTICIPATION IN ANY ELEMENT OF THE SCIENTIFIC PROGRAM, EXCEPT FOR THE VIEWING OF EXHIBITS. BY CAREFULLY SCHEDULING HIS TIME, A PHYSICIAN CAN EARN 31 CREDIT HOURS DURING THE 3½ DAYS OF THE ASSEMBLY.

PHYSICIAN REGISTRATION AT THE 1974 ASSEMBLY IN LOS ANGELES WAS 4,978--AN ALL-TIME HIGH. CLINICAL SEMINARS--THE SMALL GROUP DISCUSSIONS--PROVED TO BE MOST POPULAR. IN 1974, 518 SEMINARS ON 40 DIFFERENT TOPICS WERE PRESENTED. WITH ATTENDANCE IN EACH SEMINAR LIMITED TO 15 REGISTRANTS, 7,770 INDIVIDUAL OPENINGS WERE AVAILABLE. IN 1974, MORE THAT 600 EDUCATIONAL PRESENTATIONS WERE SCHEDULED IN THE VARIOUS PROGRAM ELEMENTS.

OUR 1975 ASSEMBLY IN CHICAGO REGISTERED 3,622 PHYSICIANS. THE NUMBER OF EDUCATIONAL PRESENTATIONS SCHEDULED IN THE VARIOUS PROGRAM ELEMENTS WAS APPROXIMATELY 300.

ALTHOUGH THE SCOPE OF THE SUBCOMMITTEE'S HEARINGS HAS COVERED MANY ASPECTS OF THE ROLE OF PHARMACEUTICAL COMPANIES IN PROVIDING CONTINUING MEDICAL EDUCATION, WE HAVE BEEN REQUESTED TO CONFINE OUR STATEMENT TO COMMERCIAL AND SCIENTIFIC EXHIBITS. ACCORDINGLY, OUR TESTIMONY WILL BE LIMITED TO THAT TOPIC. WE WERE SPECIFICALLY REQUESTED TO COVER THE FOLLOWING POINTS: THE CONTENT AND PURPOSE OF COMMERCIAL AND SCIENTIFIC EXHIBITS AND WHAT DISTINGUISHES ONE FROM THE OTHER; WHO SPONSORS THEM; WHO PREPARES THEM; WHAT KIND OF EDITORIAL REVIEW IS EXERCISED; ARE ANY EXHIBITS REJECTED; AND THE EXTENT TO WHICH CONVENTIONS ARE DEPENDENT ON DRUG COMPANY SUPPORT. WE WILL DISCUSS THESE POINTS IN THE ORDER IN WHICH THEY HAVE BEEN LISTED.

THE FIRST QUESTION CONCERNS THE CONTENT AND PURPOSE OF COMMERCIAL AND SCIENTIFIC EXHIBITS. ALTHOUGH WE CONSIDER BOTH TYPES OF EXHIBITS TO BE VALUABLE MECHANISMS FOR PROVIDING CURRENT MEDICAL INFORMATION TO OUR MEMBERS, WE RECOGNIZE THAT THE PRIMARY PURPOSE OF COMMERCIAL EXHIBITS IS TO PROMOTE THE PRODUCTS OF THOSE PURCHASING THE EXHIBIT SPACE. ON THE OTHER HAND, THE PRIMARY PURPOSE OF SCIENTIFIC EXHIBITS IS TO PROVIDE INFORMATION TO PHYSICIANS THROUGH THE DISSEMINATION OF THE RESULTS OF SCIENTIFIC RESEARCH. UNLIKE COMMERCIAL EXHIBIT SPACE, SCIENTIFIC EXHIBIT SPACE AT OUR ANNUAL MEETING CANNOT BE PURCHASED. RATHER, IT IS AWARDED ON THE BASIS OF THE EXHIBIT'S RELEVANCE TO FAMILY PRACTICE AS WELL AS QUALITY, LEGIBILITY, SCIENTIFIC ACCURACY AND ETHICAL CONTENT. WHEREAS A PROSPECTUS IS SENT TO POTENTIAL COMMERCIAL EXHIBITORS, THE ACADEMY DOES NOT SOLICIT SCIENTIFIC EXHIBITS EXCEPT IN THOSE INSTANCES IN WHICH THE EXHIBIT HAS BEEN VIEWED BY A MEMBER OF OUR SCIENTIFIC PROGRAM COMMITTEE

AT SOME OTHER MEDICAL MEETING AND HAS BEEN RECOMMENDED FOR PRESENTATION AT OUR MEETING ON THE BASIS OF FULFILLING THE CRITERIA PREVIOUSLY MENTIONED. UNSOLICITED EXHIBITS ARE ACCEPTED OR REJECTED FOLLOWING A REVIEW OF INFORMATION SUBMITTED BY THE PROSPECTIVE EXHIBITOR IN THE SCIENTIFIC EXHIBIT APPLICATION FORM WHICH IS ATTACHED TO THIS TESTIMONY AS EXHIBIT A.

A REVIEW OF THIS APPLICATION FORM BRINGS OUT SEVERAL ADDITIONAL DIFFERENCES BETWEEN COMMERCIAL AND SCIENTIFIC EXHIBITS. WHEREAS ONE OF THE PURPOSES OF COMMERCIAL EXHIBITS IS ADMITTEDLY THE PROMOTION OF PARTICULAR PRODUCTS, THERE IS A STRICT PROHIBITION AGAINST SELLING OR OFFERING FOR SALE ANY COMMERCIAL ITEM IN CONJUNCTION WITH A SCIENTIFIC EXHIBIT. WHEREAS SALES REPRESENTATIVES OF PHARMACEUTICAL COMPANIES CAN AND DO STAFF COMMERCIAL EXHIBITS, THERE IS A STRICT PROHIBITION AGAINST SUCH REPRESENTATIVES PERFORMING A SIMILAR FUNCTION IN CONJUNCTION WITH A SCIENTIFIC EXHIBIT. WHEREAS COMMERCIAL EXHIBITS DO PROMOTE DRUG PRODUCTS BY TRADE NAME, ONLY GENERIC NAMES MAY BE USED IN THE BODY OF SCIENTIFIC EXHIBITS AND ANY REFERENCES TO SUCH GENERIC DESIGNATIONS MUST BE AFFORDED EQUAL EMPHASIS.

IN TESTIMONY PRESENTED BEFORE THIS SUBCOMMITTEE ON APRIL 28TH BY DR. RICHARD CROUT, DIRECTOR OF THE FOOD AND DRUG ADMINISTRATION'S BUREAU OF DRUGS, EMPHASIS IS GIVEN TO THE FACT THAT MATERIAL CONTAINED IN SCIENTIFIC EXHIBITS USUALLY IS NOT SUBJECT TO THE SAME RESTRICTIONS PLACED ON DRUG LABELING. THE THRUST OF DR. CROUT'S TESTIMONY SEEMED TO BE THAT ALTHOUGH COMMERICAL EXHIBITS CAN BE REGULATED ADEQUATELY, SCIENTIFIC EXHIBITS PROVIDE A MECHANISM WHEREBY DRUG COMPANIES CAN PROMOTE THEIR PRODUCTS WITHOUT BEING SUBJECT TO THE USUAL REQUIREMENTS FOR DRUG LABELING AND ADVERTISING.

HOWEVER, BECAUSE THERE IS A DIFFERENCE BETWEEN COMMERCIAL EXHIBITS AND SCIENTIFIC EXHIBITS, WE BELIEVE THERE MAY BE GOOD REASON THEY SHOULD NOT BE SUBJECT TO THE SAME RESTRICTIONS. AGAIN, LET ME EMPHASIZE THE POINT THAT THE PRIMARY PURPOSE OF SCIENTIFIC EXHIBITS IS EDUCATION WHEREAS THE PRIMARY PURPOSE OF COMMERCIAL EXHIBITS IS PROMOTION. I DON'T BELIEVE THAT PHYSICIANS VIEWING A SCIENTIFIC EXHIBIT WILL ACCEPT IT FOR ANY MORE THAN WHAT IT IS, THE RESEARCH FINDINGS OF A PARTICULAR EXHIBITOR. NOR DO I BELIEVE THAT THE DISSEMINATION OF INFORMATION THROUGH A SCIENTIFIC EXHIBIT WILL ENCOURAGE PHYSICIANS TO USE DRUGS IN A MANNER OR FOR A PURPOSE FOR WHICH THEY ARE NOT APPROVED. THE FDA STILL HAS THE FINAL ATUHORITY TO DETERMINE THE PROPER USE OF A PARTICULAR DRUG AND THE APPLICATION OF THE FINDINGS OF A PARTICULAR RESEARCHER ARE LIMITED BY THIS AUTHORITY. ON THE OTHER HAND, AS LONG AS A RESEARCH EFFORT REPRESENTS THE LEGITIMATE FINDINGS OF THE RESEARCHER AND THE RESULTS ARE OBJECTIVELY PRESENTED, WE DON'T BELIEVE THE MATERIAL SHOULD BE WITHHELD FROM THE MEDICAL COMMUNITY SOLEY ON THE BASIS OF THE FACT THAT IT MAY BE FAVORABLE TO A PARTICULAR DRUG. AS A REVIEW OF THE ATTACHED SCIENTIFIC EXHIBIT APPLICATION FORM WILL SHOW, THE ACADEMY'S REQUIREMENTS FOR SCIENTIFIC EXHIBITS HAVE BEEN ESTABLISHED FOR THE PURPOSE OF INSURING THAT THESE EXHIBITS ARE NOT IMPROPER OR UNETHICAL AND THAT THEY SERVE AN EDUCATIONAL FUNCTION.

IN RESPONSE TO THE QUESTION OF WHO SPONSORS THESE EXHIBITS, A LARGE PERCENTAGE OF COMMERCIAL EXHIBITS ARE SPONSORED BY PHARMACEUTICAL COMPANIES. THESE ARE READILY IDENTIFIABLE SINCE THE EXHIBIT SPACE IS PURCHASED BY THE COMPANY WISHING TO UTILIZE IT.

IN THE CASE OF SCIENTIFIC EXHIBITS, THE SPONSORS MAY INCLUDE PUBLIC AND PRIVATE NON-PROFIT ORGANIZATIONS, SUCH AS PROFESSIONAL AND LAY HEALTH ORGANIZATIONS AND MEDICAL SCHOOLS, AS WELL AS GOVERNMENTAL AGENCIES AND THE ARMED SERVICES. IN ADDITION, A LARGE NUMBER OF SCIENTIFIC EXHIBITS ARE SUPPORTED BY PAHRMACEUTICAL COMPANIES TO ONE DEGREE OR ANTOEHR. IN REVIEWING THE SCIENTIFIC EXHIBITS RECEIVING DRUG COMPANY SUPPORT AT PAST ACADEMY MEETINGS, IT APPEARS THAT THE FIGURE OF 80% CITED IN DR. CROUT'S EARLIER TESTIMONY IS REASONABLE.

IT IS ACCURATE TO SUGGEST THAT DRUG COMPANIES ARE LIKELY TO SUPPORT THOSE EXHIBITS WHICH ARE FAVORABLE TO A PARTICULAR PRODUCT OF THE COMPANY. CERTAINLY, IT WOULD BE DIFFICULT IF NOT IMPOSSIBLE TO ENVISION A SITUATION IN WHICH A DRUG COMPANY WOULD SUPPORT AN EXHIBIT WHICH WAS UNFAVORABLE TO A PRODUCT OF THAT COMPANY. HOWEVER, IF THE MATERIAL CONTAINED IN AN EXHIBIT PRESENTS LEGITIMATE FINDINGS BASED ON SCIENTIFIC INVESTIGATION, WE BELIEVE IT SERVES A VALUABLE EDUCATIONAL FUNCTION, REGARDLESS OF SPONSORSHIP. SHOULD ORGANIZATIONS SUCH AS THE ACADEMY FIND THEMSELVES IN THE POSITION OF NOT HAVING SCIENTIFIC EXHIBITS SUPPORTED BY DRUG COMPANIES, THE RESULT WOULD NOT BE BETTER SCIENTIFIC EXHIBITS BUT THE PROBABLE ABSENCE OF SCIENTIFIC EXHIBITS ALTOGETHER.

WE WERE ASKED TO PROVIDE INFORMATION ON WHO PREPARES COMMERCIAL AND SCIENTIFIC EXHIBITS. IN THE CASE OF COMMERCIAL EXHIBITS, THE EXHIBIT IS PREPARED BY THE PHARMACEUTICAL COMPANY OR OTHER ORGANIZATION PURCHASING THE EXHIBIT SPACE. PRESUMABLY, THE ACTUAL CONSTRUCTION OF THE EXHIBIT MAY BE DONE IN-HOUSE OR BY AN EXHIBIT FIRM RETAINED BY THE COMPANY. IN ANY EVENT, THE FORMAT AND CONTENTS WOULD BE PRESENTED IN ACCORDANCE WITH THE SPECIFICATIONS OF THE

PURCHASER OF THE EXHIBIT SPACE.

SCIENTIFIC EXHIBITS WHICH ARE SUPPORTED BY DRUG COMPANIES MAY BE PREPARED BY DRUG COMPANIES, BY THE INDIVIDUAL OR INDIVIDUALS WHO CONDUCTED THE RESEARCH OR BY AN EXHIBIT FIRM RETAINED DIRECTLY BY THE INVESTIGATOR AND REIMBURSED BY THE DRUG COMPANY. ALTHOUGH THE DRUG COMPANY MAY EXERCISE SOME CONTROL OVER THE FORMAT USED FOR EXHIBITS WHICH IT SPONSORS, THE DATA AND CONCLUSIONS WHICH ARE THE BASIS OF THE EXHIBIT WOULD REMAIN THE RESPONSIBILITY OF THE INVESTIGATOR. IN THIS CONNECTION, I SHOULD MENTION THAT ALTHOUGH A DRUG COMPANY MIGHT EXERCISE CONTROL OVER THE EXHIBIT FORMAT IN ORDER TO INSURE EFFECTIVE VISUAL PRESENTATION OF THE MATERIAL, THE ACADEMY PLACES RESTRICTIONS ON THE FORMAT OF EXHIBITS PRESENTED AT OUR MEETING IN ORDER TO INSURE THAT THIS MATERIAL IS NOT PRESENTED IN A BIASED MANNER WHICH WOULD GIVE SPECIAL EMPHASIS TO A PARTICULAR DRUG. FOR EXAMPLE, THE ACADEMY'S REGULATIONS STATE: "COMPARISONS OF EFFICACY,CONTRAINDICATIONS, MODES OF ADMINISTRATION, ET CETERA,BETWEEN COMPETITIVE DRUGS SHALL BE FULLY PRESENTED. EDITORIAL CONCLUSIONS SHOULD BE MADE WITH ETHICAL RESTRAINT." OUR REGULATIONS FURTHER PROVIDE THAT "ALL GENERIC DESIGNATIONS OF COMPETITIVE DRUGS SHALL BE USED WITH EQUAL EMHASIS ON HEIGHT,WEIGHT, COLOR OR TYPE FACE OF LETTERING."

SCIENTIFIC EXHIBITS OTHER THAN THOSE SUPPORTED BY DRUG COMPANIES WOULD BE PREPARED BY THE INVESTIGATOR OR THE INSTITUTION OR AGENCY INVOLVED. FOR EXAMPLE, ONE OF THE EXHIBITS WHICH WON AN AWARD FOR EXCELLENCE AT OUR 1975 SCIENTIFIC ASSEMBLY WAS PREPARED SOLEY BY THE INVESTIGATOR, WHO HAND-PRINTED HIS DATA ON POSTER BOARD.

WE WERE REQUESTED TO INDICATE THE TYPE OF EDITORIAL REVIEW WHICH IS EXERCISED OVER THE CONTENT OF EXHIBITS AS WELL AS WHETHER OR NOT EXHIBITS ARE REJECTED. BECAUSE THESE QUESTIONS ARE INTERRELATED, I WOULD LIKE TO RESPOND TO BOTH OF THEM TOGETHER.

AS I PREVIOUSLY MENTIONED, BEFORE A SCIENTIFIC EXHIBIT IS ACCEPTED FOR PRESENTATION AT OUR ANNUAL MEETING, THE PROSPECTIVE EXHIBITOR MUST SUBMIT A SCIENTIFIC EXHIBIT APPLICATION FORM TO THE ACADEMY. THE FORM MUST BE ACCOMPANIED BY A TRANSCRIPT OF ALL COPY APPEARING IN THE EXHIBIT TOGETHER WITH A PHOTOGRAPH OR SKETCH, OR THE EXHIBIT MUST BE FULLY OUTLINED. THIS APPLICATION IS FORWARDED TO THE CHAIRMAN OF THE SUBCOMMITTEE ON SCIENTIFIC EXHIBITS FOR HIS REVIEW. IF, ON THE BASIS OF THE MATERIAL CONTAINED IN THE APPLICATION, THE CHAIRMAN OF THIS SUBCOMMITTEE DETERMINES THAT THE EXHIBIT DOES NOT CONFORM TO THE REGULATIONS, THE EXHIBIT IS REJECTED OR THE EXHIBITOR MAY BE REQUESTED TO MAKE APPROPRIATE CHANGES TO CONFORM TO THE REGULATIONS. IN ADDITION, THE EXHIBIT MAY BE REJECTED--AND THEY HAVE IN THE PAST--IF THE MATERIAL IS NOT RELEVANT TO FAMILY PRACTICE OR THE EXHIBIT APPEARS TO BE OF POOR QUALITY.

IN REQUESTING AN EXHIBITOR TO MAKE MODIFICATIONS IN THE EXHIBIT, OBVIOUSLY THE ACADEMY WOULD NOT REQUEST THAT THE DATA OR CONCLUSIONS BE CHANGED. HOWEVER, IF--FOR EXAMPLE--AN EXHIBIT CONTAINED REFERENCE TO A SPECIFIC DRUG COMPANY, THIS WOULD BE A VIOLATION OF OUR REGULATIONS AND THE EXHIBIT WOULD HAVE TO BE MODIFIED OR REJECTED.

IN THE PAST, SOME PROBLEMS HAVE EXISTED IN EXERCISING EDITORIAL REVIEW OVER SCIENTIFIC EXHIBITS ONCE THEY HAVE BEEN SET UP AT OUR SCIENTIFIC ASSEMBLY. LAST NOVEMBER, THE ACADEMY REQUESTED THE PMA

LIAISON COMMITTEE ON SCIENTIFIC EXHIBITS TO WORK WITH US AT OUR NEXT SCIENTIFIC ASSEMBLY IN BOSTON IN ORDER TO IMPROVE OUR ABILITY TO EXERCISE THIS ON-THE-SPOT REVIEW. THE FUNCTION OF THIS LIAISON COMMITTEE IS TO REVIEW ALL SCIENTIFIC EXHIBITS AFTER THEY HAVE BEEN ASSEMBLED AND PRIOR TO THEIR OPENING IN ORDER TO REPORT ANY INFRACTIONS OF OUR EXHIBIT REGULATIONS. EXHIBITORS RESPONSIBLE FOR THOSE EXHIBITS IN VIOLATION OF THE REGULATIONS WILL BE REQUESTED TO CORRECT THE VIOLATIONS PRIOR TO THE OPENING OF THE EXHIBITS. SUCH CORRECTIONS WILL BE ACCOMPLISHED BY MASKING IMPROPER OR INAPPROPRIATE MATERIAL. AGAIN, THE ACADEMY WOULD NOT ATTEMPT TO CHANGE THE DATA OR CONCLUSIONS, BUT RATHER CORRECT IMPROPER PRESENTATIONS, SUCH AS REFERENCE TO A SPECIFIC COMMERCIAL PRODUCT.

ALTHOUGH THE LIAISON COMMITTEE ON SCIENTIFIC EXHIBITS IS A BODY OF THE PHARMACEUTICAL MANUFACTURERS ASSOCIATION, WE HAVE EVERY REASON TO EXPECT THAT THEIR INPUT WILL DO MUCH TO INSURE THE HIGHEST ETHICAL AND SCIENTIFIC STANDARDS FOR THESE EXHIBITS. SINCE THE MEMBERSHIP OF THE PMA IS BROADLY REPRESENTATIVE OF THE DRUG INDUSTRY, IT SEEMS APPARENT THAT THIS COMMITTEE WILL HAVE AN INTEREST IN INSURING THAT EXHIBITS SPONSORED BY PARTICULAR DRUG FIRMS NOT PRESENT BIASED OR UNETHICAL MATERIAL. AND, I SHOULD REITERATE THE POINT THAT THE LIAISON COMMITTEE WILL NOT BE REVIEWING THE EXHIBIT AGAINST A SET OF CRITERIA ESTABLISHED BY THE INDUSTRY--BUT AGAINST THOSE ESTABLISHED BY THE ACADEMY.

ONE FURTHER COMMENT WHICH I WOULD LIKE TO MAKE REGARDING THE REJECTION OF EXHIBITS IS THAT EACH SCIENTIFIC EXHIBIT IS EVALUATED AT THE TIME OF THE SCIENTIFIC ASSEMBLY IN TERMS OF ITS VALUE TO FAMILY PRACTICE, EDUCATIONAL VALUE, APPEARANCE OR FORMAT AND SCIENTIFIC CONTENT. I HAVE ATTACHED A COPY OF THIS EVALUATION FORM AS APPENDIX B. THE FORM IS USED TO DETERMINE THOSE EXHIBITS DESERVING RECOGNITION AS OUTSTANDING EXHIBITS, AND IT IS COMPLETED

BY MEMBERS OF THE ACADEMY'S COMMITTEE ON SCIENTIFIC PROGRAM. FURTHERMORE, THE COMPLETED EVALUATION FORMS PROVIDE AN ADDITIONAL CRITERIA TO BE USED IN DETERMINING WHETHER A PARTICULAR EXHIBIT SHOULD BE ASSIGNED SPACE IN FUTURE YEARS, IF THE EXHIBITOR INVOLVED SUBMITS AN APPLICATION FOR SUCH SPACE.

AS REGARDS EDITORIAL REVIEW AND REJECTION OF COMMERCIAL EXHIBITS, FOR THE MOST PART, SUCH EXHIBITS ARE ACCEPTED UNLESS THEY ARE DEEMED INAPPROPRIATE TO A MEETING OF THIS TYPE OR UNLESS THERE HAS BEEN A PREVIOUS INSTANCE OF IMPROPER OR UNETHICAL CONDUCT BY THE EXHIBITOR INVOLVED. THERE HAS BEEN AT LEAST ONE INSTANCE IN WHICH A COMMERCIAL EXHIBITOR WAS BODILY REMOVED FROM THE EXHIBIT FLOOR BECAUSE HE ENGAGED IN SIDE-SHOW-TYPE TACTICS.

THE FINAL AREA WHICH WE WERE REQUESTED TO COVER IS THE EXTENT TO WHICH CONVENTIONS ARE DEPENDENT ON DRUG COMPANY SUPPORT. AS I HAVE PREVIOUSLY STATED, SCIENTIFIC EXHIBITS ARE AWARDED SPACE AT OUR CONVENTION ON THE BASIS OF THEIR EDUCATIONAL VALUE. SINCE SUCH EXHIBITS DO NOT PRODUCE INCOME, DRUG COMPANY SUPPORT OF THEM DOES NOT EFFECT THE ACADEMY'S OVERALL ABILITY TO STAGE ITS SCIENTIFIC ASSEMBLY. HOWEVER, BECAUSE A SIGNIFICANT NUMBER OF THESE EXHIBITS ARE SUPPORTED BY DRUG COMPANIES, SUCH SUPPORT IS ESSENTIAL TO INSURE THAT THIS PARTICULAR ELEMENT OF THE SCIENTIFIC PROGRAM IS POSSIBLE. WE DO BELIEVE SCIENTIFIC EXHIBITS ARE AN IMPORTANT ELEMENT OF CONTINUING EDUCATION, AND AS I HAVE MENTIONED, THE ABSENCE OF DRUG COMPANY SUPPORT WOULD NOT MEAN BETTER SCIENTIFIC EXHIBITS BUT A PROBABLE ABSENCE OF SCIENTIFIC EXHIBITS ALTOGETHER.

ON THE OTHER HAND, THE INCOME WHICH IS DERIVED FROM THE SALE OF COMMERCIAL EXHIBIT SPACE IS AN IMPORTANT FACTOR IN THE ACADEMY'S

ABILITY TO STAGE ITS ANNUAL MEETING. IN PAST YEARS, THE TOTAL COST OF HOLDING OUR ANNUAL MEETING HAS SIGNIFICANTLY EXCEEDED THE INCOME WHICH IT PRODUCES FROM ALL SOURCES. FOR EXAMPLE, IN THE YEAR 1975, TOTAL INCOME WAS $475,463 AND TOTAL EXPENSES WERE $579,031. EXPENSES EXCEEDED INCOME BY $103,569. OF THE INCOME WHICH WAS RECEIVED, $247,000--OVER 50%--WAS RECEIVED FROM THE SALE OF TECHNICAL EXHIBIT SPACE. SINCE A LARGE PART OF THIS EXHIBIT SPACE WAS SOLD TO DRUG COMPANIES, IT IS OBVIOUS THAT THEIR PARTICIPATION IS IMPORTANT IN INSURING THAT IT IS ECONOMICALLY POSSIBLE TO HOLD OUR ANNUAL MEETING.

BEFORE CONCLUDING, I WOULD LIKE TO MAKE ONE ADDITIONAL POINT. ALTHOUGH DRUG COMPANY SUPPORT OF SCIENTIFIC AND COMMERCIAL EXHIBITS HAS BEEN AN IMPORTANT FACTOR IN THE ACADEMY'S ABILITY TO PROVIDE HIGH QUALITY, CONTINUING EDUCATION TO OUR MEMBERS, IT HAS NOT PRODUCED THE RESULT THAT THE TOTAL EDUCATIONAL CONTENT IS CONTROLLED BY THE DRUG INDUSTRY. SCIENTIFIC EXHIBITS ARE BUT ONE OF NINE ELEMENTS IN OUR SCIENTIFIC PROGRAM. ATTACHED TO THIS STATEMENT AS EXHIBIT C IS A LIST OF THESE NINE PROGRAM ELEMENTS. WHILE IT IS TRUE THAT THE INCOME DERIVED FROM THE SALE OF COMMERCIAL EXHIBIT SPACE TO PHARMACEUTICAL COMPANIES HAS PROVIDED FINANCIAL SUPPORT FOR THE ENTIRE SCIENTIFIC ASSEMBLY, IT IS ALSO TRUE THAT THE PURCHASE OF EXHIBIT SPACE DOES NOT GIVE SUCH PHARMACEUTICAL COMPANIES ANY CONTROL OVER THE EDUCATIONAL CONTENT OF THE NINE PROGRAM ELEMENTS WHICH COMPRISE THE SCIENTIFIC PROGRAM.

MR. CHAIRMAN, WE HAVE ATTEMPTED TO COVER EACH OF THE AREAS OUTLINED IN YOUR INVITATION TO APPEAR BEFORE THIS SUBCOMMITTEE.

WE HOPE THAT THE INFORMATION WHICH WE HAVE PRESENTED WILL BE OF BENEFIT TO YOU IN YOUR DELIBERATIONS. AT THIS TIME, DR. HUFFMAN AND I WILL BE HAPPY TO ANSWER ANY ADDITIONAL QUESTIONS WHICH YOU MIGHT HAVE.

-12-

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EXHIBIT A

AMERICAN ACADEMY OF FAMILY PHYSICIANS

THE SCIENTIFIC EXHIBIT

for the

Twenty-eighth Annual Scientific Assembly—September 20-23, 1976

BOSTON

FOREWORD

Each year, the scientific exhibits acquire increasing stature as a teaching discipline at our Assembly. Members are anxious to learn about new developments in medicine, and the Assembly program provides nonconflicting hours for exhibit study. Hence, the scientific exhibitor, if selected for the Academy Assembly, has a unique opportunity to teach.

In order to select the most suitable exhibits, certain criteria have been developed, including application to family practice as well as the usual standards of quality, legibility, scientific accuracy and ethical content.

Regrettably, it will be impossible to accept every exhibit offered to us. The more information you give on the attached application form, the easier it will be for us to select those exhibits of the greatest value to family physicians.

We appreciate your desire to join us in Boston. If we are unable to accept your exhibit, we encourage you to apply again in future years. If your exhibit is selected, we look forward to meeting you. We will make an earnest effort to provide a pleasant and worthwhile experience.

JAMES M. HURLEY, M.D., *Chairman*
Subcommittee on Scientific Exhibits

DEADLINE FOR APPLICATION—May 1, 1976

ACADEMY REGULATIONS FOR SCIENTIFIC EXHIBITS

1. The author or authors of the exhibit shall be fully responsible for the content, arrangement and presentation of all data in the exhibit.

2. Except in rare emergency situations, one of the authors of the scientific exhibit is expected to be present at the Assembly and in charge of the exhibit. If this is not possible, the applicant (exhibitor) is expected to inform the Academy promptly. The exhibit *must* be staffed by a qualified demonstrator during the "open" hours of the Assembly.

3. An exact transcript of all copy to appear in the exhibit together with a photograph or sketch, must be submitted before any favorable action may be taken on the application. If this cannot be done, a full outline must be given in the attached application.

4. Accurate, well-documented and up-to-date pharmaceutical research provides needed medical information. Exhibits based on such data are welcome; however, they must be presented in an ethical manner. Comparisons of efficacy, contraindications, modes of administration, *et cetera*, between competitive drugs shall be fully presented. Editorial conclusions should be made with ethical restraint.

5. Only generic names of drugs and other products may be used in the body of the exhibit. The first appearance of the generic name may be marked by an asterisk, with the brand name of the product as a footnote at the bottom of the exhibit, not exceeding ½" in height. (When a number of different products appear in the exhibit copy, exception to this rule may be granted at the discretion of the committee.) Commercial product names are not permitted in the title of an exhibit.

6. All generic designations of competitive drugs shall be used with equal emphasis on height, weight, color or type face of lettering.

7. Devices designed only to attract attention, through the use of motion, sound, excessive illumination or flagrant use of color should be avoided. This does not imply that an exhibit may not be attractive or employ any legitimate device to emphasize or clarify its data.

8. Sound movies or recordings may be used only if they do not detract from adjoining exhibits, and are located in the exhibit to avoid blocking the aisle. Intent to use such devices should be clearly stated at the time application is made.

9. No reference to or credit for financial aid shall be indicated in the exhibit.

10. Sales representatives of commercial firms are not permitted to staff a scientific exhibit at any time.

11. Exhibitors are encouraged to discuss their work with visiting physicians, both singly and in groups. It is expected that these discussions, like the exhibit itself, will be marked by ethical restraint and that presentations will be made in a manner that in no way disrupts or detracts from adjoining presentations.

Scientific Exhibit Application

AMERICAN ACADEMY OF FAMILY PHYSICIANS

TWENTY-EIGHTH ANNUAL SCIENTIFIC ASSEMBLY

BOSTON, SEPTEMBER 20-23, 1976

The following information is necessary to guide the Subcommittee on Scientific Exhibits in its selections and to assure adequate physical facilities at the Assembly for accepted exhibits. Please read carefully, fill in completely (type if possible), and return, *in duplicate* (see address on last page) no later than May 1, 1976.

1. EXACT EXHIBIT TITLE _____

2. PERSONNEL (highest degree attained)

   A. Senior Exhibitor _____

   Title _____

   Organization _____

   Address _____ Zip _____

   Telephone _____

   B. List all other physicians and senior scientists who conducted the research on which the exhibit is based; only these names will appear in the printed program:

   Name _____

   Title _____

   Address _____

   _____

   C. Contact (Individual to whom correspondence should be addressed, if not the senior exhibitor)

   Name _____

   Address _____ Zip _____

   Telephone _____

3. BOOTH DESCRIPTION FOR PROGRAM—Please describe the exhibit in 50 words, as you would like to have it appear in the printed Official Program. The Academy reserves the right to edit your copy if necessary. Please type double-spaced.

This application must be returned in duplicate

4. EXHIBIT INCLUDES: Charts_____ Posters_____ Photos_____ Microphotos_____
X-rays_____ Other Transparencies_____ Machines_____ Specimens_____
Movies_____ View Boxes_____ Other Equipment_____

5. BOOTH SIZE: Scientific booths are divided by drapery hung on pipes; side walls are 6 feet deep; all walls are 8 feet high. Floor space is limited—please be accurate in giving your exhibit dimensions. Exhibits exceeding 8 feet in height or 6 feet in depth are not accepted by special ruling.

MAXIMUM LINEAR BOOTH LENGTH desired. . . . . _____feet

Can use MINIMUM LINEAR LENGTH of . . . . . _____feet

Is this a pre-built exhibit? Yes ☐ No ☐

6. COMMERCIAL PRODUCTS: In determining the educational value of an exhibit, it is helpful to the committee to know if branded pharmaceuticals were used in the clinical work on which the exhibit is based (see Academy regulations 5 and 6 on page 2 of this application). Copy concerning drug research must be presented in an ethical manner without commercial emphasis on the brand name and without deliberate disparagement of commercial products. Failure to abide by this ethical restriction, in spirit as well as letter, will be considered cause for removal of the exhibit from the auditorium.

Product (Generic Name)	Brand Name	Is Brand Name Used In Exhibit?	Manufacturer

Name of sponsor_____

7. HISTORY: Has this exhibit been presented in whole or in part at any previous Assembly of the Academy?

Yes ☐ No ☐ If yes, when?_____

Has it been presented at any other medical society meeting?_____

8. EXHIBIT COPY: Please enclose, in duplicate, booklet or folder giving copy as it will appear in the exhibit. If not available, detailed information must be given in the space below concerning the research on which this exhibit will be based or the teaching material to be presented. If complete exhibit copy is submitted, it is not necessary to fill in the space below.

Exhibit Description

A. OBJECTIVES

B. FINDINGS

73-617 O - 76 - 22

C. CONCLUSIONS

The undersigned agrees that (1) this exhibit will be staffed by qualified demonstrators during the "open" hours of the Assembly, (2) that all demonstrations will be conducted in an ethical, professional manner, and (3) that no commercial item will be sold or offered for sale by exhibitor or other personnel in the Scientific Exhibit Section.

Signed_____

This form, when completed, shall be forwarded promptly to Mr. Edward E. Daleske, Secretary, Committee on Scientific Program, American Academy of Family Physicians, 1740 W. 92nd St., Kansas City, Missouri 64114.

EXHIBIT B

American Academy of Family Physicians
SCIENTIFIC EXHIBIT EVALUATION FORM

Senior Exhibitor _____
Exhibit Title _____
Meeting _____
Exhibit Number _____

		Excellent (8)	Good (6)	Fair (4)	Poor (2)
I.	Value to Family Practice	_____	_____	_____	_____
		Excellent (4)	Good (3)	Fair (2)	Poor (1)
II.	Educational Value (purpose, conclusions)	_____	_____	_____	_____
		Excellent (4)	Good (3)	Fair (2)	Poor (1)
III.	Appearance or Format (clarity, design, esthetic value)	_____	_____	_____	_____
		Excellent (4)	Good (3)	Fair (2)	Poor (1)
IV.	Scientific Content (valid, properly structured, timely)	_____	_____	_____	_____
	Subtotals	_____	_____	_____	_____

TOTAL: _____ points out of a possible 20 points.

Exhibitor staffing: Needed _____ Not Needed _____ Exhibitor present: Yes _____ No _____

_____
(Evaluator's name)

EXHIBIT C



HYNES VETERANS AUDITORIUM, BOSTON

SCENE OF EXPANDED MEDICAL EDUCATION OPPORTUNITIES

The 1976 Scientific Assembly program offers nine diverse modalities and more than 100 practical subjects to suit your learning needs and preferences. You can build a personalized curriculum around the following elements planned by fellow physicians who serve on the Committee on Scientific Program:

- Clinical Seminars
- Continuing Education Courses
- Dialogue
- Live Teaching Demonstrations
- Programmed Instruction
- Taping and Strapping Demonstrations
- Fractures Demonstrations
- Scientific Exhibits
- Lectures

Participating physicians can earn one hour of prescribed AAFP credit for each hour spent in any portion of the scientific program (excluding the viewing of scientific exhibits). By carefully scheduling your time (that is, from 8:00 a.m. to 5:00 p.m. Monday, Tuesday and Wednesday of the Assembly and 8:00 a.m. to noon on Thursday), it would be possible to earn more than 30 approved continuing medical education hours.

Each of the nine program elements is described below. Advance registration is advised for the first two, which also involve a fee. The other seven elements are free of charge. The Committee on Scientific Program suggests that you register immediately for any Seminars or Continuing Education Courses you may wish to participate in. Having received notification that your registration is complete, you will then be able to plan the rest of your time to best advantage.

STATEMENT BY:

Mr. J. E. Raeben, President, Visual Information Systems,

Division of Republic Corporation

before Subcommittee on Monopoly,

U. S. Senate, Small Business Committee,

May 10, 1976

Summary

The following statement identifies Visual Information Systems (VIS) activities. I briefly describe those concerned with medical communication or education and, at greater length, deal with those likely to be of special interest to the Subcommittee. As each activity is discussed, an effort is made to respond in that section to the Subcommittee's concerns as suggested in its invitation letter of April 6.

1. About VIS

Our company was formed in October 1962 and became part of Republic Corporation in 1969. Its first activities were in closed circuit television at conventions. Since then we have provided business and university instruction on videotape and made smaller forays into sports and entertainment. VIS' principal activities and those of interest to the Subcommittee are in medicine and this statement is limited to them.

2. VIS medical activities are: Convention Television, begun in 1962;
The Network For Continuing Medical Education (NCME), begun in 1965;
Audio Visual Education in Neurosurgery (AVENS), begun in 1969;
Amercian Academy of Ophthalmology And Otolaryngology Continuing Education on Television, begun in 1971;
Physicians Radio Network (PRN) begun in 1974

3. Convention Television

Convention T.V. began in 1962 at a meeting of the American Dental Association. The service, to which we attach the trade name Hotelevision, enabled

dentists in their own hotel rooms to watch programs on dentistry prepared by the Association. Since that date, Hotelevision and other forms of convention television have been supplied by us to about 70 Association meetings, predominately for physicians. All programming is developed under the direction of the Association and the service has been supported by perhaps a dozen pharmaceutical companies in all. When product messages accompany the telecast, they are always unrelated to the content of the program. To my knowledge, no studies have been done to measure the impact on physicians prescribing practices.

4. NCME

The Network For Continuing Medical Education (NCME) was established in 1965 with the support of Roche Laboratories. This was the first effort in any field to use the emerging video technology for publishing. We employ a "journal" concept with three "articles" or programs in each reel. Unlike the way motion pictures had been used, we put forward the then un-usual notion that an audience as small as a single viewer was adequate and that viewing should take place at the convenience of the viewer, not the exhibitor. These ideas today seem commonplace. In 1972, with the introduction of the videocassette machine, a cassette subscription plan was developed. This has enabled hospitals to build their own videocassette libraries and offer them as community resources. Today all 700 NCME hospitals subscribe, paying $1,200 to $1,600 yearly. The remainder of the cost is borne by Roche Laboratories. About 145,000 physicians have regular access to NCME programs.

5. In 1965, when the Network began, we hoped the programs would be produced either by medical schools, just then acquiring videotape equipment, or by government facilities, such as, for example, the National Naval Medical Center, or Walter Reed Army Medical Center. In short, we expected to vest responsibility for producing the programs in the academic medical community. That programming, however, proved to be, too often, not suitable for distribution for technical or production reasons. It became more effective for NCME to provide direct production assistance to the physicians, at those same medical centers, whose work was to be the subject of the programs. (Programs produced by medical centers are, however, still occasionally distributed on NCME.) The Network presently distributes each year 69 different programs. These are assembled onto 23 one-hour reels, and are sent out one reel every-other-week, except monthly in summer. I would like to put into the record now this catalog which lists NCME's programs and their participants.

6. Program subjects and their content are determined through the following process: First of all, we have a National Board of physicians concerned with education. That Board includes such persons as Lowell Coggeshall, formerly Vice President at the University of Chicago and the nation's first Assistant Secretary for Medical Affairs at Health, Education and Welfare , George Aagaard, one of the nation's first deans of continuing medical education, now Professor at Washington University School of Medicine and Edmund Pellegrino, Chairman of the Board, Yale- New Haven Medical Center. A complete list is provided in an appendix to this statement. This Board provides overall direction to the Network. For example, at the Board's meeting

later this May, it will discuss steps NCME must take to meet the accreditation requirements of various accrediting bodies; another group, the Medical Advisory Committee, is made up of physicians, also listed in the appendix, who meet regularly to select specific program subjects and often to suggest participants for them. The participant himself then determines the specific content of his program, although NCME staff guide him on ways to make programs more visual. The staff also sometimes recommends for or against programs based on their suitability for television. The sponsor does not ask us and we do not volunteer to produce particular programs or to treat programs in any particular way. As with any publisher or broadcaster, our goal is to provide subjects likely to be of interest and value to large audiences, because if they are not, doctors will not watch, hospitals will not subscribe and our sponsor will cease his own support. If one were to view these programs - and they are the silent witnesses here - he would find that large numbers of them do not deal with drugs and that when they do, it may be as much against as for their greater use.

7. Product Messages appear preceding each program on the reel. The schedule of product messages is prepared months in advance by the sponsor without knowledge of the programs they are to accompany. However, when the reel is assembled, our staff takes care to avoid even the accidental juxtaposing of product messages with programs dealing with the same clinical subject. No study has ever been done to determine the extent to which product messages on NCME affect drug prescribing

practices. And, of course, no studies have been done on the influence of the programs themselves on drug prescribing. Few of these programs, if any, would lend themselves to such study. Surveys are done to determine if physicians watch our programs and find them useful. But although our audience attendance figures appear to us gratifyingly high, the sincerest form of flattery is the fact that 700 hospitals, including many of the nation's important clinical centers, pay their own money to subscribe. Perhaps I should add here that NCME has rendered assistance to the National Library of Medicine, has disseminated information to physicians at the request of the National Center for Disease Control and for many years supplied programs without cost to VA and other government hospitals and institutions.

8. AVENS

Audio Visual Education In Neurosurgery (AVENS) is a videocassette, monograph, and slide service, in popular terms, a multi-media system. Subjects and their presenters are chosen by the Society of Neurological Surgeons which began the project with the support of a grant from the John and Mary R. Markle Foundation. The programs are sold to neurosurgical training centers and to individual neurosurgeons. But as the number of subscribers is small (about 125 have bought the first series of 10 programs), the project is far from self-sustaining. Because the distribution and production apparatus constructed for NCME is available to us, we can continue AVENS at our own expense and we are doing so. Some of the programs are said to be among the better teaching of any sort ever done in neurosurgery. Without foundation support, however, AVENS could not have been begun and without our own assistance, I am not sure it could continue.

9. AAOO

The American Academy Of Ophthalmology And Otolaryngology established in 1971 a continuing education series in videocassette form. We act as the Academy's video publisher. Production monies have been provided by the Academy. Program subjects and presenters are selected by the Academy, and the majority of programs are produced by the presenter, often with university television facilities. The programs are sold to ophthalmologists and otolaryngologists. About 200 subscriptions have been sold to each group. The cost to duplicate, market and distribute these programs is borne by VIS. The service will require a very substantial increase in sales to be self-sustaining. As with AVENS, we are pleased to be associated with the project because, in the long-run, it should enhance our position as a publisher of good medical education in television form. As with AVENS, however, it raises the question, can such education exist, at present, and in these forms, without subsidization?

10. PRN

Physicians Radio Network, or PRN, is a 24-hour a day, 7-day a week medical news, information and education service for physicians. It is transmitted over special FM frequencies. It requires a special receiver provided by us without charge to the doctor. Research on the technical feasibility of PRN was begun by us in 1965. Over a period of several years I tried unsuccessfully to interest both the federal health establishment and major medical associations either in supporting this project, or in joining me in a common search for support. In April, 1974 broadcasting was started in two cities to test whether physicians would listen and whether they might pay for the

service. We learned that a physician-subscription approach was impractical. However, listenership was quite high and it encouraged us to go to industry for support. We offered PRN both as a new advertising medium and one with high prospective social utility. Today, 5 companies advertise on PRN. They are: CIBA Pharmaceutical Company, Merck Sharp & Dohme, Pfizer Laboratories Division, Roche Laboratories, Smith Kline & French Laboratories and S.K. & F. Company. We hope to have more sponsors in time. There are eight one-minute advertising messages in each hour. These fall in a strict rotation planned weeks ahead, clearly with no knowledge of the news items they will eventually be adjacent to. The service now broadcasts in 23 cities to about 21,000 physicians. By year end we should be on-the-air in 30 cities, reaching 75,000 physicians. Our longer term goal is about 150,000 physicians, half the nation's doctor population but far more than half of those active in patient care. At present, PRN programming is a single hour, repeated 24 times and changed each day. In each city, local medical news is also broadcast every hour, generally with the cooperation of the local medical society. This summer we expect to begin updating the programming twice each day and we can broadcast an important bulletin anytime. Although PRN has been primarily a news service, we are now beginning a continuing education series as well. This is a program, 15 minutes or so long, and repeated 20 to 30 times in the week. It is accompanied by printed self-assessment forms mailed to the physician. If our audience finds this programming useful, we think it should be a regular part of the radio service. If practicable, we may offer blocks of time for teaching purposes to the general and special medical assocations which presently have the training and education responsibility

for their constituencies.

It seems to us surprising that until PRN, medicine had no medium to transmit important information quickly. If regional or national health crises threatened, there was no way, except in sometimes inappropriate public media, for medicine to communicate about it. Setting aside the matter of emergencies, if a scientific advance of any importance takes place, many months normally elapse from authorship to publication. Many more months elapse before confirmation or rebuttal by others. Our hope is that by accelerating this process, by telescoping into a few months, or even weeks, a dialogue that now may take years, PRN can have a genuine impact on medical progress not achievable in any other way.

PRN news is reported by medical correspondents throughout the United States and by a permanent office staff of experienced medical journalists. Decisions on which 25 or 30 stories make up the daily broadcast are made solely by the editorial staff. Advertisers play no part. We have made a considerable effort to determine how much physicians listen to PRN and I believe we have excellent information on this, better perhaps than that possessed by many other media used for medical communication or education. We assume also, that sponsors will ultimately try to employ advertising measurement techniques on PRN messages.

11. It is certainly important to protect education from undue bias. And, it is always reasonable to fear that he who pays the piper may call the tune. That, however, has not been our experience and I hope that my presentation bears this out.

Thank you.

Appendix A

NCME

BOARD OF CONSULTANTS

George N. Aagaard, M.D. (Chairman)
Professor of Medicine
University of Washington
School of Medicine

Richard M. Bergland, M.D.
Chairman, Neurosurgery Department
The Milton S. Hershey Medical Center

Edward Bird, M.D.
Division Cancer Control
National Cancer Institute

Lowell T. Coggeshall, M.D.
Trustee and Vice-President Emeritus
University of Chicago

Marvin Johnson, M.D.
St. Joseph Hospital
Medical Education Department
Denver, Colorado

Attallah Kappas, M.D.
Professor and Physician-In-Chief
Rockefeller University
New York, N.Y.

Robert Moseley, M.D.
Professor and Chairman
Department of Radiology
University of New Mexico
School of Medicine

MEDICAL ADVISORY COMMITTEE

James J. Bulger, M.D.
Family Physician
115 College Park Medical Center
Great Falls, Montana

Dr. Bulger is a practicing family physician with a special interest in cardiology. He represents our viewing audience in Montana. He advises us on topics that hold interest at his hospital in Great Falls and tells us of presenters who are good on camera. He is also asked, at times, to review scripts prior to production to make certain that all points are covered.

George Eckert, D. O.
Doctor's Hospital
Columbus, Ohio

Dr. Eckert is the single osteopathic physician on our medical advisory committee. He has a private practice and recommends topics of interest to himself as well as his colleagues. In addition, he reports to us on the reception of NCME programs at Doctor's Hospital in Columbus, Ohio.

P. R. Garver, M.D.
Diplomate, Board of Family Practice
Medical-Dental Building
Show Low, Arizona

Dr. Garver has a large general practice in Arizona and is constantly on the look-out for continuing medical education opportunities. We depend on him for advice as he uses NCME as a primary source of postgraduate education for himself and the other physicians and paramedics in the community.

Alan L. Goldberg, M.D.
Family Physician
2805 Grand Concourse
Bronx, New York
Assistant Visiting Physician, Department of Medicine
Bronx Lebanon; Department of Electrocardiology

Dr. Goldberg is a family physician with a large, busy practice and is active with the state chapter of the American Academy of Family Physicians. He represents our audience and advises us on the appropriateness of material we present. He frequently screens programs prior to distribution.

Marvin Johnson, M.D.
Director of Medical Education
St. Joseph Hospital
Denver, Colorado

Dr. Johnson wears several hats on the medical advisory committee. He is a practicing surgeon and a director of medical education at his hospital in Denver. In addition, he is a member of the AMA's Council on Scientific Assembly and the Committee on Continuing Medical Education. Dr. Johnson provides us with topics that appeal to his hospital staff, names of presenters considered very good in the western United States, and advice on improving NCME programs to meet the requirements for continuing medical education credit of the AMA.

Mortimer J. Lacher, M.D.
Attending Physician, Medical Oncology Service
Memorial Hospital for Cancer and Allied Diseases

Dr. Lacher is NCME's resource for developments and presenters in the field of cancer.

Richard D. Levere, M.D.
Professor of Medicine
Chief of the Hematology Division
Vice Chairman, Department of Medicine
Assistant to the Dean for Interdisciplinary Programs
State University Hospital, Downstate Medical Center

Dr. Levere provides the NCME staff with program ideas and teachers in the field of internal medicine and hematology. He frequently screens the programs prior to distribution.

Harold C. Neu, M.D.
Associate Professor of Medicine
Associate Professor, Pharmacology
Head of Infectious Diseases
Columbia University College of Physicians and Surgeons

Dr. Neu, as an academic physician with a small private practice, has wide-ranging contacts with specialists in many fields, has an interest in continuing medical education by videotape and first-hand experience with it, and has an ability to choose on-camera talent--that is experts in their medical field with a flare for on-camera presentation.

Edmund D. Pellegrino, M.D.
Chairman of the Board
Yale-New Haven Medical Center

Dr. Pellegrino, as an educator and practicing physician, has a special interest in the use of videotape for postgraduate education of physicians. He frequently recommends topics for use on NCME as well as appropriate teachers.

Charles Plotz, M.D.
Director, Office of Continuing Medical Education
Professor of Medicine
Chairman of the Department of Family Practice
Downstate Medical Center

Dr. Plotz has a unique role on our advisory committee. As a director of continuing medical education and chairman of the department of family practice, he advises us on the kinds of programs that appeal to practicing physicians at his institution and often acts as a sponsor for special continuing medical education credit. He is an authority in the field of rheumatoid arthritis and provides us with program topics and experts in the field as teachers for NCME.

Isadore Rossman, M.D.
Medical Director of the Home Care Department
Associate Professor of Community Medicine
Albert Einstein Medical College

Dr. Rossman was selected for the committee because he has a private internal medicine practice and an interest in geriatric medicine. He advises us on topics in his area of interest.

Rafael C. Sanchez, M.D.
Associate Dean
Louisiana State University School of Medicine
New Orleans, Louisiana

Dr. Sanchez, as a former practicing internist and family physician, and as a current director of continuing medical education for LSU School of Medicine, advises us on topic selection, presenters, and developments in the field of continuing medical education. LSU produces its own continuing medical education videotapes and provides them and NCME videotapes to a network of hospitals in and around New Orleans. Dr. Sanchez is a key source of information on the appropriateness of topics for our NCME audience.

Travis Smith, M.D., Chief of Staff
Hendrick Memorial Hospital, Abilene, Texas

Dr. Smith advises us on the reception of programs by community hospital MD's.


NCME
Videocassette
Library
A catalog of television programs for physicians and other health professionals, available to institutions participating in the Network for Continuing Medical Education. Supported by Roche Laboratories.
NCME Videocassette Library
1976 Edition
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THE NCME MASTER LIBRARY

The NCME Master Videocassette Library, with approximately 700 medical education programs, is the most extensive collection of telecasts available to hospitals and medical schools. Presented by leading physicians and medical researchers, many of these programs were produced at medical centers and are available to NCME subscribers to supplement their basic regularly scheduled service. This catalog also includes listings from the American Medical Students Association (AMSA) Video Journal and the American College of Physicians Television Hospital Clinics. Code letters AMSA and ACP before the catalog numbers identify these programs.

HOW TO ORDER

Requests for programs may be submitted on order forms in the back of the catalog. Please allow six weeks for an order to be delivered. Videocassettes are shipped at library rate. (Faster delivery can be made by airshipment, provided the borrower agrees in writing to assume the additional cost.) Tapes may be returned library rate and should be insured for $50.

Subscribers to NCME's videocassette service may purchase individual titles for addition to their own Cassette Resource Center. Rentals are for a two-week period. (A modest fee is charged for master library videocassettes to defray the cost of tape stock, duplication, shipping and inspection.)

NON-CASSETTE EQUIPMENT

If the equipment to which you have access uses tape in any form other than the videocassette and you want information on how to use NCME, please write or phone.

PROGRAM REVIEW CARD

A program review card accompanies each Master Library videocassette. NCME asks that the person for whom the order was placed fill out the program review card after using the program. As re-evaluation of these programs is a continuous process, your return of the program review card is essential to helping NCME maintain this important source of televised medical information.

Whenever a program, in the opinion of the reviewer, has some important inadequacy, there will be no charge. However, the reviewer must state the reason on the program review card, and return it within two weeks after receipt of the videocassette.

PROGRAM SUBMISSIONS INVITED

Should you wish NCME to consider a program produced by your institution for inclusion in the catalog, NCME will arrange to screen it for possible national distribution. In making your programs available, you increase the effectiveness of videocassettes in continuing medical education.

Tape library services are made possible by Roche Laboratories through its support of the production and regular distribution of all NCME telecasts.

Duplication or reproduction in any form of all or any portion of the videotape programs listed in this Catalog without the express written consent of NCME is not permitted.

The Network for Continuing Medical Education
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New York, New York 10023
(212) 541-8088

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USE OF NCME FOR CONTINUING EDUCATION CREDIT

Increasing numbers of professional organizations, state medical associations and specialty groups are encouraging—and in some cases requiring—their members to participate in continuing education activities. The use of NCME programs is widely acceptable toward partially meeting the requirements of these various organizations. A review of some professional CME requirements may be useful.

To earn the Physician's Recognition Award of the American Medical Association, physicians must participate in 150 hours of continuing medical education during a three-year period. The AMA divides CME activities into six categories and five sub-categories. NCME programs are routinely accepted in category 5A which has a limit of 22 hours. Once category 5A is filled, they are acceptable in category 2 up to a limit of 45 hours. Specially designated NCME programs are acceptable for category 1 credit under provisions governing the individual use of audio visual materials. Credit of this type is limited to 45 hours in total.

The American Academy of Family Physicians requires 150 hours in a three-year period and divides its activities into two categories: Prescribed and Elective. Prescribed refers to activities specifically produced for the field of Family Practice while Elective comprehends all other programs. NCME programs are usually accepted as Elective but sometimes as Prescribed. Total hours required by other professional societies may vary.

The American Osteopathic Association continuing medical education program specifies that a particular category of credit be determined by the Director of Medical Education at the osteopathic hospital or school where the activity is conducted. The DME also forwards attendance rosters to AOA headquarters, and participation is entered on the computer record of the members involved. NCME programs are most often accredited under category 2A or 2D at the recommendations of the DME.

NCME's service includes a self-assessment quiz which physicians use to evaluate their knowledge of teaching points made in NCME programs. Upon completion of the quiz, it may be retained for the physician's own record or, if desired, sent to NCME for confidential filing.

> Not all NCME programs have self-assessment quizzes. When those programs which do are ordered from the catalog, one copy of the self-assessment quiz will be enclosed. You may duplicate the quiz as your needs require. Please make sure the Social Security number is clearly written if the quiz is returned for credit.

To receive credit, the quiz *must* be returned to NCME. The quiz can be mailed directly to NCME or given to the Director of Medical Education, the Medical Staff Secretary, Chairman of the Education Committee or another appropriate person who collects all quizzes and sends them in bulk to NCME every two weeks. Physicians may request their NCME activity records at anytime.

If there are subsequent questions, please phone NCME at (212) 541-8088.

TABLE OF CONTENTS

Adolescent Medicine 4
Allergy 5
Anesthesiology 6
Biochemistry 8
Cardiology and Cardiovascular Disease 9
Dermatology 17
Drug Abuse 19
Embryology 21
Emergency Medicine and Trauma 22
Endocrinology 27
Family Practice 30
Gastroenterology 30
Genetics 34
Geriatrics 37
Hematology 37
Hospital Medicine 41
Infectious and Parasitic Disease 43
Instrumentation and Computers 47
Intensive Care 51
Internal Medicine 53
Legal Medicine 58
Musculoskeletal and Connective Tissue Disorders 60
Nephrology 62
Neurology 63
Nursing and Paramedical Programs 69
Nutritional and Metabolic Disorders 73
Obstetrics and Gynecology 75
Oncology 80
Ophthalmology 83
Orthopedics 84
Otolaryngology 88
Pathology 89
Pediatrics 90
Pharmacology 98
Physiology 102
Practice Management and Medical Economics 102
Psychiatry 104
Public Health 112
Pulmonary Disease 116
Radiology 119
Rehabilitation and Physical Medicine 122
Reproduction, Fertility and Sexuality 125
Space Medicine 128
Surgery (includes general, abdominal, cardiovascular, orthopedic, neurologic, pediatric, plastic, thoracic, etc.) 129
Topical and Historical Programs 134
Urology 138
Virology 140

ADOLESCENT MEDICINE

A NEW HOSPITAL APPROACH TO ADOLESCENT CARE, with Michael I. Cohen, M.D., Director of the Division of Adolescent Medicine; and Iris Litt, M.D., Assistant Director. The division is part of the Department of Pediatrics, Montefiore Hospital in New York City.

What advantages are there to an adolescent center within a hospital? With the help of young patients, Dr. Cohen and Dr. Litt show many benefits of establishing an adolescent unit as a distinct service of a general hospital.
(17 minutes) (in color) **1411107**

DEALING WITH EPILEPSY: THE SOCIAL PROBLEM, with Mary Louise Scholl, M.D., Associate Pediatrician, Massachusetts General Hospital, and Assistant Professor of Pediatrics, Harvard Medical School, Boston, Massachusetts. With proper physician guidance, epileptics can live nearly normal lives. Dr. Scholl reviews typical problems with three patients and explains what physicians can do to help.
(21 minutes) (in color) **0415130**

DRUG ABUSE: RECOGNIZING AND TREATING ACUTE REACTION TO AMPHETAMINES AND SEDATIVE HYPNOTICS, with David E. Smith, M.D., Instructor, Department of Pharmacology, University of California School of Medicine, San Francisco; and Medical Director, Haight-Ashbury Medical Clinic.

Why do most young drug users avoid family physicians when their drug experiences get them in trouble? Dr. Smith discusses this problem with several drug users. He also gleans detailed descriptions from the young users of drug "trips" and "bad trips." Finally, Dr. Smith describes the kinds of adverse reactions to amphetamines and sedative hypnotics, and outlines suggested therapy. (19 minutes). **0411822**

DRUG ABUSE: RECOGNIZING AND TREATING ACUTE REACTIONS TO HALLUCINOGENS, with David E. Smith, M.D., Instructor, Department of Pharmacology, University of California School of Medicine, San Francisco; Medical Director, Haight-Ashbury Medical Clinic.

In this telecast, experienced young people candidly discuss "bad trips" and other adverse symptoms conected with drug abuse. The program concludes with a concise rundown of recommended therapy. (19 minutes). **0411923**

GETTING A FIX ON HEROIN. Treatment of heroin-addicted patients is clouded by popular misconceptions and mythologies about heroin use and treatment. SAMA cameras take you to drug abuse treatment facilities in New York and San Francisco to explore the problems of the drug addict and the problems of treating him.

Speaking for SAMA is Dahlia Kirkpatrick, a third-year student at Yale who discusses the issues with Dr. Herbert Kleber, Director of the Drug Abuse Unit of the Connecticut Mental Health Center. (34 minutes) (in color) **SAMA 2810453**

INFECTIOUS MONONUCLEOSIS, with James C. Niederman, M.D., Associate Clinical Professor of Epidemiology and Medicine, Yale University School of Medicine, New Haven, Conn. Diagnosis and treatment of an important clinical entity—plus some of the burgeoning research implications. (16 minutes) (in color) **0919525**

INSIDE THE DOOR: A FREE CLINIC. What's free about a free clinic besides the medical care? The spirit is. And the cooperation between professionals and students of all disciplines. SAMA cameras take you to The Door, a free clinic in New York City, that offers adolescents full medical care as well as legal, social and educational counseling. Medical students and physicians at The Door talk about how they believe free clinics can help humanize medical treatment.
(23 minutes) (in color) **SAMA 2810149**

MANAGEMENT OF ADOLESCENT SYMPTOMS. Methods of dealing with the problems of adolescents — particularly experimentation with sex and drugs — are demonstrated with groups of teenagers, by Steven R. Homel, M.D., Department of Pediatrics, Jefferson Medical College and Hospital of Philadelphia, Pa.
(18 minutes). **1308004**

MEDICAL PROBLEMS OF ADOLESCENT HEROIN ABUSE, with Michael I. Cohen, M.D., Director of the Division of Adolescent Medicine; and Iris Litt, M.D., Assistant Director. The Division is part of the Department of Pediatrics, Montefiore Hospital in New York City.

An in-depth interview is conducted with a 14-year-old heroin addict under treatment. The youth describes his life with drugs followed by comments on the interview by the physicians.
(14 minutes) (in color) **1311025**

REACHING THE ADOLESCENT PATIENT. How can the physician "communicate" with the adolescent patient whose physical problems so often are linked to his emotional state? Using groups of youngsters at different age levels, Steven R. Homel, M.D., Department of Pediatrics, Jefferson Medical College and Hospital of Philadelphia, demonstrates techniques and methods that can be applied to general practice.
(18 minutes). **1807905**

RECOGNIZING ROLES IN JUVENILE DIABETES, with Donnell D. Etzwiler, M.D., Director, Diabetes Education Center, and Pediatrician, St. Louis Park Medical Center, Minneapolis. A pediatrician gives guidelines for early diagnosis and management of juvenile diabetes and shows which responsibilities of good control should be assigned to physician, health professional, and patient. This presentation was produced with the cooperation of the Council on Scientific Assembly of the American Medical Association.
(16 minutes) (in color) **1822934**

SCOLIOSIS: SIGNIFICANCE OF EARLY DETECTION, with Hugo A. Keim, M.D., Director of the Scoliosis Clinic, New York Orthopaedic Hospital; and David B. Levine, M.D., Associate Attending Orthopaedic Surgeon, Hospital for Special Surgery, and Clinical Associate Professor, Cornell University Medical College, New York City.

Approximately one adolescent in 10 has idiopathic scoliosis, which too often is not recognized until the curvature has become debilitating. Two orthopedists show what to look for on x-rays and how to conduct a simple office examination that makes earlier diagnosis and treatment easier. (15 minutes) (in color) **1914726**

THE DOCTOR-ADOLESCENT RELATIONSHIP. The adolescent frequently needs an outlet to express his doubts and concerns. How the physician can serve as this outlet during a clinical visit is demonstrated by Steven R. Homel, M.D., of the Department of Pediatrics, Jefferson Medical College and Hospital, Philadelphia, Pa.
(30 minutes). **0408319**

THE NEW HEROIN SCENE: A PERSPECTIVE FOR PHYSICIANS, with George R. Gay, M.D., Chief of the Medical Section and Director of the Heroin Clinic of the Haight-Ashbury Clinic, and Instructor in Toxicology at the University of California at San Francisco. A new group of drug abusers appears to be growing in numbers across the country — the "middle-class junkies" who are "dropping out" of society on heroin. The dimensions of the problem are explored by Dr. Gay. He also outlines treatment for the young abusers who are consumed by the chemical aspects of life rather than the human aspects. (22 minutes) **1413530**

THE THREAT OF SUICIDE. Those who commit suicide frequently attempt to communicate their desperation to their physicians. How can we be sensitive to these warnings, either open or covert, and how can we help a patient once his suicidal intentions are known? Two medical students discuss this issue with Harvey Resnik, M.D., Chief of the Mental Health Emergencies Section, National Institute of Mental Health and Clinical Professor of Psychiatry, George Washington University School of Medicine.
(27 minutes) (In color) **SAMA 2810757**

THRESHOLD TO ADDICTION, with Steven R. Homel, M.D., Department of Pediatrics, Jefferson Medical College and Hospital of Philadelphia. Dr. Homel discusses the reasons for drug addiction with two teen-agers and a young medical student. Their answers are candid, and offer insight into the attitudes of young people today.
(23 minutes) (in color) **2010206**

THE TREATMENT OF ACNE, with Paul Lazar, M. D., Associate Professor of Dermatology, Northwestern University Medical School, Chicago. When an adolescent with acne doesn't respond to topical therapy and a year of tetracycline treatment do you consider steroid therapy, topical or systemic, or acne surgery? Dr. Lazar recommends the progression to follow in treating acne. (19 minutes) (in color) **2020133**

ALLERGY

A CURRENT APPROACH TO POISON OAK/POISON IVY DERMATITIS, with William L. Epstein, M.D., Professor and Chairman, Department of Dermatology, University of California, San Francisco. Dr. Epstein demonstrates various treatments for severe and mild cases of poison oak and poison ivy dermatitis, along with the problems of re-exposure, cross-sensitivity with other substances and other dermatological complications. He also describes various de-sensitizing and hypo-sensitizing agents. This program will benefit the non-dermatologist.
(15 minutes) (in color) **0313364**

ANAPHYLACTIC REACTIONS TO DRUGS. Drug allergies in various degrees of severity have been found to occur in as many as 10 to 15 per cent of patients. But the one feared most by physicians because it can lead to death is the anaphylactic reaction. Bernard B. Levine, M.D., Associate Professor of Medicine, Department of Internal Medicine, New York University Medical Center, points out the clinical and pathologic signs of anaphylaxis and makes recommendation on treatment. (15 minutes). **0106312**

A PRACTICAL APPROACH TO ALLERGIC DERMATOSES IN CHILDREN, with Vincent J. Fontana, M.D., Professor of Clinical Pediatrics, New York University College of Medicine, New York City. Dr. Fontana demonstrates ways in which the general physician can arrive at positive diagnoses of both common and less-frequently encountered allergic dermatoses in children. He follows each diagnosis with its recommended treatment.
(18 minutes) (in color) **1614937**

ASTHMA — PART I — ETIOLOGY AND DIAGNOSIS. A new and more active differentiation between Asthma of known and unknown origin has been established by Irving H. Itkin, M.D., Chief, Department of Asthma Allergy, National Jewish Hospital and Research Center and Assistant Professor of Medicine, University of Colorado Medical Center. Dr. Itkin demonstrates the Quantitative Inhalation Challenge Apparatus, a new tool useful in distinguishing between Asthma of known and unknown origin, and explains how this differentiation is useful in the management of the disease. (18 minutes). **0107418**

ASTHMA — PART II — MANAGEMENT AND REHABILITATION. Treatment of the acute attack of asthma as well as a program directed towards long term management is illustrated by Irving H. Itkin, M.D., Chief, Department of Asthma Allergy National Jewish Hospital and Research Center and Assistant Professor of Medicine, University of Colorado Medical Center, Denver, Colorado. (18 minutes). **0107519**

CHILDHOOD ALLERGY: THE GREAT MASQUERADER, with Roland B. Scott, M.D., Professor of Pediatrics and Child Health, Howard University School of Medicine, Washington, D.C. Demonstration of the subtle early signs and symptoms of allergy which may eventually lead to serious, debilitating disease. Emphasis is on the child with "too many colds."
(20 minutes) (in color) **0321280**

HOW I APPROACH ATOPIC DERMATITIS, with Marion B. Sulzberger, M.D., Professor Emeritus, New York University School of Medicine, and Clinical Professor of Dermatology, University of California in San Francisco. Atopic eczema is a perplexing problem for the dermatologist and the general practitioner. Dr. Sulzberger describes the "chain multiple factor" approach to the disease in terms of occurrence and treatment.
(15 minutes) (in color) **0813419**

MANAGEMENT OF THE AMBULATORY ASTHMATIC CHILD, with Vincent J. Fontana, M.D., Director of the Department of Pediatrics, St. Vincent's Hospital; and Medical Director, New York Foundling Hospital, both in New York City. Dr. Fontana describes the steps that a general practitioner can take to alleviate the symptomatology of asthma, the prevention of future episodes and the avoidance of complications of bronchial asthma.
(16 minutes) (in color) **1314139**

PREDICTION OF PENICILLIN ANAPHYLAXIS BY SKIN TESTS. Screening patients to determine the presence of an allergy to penicillin by a simple test is shown by Bernard B. Levine, M.D., Associate Professor of Medicine, Department of Internal Medicine, New York University Medical Center. (18 minutes). **0607719**

THE TREATMENT OF BRONCHIAL ASTHMA, with Frank Perlman, M.D., Clinical Professor of Medicine, University of Oregon School of Medicine, Portland. How to treat the asthmatic patient early to avoid a potential respiratory crisis.
(16 minutes) (in color) **2020032**

WHAT'S CAUSING THE INCREASED INCIDENCE OF PHOTOSENSITIVE REACTIONS?, with John H. Epstein, M.D., Associate Clinical Professor of Dermatology, University of California at San Francisco, and Chief of Dermatology at Mount Zion Hospital and Medical Center in San Francisco. Increased public obsession with sunbathing and an ever increasing number of photosensitizers in our environment are reaching the stage where almost every physician can expect to see patients exhibiting phototoxic or photoallergic reactions. Diagnosis and treatment of the following are explored in this telecast: phototoxicity, photoallergy, exogenous photosensitizers, therapeutic and antibacterial agent reactions and plant-induced photosensitivity.
(15 minutes) (in color) **2313502**

ANESTHESIOLOGY

ACUPUNCTURE ANAESTHESIA AS PERFORMED IN THE PEOPLE'S REPUBLIC OF CHINA, with Samuel Rosen, M.D., Clinical Professor of Otology Emeritus, Mt. Sinai School of Medicine, and Consulting Otologist, New York Eye and Ear Infirmary, and Ben Park, Director of Programming, NCME. Films of major surgery under acupuncture anaesthesia, received from mainland China, provide the basis of Dr. Rosen's observations about acupuncture anaesthesia and how it works. His comments carry the added weight of his personal experience in witnessing 15 such operations in China late in 1971.
(21 minutes) (in color) **0115528**

ACUPUNCTURE IN WESTERN MEDICINE. Sung J. Liao, M.D., Associate Professor of Rehabilitative Medicine at Boston University School of Medicine, shows two medical students how he performs acupuncture therapy for the relief of pain and discusses the integration of this treatment into his practice of medicine.
(19 minutes) (in color) **SAMA 2810758**

THE ASPHYXIATING PATIENT: HOW AND WHY I INTUBATE, with Richard J. Ward, Professor, Department of Anesthesiology, University of Washington School of Medicine, Seattle. The steps involved in endotracheal intubation are reviewed and the procedure then performed on a surgical candidate.
(13 minutes) (in color) **0117933**

BIOMEDICAL ANALYSIS: SPEED, ACCURACY, SENSITIVITY

The uses of gas chromatography in the analysis of anesthetic effects, in blood studies and in obstetrics is shown by Harold B. White, Ph.D., Professor of Biochemistry; Leonard Fabian, M.D., Chairman, Department of Anesthesiology; and Winfred L. Wiser, M.D., Professor of Obstetrics and Gynecology, University of Mississippi Medical Center, Jackson, Miss.
(18 minutes). **0208405**

EPIDURAL BLOCK. Utilization of this procedure on a patient about to undergo cesarean section is demonstrated by Irving M. Pallin, M.D., Director of Anesthesiology, The Jewish Hospital of Brooklyn, New York. (17 minutes). **0502911**

LOCAL ANESTHESIA: THREE EFFECTIVE TECHNIQUES, with William C. North, M.D., Professor and Chairman of the Department of Anesthesiology, the University of Tennessee College of Medicine, Memphis. When and how to use three methods of local anesthesia for most minor office procedures. Includes: field block, digital block, intravenous regional block.
(14 minutes) (in color) **1220720**

MAINTAINING THE AIRWAY, with Richard Zepernick, M.D., Instructor in Anesthesiology, Department of Surgery, Tulane University School of Medicine. How to keep the patient breathing in an emergency. (16 minutes). **1301201**

SIM ONE — ANESTHESIOLOGICAL TRAINING SIMULATOR: PART I, DEVELOPMENT

An electro-mechanical, computer-controlled manikin has been developed to train physicians in anesthesiologic procedures at the University of Southern California School of Medicine. Describing this life-like device are J. S. Denson, M.D., Professor, Chairman, Division of Anesthesiology, and Stephen Abrahamson, Ph.D., Director, Division of Research in Medical Education, along with two engineers from the Aerojet Corporation, Azusa, California, the firm which built Sim One.
(25 minutes). **1904708**

SIM ONE — ANESTHESIOLOGICAL TRAINING SIMULATOR: PART II, APPLICATION

A resident physician performs an endotracheal intubation on Sim One, an electro-mechanical manikin built to duplicate human cardiopulmonary functions and used as a teaching tool at the University of Southern California School of Medicine. J. S. Denson, M.D., and Stephen Abrahamson, Ph.D., members of the school's faculty, conduct the program. (17 minutes). **1904809**

THE TEAM APPROACH TO CHRONIC PAIN, from the Pain Control Center of Temple University Hospital, Philadelphia. With Mary E. Moore, M.D., Ph.D., Assistant Professor of Medicine, Section of Rheumatology, and psychologist; Edward J. Resnick, M.D., Associate Professor of Orthopedic Surgery, and coordinator, Pain Control Center; Richard Eller, M.D., Associate Professor of Anesthesiology; Shavarsh Chrissian, M.D., Assistant Professor of Rehabilitation Medicine; and Marc Flitter, M.D., Assistant Professor of Neurosurgery.

Current theories of pain are leading to new methods for treatment. The techniques demonstrated here can work for you and your patients. This program was produced with the cooperation of the Department of Continuing Medical Education, Temple University.
(16 minutes) (in color) **2023841**

THERAPEUTIC ANESTHESIA FOR LOCALIZED PAIN, with William C. North, M.D., Professor and Chairman, Department of Anesthesiology, University of Tennessee School of Medicine, Memphis. A demonstration of two anesthetic techniques for localized pain: intercostal nerve block and myofascial block. The emphasis is on technique, risks, and complications.
(13 minutes) (in color) **2020934**

U.S. ACUPUNCTURE: STATUS REPORT 1973, with physicians and scientists from Boston; Cincinnati; Los Angeles and Canoga Park, California; New York City; St. Louis; and Washington, D.C. . . . moderated by John J. Bonica, M.D., Director of the Anesthesia Research Center, University of Washington, Seattle, and Chairman of the NIH ad hoc Committee on Acupuncture.

NCME explores the state of acupuncture research and practice in the U.S. with visits to acupuncture clinics, classrooms, and research labs and interviews with acupuncture researchers.
(30 minutes) (in color) **2118608**

BIOCHEMISTRY

BIOMEDICAL ANALYSIS: SPEED, ACCURACY, SENSITIVITY

The uses of gas chromatography in the analysis of anesthetic effects, in blood studies and in obstetrics is shown by Harold B. White, Ph.D., Professor of Biochemistry; Leonard Fabian, M.D., Chairman, Department of Anesthesiology; and Winfred L. Wiser, M.D., Professor of Obstetrics and Gynecology, University of Mississippin Medical Center, Jackson, Miss.
(18 minutes). **0208405**

CANCER MANAGEMENT: THE FUTURE OF CEA, with E. Douglas Holyoke, M.D., Chief of the General Surgery Service at Roswell Park Hospital, Buffalo, N. Y., interviewed by Alan L. Goldberg, M.D., family physician in private practice in the Bronx, N. Y. A look at the possible future of carcinoembryonic antigen as a diagnostic aid, a prognostic indicator, and as a monitoring test for patients with cancer. Dr. Holyoke uses case histories to illustrate the various uses of the antigen. (18 minutes) (in color) **0317875**

CELLULAR DISTURBANCES:
A NEW CONCEPT OF OBESITY

Some extremely obese patients may be incapable of losing weight and maintaining the loss because they may have acquired an excessive number of fat cells early in life. This is one of the findings of Jules Hirsch, M.D., Professor and Senior Physician to The Hospital, Rockefeller University, New York. (19 minutes). **0308704**

FINGERPRINTING MYOCARDIAL INFARCTION SERUM ENZYMES. How serum enzyme analysis is used to increase diagnostic accuracy and what prognostic data are furnished are demonstrated by John S. Ladue, M.D., Ph.D., Assistant Professor of Clinical Medicine, Cornell University School of Medicine, New York.
(15 minutes). **0608104**

GASTROINTESTINAL CYTOLOGY
A VALUABLE DIAGNOSTIC PROCEDURE: PART I

"Application and Results." Confirming a diagnosis without surgery through the use of Papanicolaou staining of cells from the gastrointestinal tract — with Charles Norland, M.D., Assistant Professor of Medicine, University of Chicago School of Medicine. (16 minutes). **0703303**

GROSS SYNOVIANALYSIS, a discussion of joint fluid analysis for the practicing physician, presented by Daniel J. McCarty, M.D., Associate Professor of Medicine, and Head of Rheumatology Section, Hahnemann Medical College and Hospital. (13 minutes). **0700411**

IMMUNOLOGY: FRONTIERS OF THERAPY, with Robert A. Good, M.D., Ph.D., Professor and Head, Department of Pathology, University of Minnesota School of Medicine, Minneapolis. Research meets clinical medicine as Dr. Good explains a "new kind of cellular engineering." The application of this new therapy is demonstrated in patients, and, in a look at the future, Good speaks of giving cancer patients "an improved immunity system" to help the "host look at cancer as the foreigner it really is."
(22 minutes) (in color) **0916519**

IMMUNOLOGY: THE FUTURE, with Robert A. Good, M.D., Ph.D., Professor and Head, Department of Pathology, University of Minnesota School of Medicine, Minneapolis.

"The next few years are really bright for immunobiology," says Dr. Good. He and his colleagues review the information already in hand which will eventually open the doors to the transplantation era and facilitate treatment and prevention of cancer. The program's emphasis is on coming immunologic tools for the clinician.
(19 minutes) (in color) **0916621**

IMMUNOLOGY: THE NEW PATHOLOGY, with Robert Good, M.D., Professor and Head, Department of Pathology, University of Minnesota School of Medicine.

In a wide-ranging discussion of recent discoveries in immunology, Dr. Good describes the function of T-cell and beta-cell systems and their meaning for clinicians.
(19 minutes) (in color) **0916418**

SPHINGOLIPIDOSIS: GENETICS

The increasing incidence of sphingolipid disease, such as Tay-Sachs, Gaucher's, and Niemann-Pick, is examined genetically by Stanley M. Aronson, M.D., Professor of Pathology, State University of New York Downstate Medical Center, and Attending Neuropathologist, Isaac Albert Research Institute, Jewish Chronic Disease Hospital, Brooklyn. (15 minutes). **1904914**

SPHINGOLIPIDOSIS — PART I — BIOCHEMICAL ASPECTS. The chemical compositions of gangliosides, sphingomyelins, sulfatides, glycolipids, and cerebrosides, as they are found in the various sphingolipid diseases, are analyzed by Abraham Saifer, Ph.D., Chief of the Biochemistry Department, Isaac Albert Research Institute of the Jewish Chronic Disease Hospital, Brooklyn.
(21 minutes). **1905015**

SPHINGOLIPIDOSIS — PART II — PATHOLOGY. Several pathologic manifestations — such as amaurotic family idiocy (Tay-Sachs disease), hepatosplenomegaly (Niemann-Pick disease), and others — grouped under the general category of sphingolipidosis, are examined and defined by Bruno W. Volk, M.D., Director of the Isaac Albert Research Institute, of the Jewish Chronic Disease Hospital, and Clinical Professor of Pathology, State University of New York Downstate Medical Center, Brooklyn. (21 minutes). **1905016**

SPHINGOLIPIDOSIS — PART III — CLINICAL ASPECTS. The specific physiologic manifestations of the Tay-Sachs and Niemann-Pick diseases and amaurotic idiocy—such as cherry red macula, clonus, severe contractions, the "frog" position of the legs, and lack of macrocephaly — are demonstrated with young patients by Larry Schneck, M.D., of the Albert Isaac Research Institute of the Jewish Chronic Disease Hospital, Downstate Medical Center, Brooklyn, New York. (13 minutes). **1905217**

STEROIDS, HORMONES AND INFLAMMATORY DISEASE, with Gerald Weissman, M.D., Associate Professor of Medicine, New York University Medical Center. Steroids, hormones, and chloroquin have been shown to counteract inflammation and tissue injury by virtue of their stabilization of lysosomes. (14 minutes). **1900318**

THE MECHANISM OF ACTION OF POLYENE ANTIBIOTICS, with Gerald Weissman, M.D., Associate Professor of Medicine, New York University Medical Center. Amphotericin B disrupts mammalian fungal membranes. (17 minutes). **1302816**

THE MEMBRANES OF CELLS AND ORGANELLES, with Gerald Weissman, M.D., Associate Professor of Medicine, New York University Medical Center. (20 minutes). **1302730**

CARDIOLOGY & CARDIOVASCULAR DISEASE

A COMPREHENSIVE INTENSIVE CARE UNIT IN A GENERAL HOSPITAL — PART I — "History and Organization" — with Albert H. Douglas, M. D., Director, Department of Medicine, and Erwin Lear, M.D., Director, Department of Anesthesiology, Queens Hospital Center; Edward Meilman, M.D., Physician-in-Chief of Medicine, and Charles Trey, M.D., Attending Physician, The Long Island Jewish Hospital. (17 minutes). **0301931**

A COMPREHENSIVE INTENSIVE CARE UNIT IN A GENERAL HOSPITAL — PART II — "Clinical Applications" — with Albert H. Douglas, M.D., Director, Department of Medicine, and Erwin Lear, M.D., Director, Department of Anesthesiology, Queens Hospital Center; Edward Meilman, M.D., Physician-in-Chief of Medicine, and Charles Trey, M.D., Attending Physician, The Long Island Jewish Hospital. (16 minutes). **0302032**

CARDIAC TRANSPLANTATION. With Denton A. Cooley, M.D., Professor of Surgery; Robert D. Bloodwell, M.D., Assistant Professor of Surgery; Grady L. Hallman, M.D., Associate Professor of Surgery; and Robert D. Leachman. M.D., Professor of Cardiology, Baylor University College of Medicine.

Cardiac transplantation deserves now to be accepted in the vast armamentarium of treatment of the coronary patient, according to Dr. Cooley. Noting this, he points out that the problems surrounding rejection of the donor heart are being met with increasingly good results. Thus, he adds, the major drawback to increased use of cardiac transplantation is the availability of donor hearts.

The implications of this — the need for a new definition of death as well as possible procedures for storing donor hearts — are discussed by Dr. Cooley and his colleagues.

The film for this presentation was taken during the first transplantation. While the procedure has not changed radically in transplantations that followed this one, the attitudes of the physicians have. No longer is the transplant patient treated like a "cardiac cripple," they contend. Rather, where possible, every attempt is made to handle him as any other postoperative case with ambulation after 48 hours the goal. (30 minutes) (in color) **2007014**

CEREBRO-VASCULAR INSUFFICIENCY — DIAGNOSIS AND MANAGEMENT. The diagnostic workup of the patient with evidence of cerebrovascular insufficiency as well as medical management of this individual is demonstrated in PART I of this two-part series. The second program concerns itself with indications for surgical referral for transient ischemic attacks, with a discussion of risks and results. Charles A. Kane, M.D., Chief, Division of Neurology, The Permanente Medical Group, Hayward, California, emphasizes diagnostic considerations and medical management, while Edwin J. Wylie, M.D., Professor of Surgery, University of California Medical Center, San Francisco, focuses on surgical problems. The exchange between the authorities provides an opportunity to examine the multifaceted sides of this common clinical problem. (PART I: 18 minutes). **0307805**
(PART II: 18 minutes). **0307806**

CLINICAL PATHWAYS OF CARDIOMYOPATHY, with John F. Stapleton, M.D., Professor of Medicine, Georgetown University Medical Center, Washington, D. C. Drawing on 17 years of experience with the Cardiomyopathy Study Unit, a distinguished cardiologist presents steps for earlier recognition of this condition in your patients. This program was produced with the cooperation of Georgetown University School of Medicine in Washington, D. C.
(16 minutes) (in color) **0323385**

CLUES TO CONGESTIVE HEART FAILURE, with Herbert J. Levine, M.D., cardiologist, Tufts-New England Medical Center, Boston, Massachusetts.
Fully developed congestive heart failure is easily recognized. Precursor signs and symptoms are not as easily diagnosed. Dr. Levine describes his office procedure for detecting congestive heart failure at its earliest stages. Careful history taking is essential to Dr. Levine's approach. Several patients are shown to demonstrate his technique. (16 minutes) (in color) **0311727**

COMMON PITFALLS IN ECG RECORDING, with Nanette K. Wenger, M.D., Professor of Medicine (Cardiology), Emory University School of Medicine, and Director of the Cardiac Clinics, Grady Memorial Hospital, Atlanta. Electrical interference, machine malfunction, and improper patient preparation and ECG recording technique can give you tracings which are impossible to interpret properly. Dr. Wenger shows how to help your office assistant avoid these pitfalls.
(11 minutes) (in color) **0317574**

CONGESTIVE HEART FAILURE — PATHOPHYSIOLOGY AND TREATMENT, with Albert N. Brest, M.D., Head, Section of Vascular Disease and Renology, Hahnemann Medical College and Hospital. (15 minutes). **0302545**

CONGESTIVE HEART FAILURE: PRECISION DIAGNOSIS, with James E. Doherty, M.D., Professor of Medicine and Pharmacology, University of Arkansas College of Medicine and Director, Division of Cardiology, V.A.-University Medical Center Hospitals, Little Rock.
Improve your skills as a diagnostician. Learn how to focus on the often-missed, early, subtle clues to congestive heart disease.
(11 minutes) (in color) **0323687**

CONGESTIVE HEART FAILURE: SUCCESSFUL MANAGEMENT, with James E. Doherty, M.D., Professor of Medicine and Pharmacology, University of Arkansas College of Medicine, and Director, Division of Cardiology, V.A.-University Medical Center Hospitals, Little Rock.
Digitalis, Diet, Diuretics, Rest and Vasodilators. When and how to prescribe most effectively.
(12 minutes) (in color) **0323788**

CORONARY ARTERY DISEASE IN MAN — PART I — PRACTICAL CONSIDERATIONS AT THE BEDSIDE. Robert J. Boucek, M.D., Professor of Medicine, University of Miami School of Medicine, describes how the clinician can improve his diagnosis of the site and degree of coronary artery disease. (22 minutes). **0305149**

CORONARY ARTERY DISEASE IN MAN — PART II — STRUCTURE AND FUNCTION OF THE CORONARY ARTERIES IN THE LOCALIZATION OF ATHEROSCLEROSIS. How the structure of the coronary artery wall, the arterial musculature, and the action of the heart relate to the specific, predetermined sites of coronary artery disease are described by Robert J. Boucek, M.D., Professor of Medicine, University of Miami School of Medicine. (19 minutes). **0305250**

CURABLE HYPERTENSION, with Ralph E. Peterson, M.D., Professor of Medicine and Director, Division of Endocrinology, New York Hospital-Cornell University Medical Center, New York City. Forty million adult Americans have blood pressures higher than 150/90; in half of them, hypertension is secondary to another disease process. Among this latter group, surgery to remove the underlying cause is effective in five per cent or one million cases. Dr. Peterson shows how to determine if a hypertensive patient is among the curable million. (16 minutes) (in color) **0315270**

THE DIAGNOSTIC CHALLENGE OF CHEST PAIN, with moderator Alfred Soffer, M.D., Professor of Cardiology at the University of Health Sciences—Chicago Medical School, Chicago, and Editor-in-Chief of *Chest*, the Journal of the American College of Chest Physicians. Participants are: R. D. Henderson, M.B., F.R.C.S. (C)., Staff Surgeon, Toronto General Hospital and Assistant Professor of the Department of Surgery at the University of Toronto, Canada; Barry William Levine, M.D., Assistant Professor of Medicine, Harvard Medical School, and Chief, Outpatient Pulmonary Services, Massachusetts General Hospital, Boston; Linda D. Lewis, M.D., Assistant Professor of Neurology, College of Physicians and Surgeons of Columbia University, and Chief of the Neurology Clinic, Neurological Institute of Columbia Presbyterian Medical Center, New York City; Morton E. Tavel, M.D., Associate Professor of Medicine at the Indiana University School of Medicine, Indianapolis.
Pinpointing the cause of your patient's chest pain poses an urgent diagnostic challenge. Join Dr. Soffer and four specialists on these clinical grand rounds as they reach a diagnosis on five patients. (32 minutes) (in color) **0423362**

DIET FOR THE CARDIAC PATIENT, with W. Jann Brown, M.D., Professor and Acting Chairman, Department of Pathology, UCLA, School of Medicine.

A major problem with most low-sodium, low-calory diets is that they are monotonous and tasteless; patients are prone to "cheat" on them. Dr. Brown, from his own kitchen, shows that the cardiac patient can enjoy near-gourmet menus. (16 minutes) (in color) **0411913**

DIGITALIS: FRIEND OR FOE? with James E. Doherty, M.D., Professor of Medicine and Director of Cardiology, University of Arkansas School of Medicine and The Little Rock Veterans Administration Hospital. Dr. Doherty points out the signs and symptoms of digitalis toxicity and provides guidelines for adjusting dosage to provide maximum benefit without adverse reactions. (This program was part of the Drug Spotlight Program of the American Society for Clinical Pharmacology and Therapeutics.)

(13 minutes) (in color) **0420152**

DOES TYPE A PERSONALITY AFFECT YOUR HEART?, with Ray H. Rosenman, M.D., Associate Director of the Harold Brunn Institute and Associate Chief of the Department of Medicine, Mount Zion Hospital and Medical Center, San Francisco; William B. Kannel, M.D., Medical Director of the Framingham Heart Study in Massachusetts; and Campbell Moses, M.D., Vice-President of Medicus Communications in New York City. Is stressful behavior a coronary risk factor? Doctor Moses moderates a lively discussion.

(18 minutes) (in color) **0421858**

ECHOCARDIOGRAPHY: SOUNDING THE HEART, with Fred Winsberg, M.D., Director of the Division of Diagnostic Ultrasound, Montreal General Hospital, and Associate Professor of Radiology, McGill University, Montreal, Quebec. When and how to use a non-invasive diagnostic procedure for your cardiac patients.

(14 minutes) (in color) **0521629**

EMERGENCY CARDIAC CARE, with William J. Grace, M.D., Department of Medicine, New York University School of Medicine, and Joseph G. Hayes, M.D., Cornell University Medical College, New York City; Costas T. Lambrew, M.D., Department of Medicine, Meadowbrook Hospital, East Meadow, New York; and Joseph Schluger, M.D., Department of Medicine, Long Island College Hospital, Brooklyn, New York. A personal plan of action is proposed to help the physician deal with patients showing early signs of myocardial infarction. Optional emergency cardiac care systems adaptable to different community needs are described, and recently modified CPR techniques are demonstrated. A Television Clinic of the American College of Physicians, 1974. Please inquire for special rental information.

(60 minutes) (in color) **ACP 2867482**

EMERGENCY TRANSVENOUS CARDIAC PACING, with Doris J. W. Escher, M.D., Attending Physician in the Division of Medicine and Physician-in-Charge of Cardiac Catheterization, Montefiore Hospital and Medical Center, Bronx, New York. The technique for emergency transvenous cardiac pacemaker insertion at the patient's bedside is shown. Intracardiac electrogram patterns are correlated with catheter position, from high right atrium to the target position at the apex of the ventricle. (19 minutes) (in color) **0520426**

EVALUATING CARDIAC CATHETERIZATION IN AORTIC VALVE DISEASE, with Adolph Hutter, M.D., Associate Director of the Coronary Care Unit and Assistant in Medicine at Massachusetts General Hospital, and Instructor in Medicine at Harvard Medical School; and Peter Block, M.D., Assistant in Medicine at Massachusetts General, and Instructor in Medicine at Harvard, Boston. Two members of the Harvard faculty demonstrate when cardiac catheterization should be performed. They show the complications associated with the operation. And, they make the distinction in the problems of catheterizing children and adults. (16 minutes). **0512916**

EVALUATING CARDIAC CATHETERIZATION IN MITRAL VALVE DISEASE, with Adolph Hutter, M.D., Associate Director of the Coronary Care Unit and Assistant in Medicine at Massachusetts General Hospital, and Instructor in Medicine at Harvard Medical School; and Peter Block, M.D., Assistant in Medicine at Massachusetts General, and Instructor in Medicine at Harvard, Boston. Two physicians on the Harvard faculty present their second NCME program on cardiac catheterization. In this telecast, they describe the indications and advantages of a generally benign right heart catheterization to determine the severity of a mitral valve lesion.

(15 minutes) (in color) **0513017**

THE EXERCISE TEST, with Elliott M. Stein, M.D., Assistant Professor of Medicine, the New Jersey College of Medicine and Dentistry; Chief of the Exercise Laboratory and Cardiac Rehabilitation, Saint Michael's Medical Center, Newark.

A fresh and comprehensive look at the real value of the dynamic exercise test. Indications, contraindications, and how a multifactorial analysis of your patient's response can help in both diagnosis and treatment of cardiovascular disease.

(16 minutes) (in color) **0523530**

FINGERPRINTING MYOCARDIAL INFARCTION SERUM ENZYMES. How serum enzyme analysis is used to increase diagnostic accuracy and what prognostic data are furnished are demonstrated by John S. Ladue, M.D., Ph.D., Assistant Professor of Clinical Medicine, Cornell University School of Medicine, New York.

(15 minutes). **0608104**

FRAMINGHAM'S CORONARY CANDIDATE: IDENTIFICATION AND PROPHYLAXIS

William B. Kannel, M.D., Thomas R. Dawber, M.D., and William P. Castelli, M.D., from the Heart Disease Epidemiology Study of the National Heart Institute, National Institutes of Health, Framingham, Massachusetts, demonstrate, with patients, the objectives and the apparent successes of the Study.

(21 minutes) (In color) **0609711**

GAS ENDARTERECTOMY — PART I — PERIPHERAL ARTERIES. The injection of CO_2 into the media of an occluded artery and the subsequent removal of the media, intima and arteriosclerotic material are shown by one of the originators of the procedure, Philip N. Sawyer, M.D., Professor of Surgery, and Head of the Vascular Surgical Service, Downstate Medical Center, State University of New York, Brooklyn.

(14 minutes). **0704701**

GAS ENDARTERECTOMY — PART II — CORONARY ARTERIES. The injection of CO_2 into a grossly occluded coronary artery separates the media, intima, and atheromatous substance from the adventitia, a procedure which greatly simplifies endarterectomy, in the view of Philip N. Sawyer, M.D., Professor of Surgery, and Head of the Vascular Surgical Service, Downstate Medical Center, State University of New York, Brooklyn. (18 minutes). **0704802**

HEMODYNAMICS — AN INSTRUCTIONAL DEVICE. A mechanical model of the cardiovascular system is demonstrated and discussed by Simon Rodbard, M.D., Chief of Cardiology, City of Hope Medical Center, Duarte, California. Designed by Dr. Rodbard as a teaching aid, the machine simulates normal blood flow, and, by opening or closing the appropriate valves, demonstrates cardiovascular dysfunction, as well.

(15 minutes). **0805303**

HOW I DO SUBCLAVIAN VENIPUNCTURE, with Josef E. Fischer, M.D., Assistant Professor of Surgery, Harvard Medical School, and Instructor in Surgery at Massachusetts General Hospital, Boston. Dr. Fischer demonstrates catheter placement in the subclavian vein and outlines indications and contraindications for the procedure.

(17 minutes) (In color) **0817830**

HOW I TREAT VARICOSE VEINS, with Robert A. Nabatoff, M.D., Chief of the Vascular Clinic, Mount Sinai Hospital and Medical Center, New York City. Injection or surgery? Dr. Nabatoff's outpatient stripping procedure, which he demonstrates, answers a number of the practical objections to surgical therapy.

(20 minutes) (In color) **0818231**

IMPLANTED PACEMAKERS: LONG-TERM FOLLOWUP, with Doris J. W. Escher, M.D., Attending Physician, Department of Medicine, Cardiology Division; and Seymour Furman, M.D., Associate Attending Surgeon, Department of Surgery, Cardiothoracic Surgery Division, Montefiore Hospital and Medical Center, Bronx, New York. How to avoid unnecessary implanted pacemaker failure. Specific tests to evaluate pacemaker function are demonstrated and the results interpreted.

(21 minutes) (In color) **0920528**

INFLUENCE OF THE EMOTIONS ON THE OUTCOME OF CARDIAC SURGERY: DIAGNOSIS AND DECISION with Janet A. Kennedy, M.D., Assistant Professor of Psychiatry; and Hyman Bakst, M.D., Assistant Clinical Professor of Medicine; both of the Albert Einstein College of Medicine in New York City. Eight distinct emotional stages have been observed in 148 cardiac surgery patients in a nine-year study. The anxieties and reactions of patients are shown for each of these stages. (20 minutes). **0910506**

INFLUENCE OF THE EMOTIONS ON THE OUTCOME OF CARDIAC SURGERY: PSYCHOLOGICAL CATEGORIES, with Dr. Kennedy and Dr. Bakst in a separate program from the one above. In this one they classify cardiac surgery patients into six groups with their distinguishing defense characteristics. Understanding these groupings during an interview with a patient can aid in predicting how the patient will be affected by surgery, whether he will accept surgery, survive it, and avail himself of the benefits of restored cardiac function. (24 minutes). **0910507**

INNOCENT HEART MURMURS IN CHILDREN, with Bernard L. Segal, M.D., Clinical Professor of Medicine, Hahnemann Medical College and Hospital, Philadelphia. Perhaps a third of all children have heart murmurs, but they are often innocent. Cardiologist Segal demonstrates an examination to differentiate innocent from organic murmurs and uses audio recordings to point out the characteristics of several common murmurs.

(13 minutes) (In color) **0917624**

INTERPRETING EKGs: A MODEL FOR NORMAL SEQUENCE OF ACTIVATION AND INTRA-VENTRICULAR CONDUCTION DEFECTS, with Peter Block, M.D., Assistant in Medicine at Massachusetts General Hospital, and Instructor in Medicine at Harvard Medical School, Boston, Massachusetts. Physicians in general practice will be able to update their knowledge of reading EKGs in this first of two telecasts on the subject. In this program, Dr. Block shows how references to normal heart function can be used to solve problems in diagnosing right and left bundle-branch block.

(18 minutes) (In color) **0913209**

INTERPRETING EKGs: CURRENT CONCEPTS OF HEART BLOCK, with Adolph M. Hutter, Jr., M.D., Instructor in Medicine, Harvard Medical School, Boston. Some heart block patients are emergencies, some are not. Some should be treated, some should not. Dr. Hutter presents a schematic review of heart block . . . and what to do — about it.
(18 minutes) (in color) **0914917**

INTERPRETING EKGs: INFARCT PATTERNS IN PRESENCE OF BUNDLE-BRANCH BLOCK, with Adolph Hutter, Jr., M.D., Associate Director of the Coronary Care Unit and Assistant in Medicine at Massachusetts General Hospital, and Instructor in Medicine at Harvard Medical School. The electrocardiographic diagnosis of myocardial infarction is difficult with the presence of bundle-branch block. Dr. Hutter will show the general physician how to recognize right and left bundle-branch block on EKGs and how to read myocardial infarctions in the presence of conduction defects. (18 minutes) (in color) **0913315**

INTERPRETING EKGs: RECOGNITION AND TREATMENT OF SUPRA-VENTRICULAR ARRHYTHMIAS — ATRIAL FIBRILLATION, ATRIAL FLUTTER AND PAROXYSMAL ATRIAL TACHYCARDIA, with Adolph M. Hutter, Jr., M.D., Associate Director of the Coronary Care Unit and Assistant in Medicine at Massachusetts General Hospital and Assistant Professor of Medicine, Harvard Medical School, Boston. With the aid of diagrams and sample EKGs, Dr. Hutter explains the mechanisms involved in supra-ventricular arrhythmias and the therapeutic maneuvers to counteract them. Emphasis is given to differentiating the arrhythmias; to the use of digitalis, its effects and signs of toxicity; and to the appropriate use of cardioversion.
(18 minutes) (in color) **0916520**

INTERPRETING EKGs: RECOGNITION AND TREATMENT OF SUPRA-VENTRICULAR ARRHYTHMIAS—SINUS BRADYCARDIA AND SICK SINUS SYNDROME, with Peter Block, M.D., Assistant in Medicine at Massachusetts General Hospital, and Assistant Professor of Medicine, Harvard Medical School, Boston. Tips for the differentiation and management of sinus bradycardia, wandering atrial pacemaker, multifocal atrial tachycardia, and sick sinus syndrome (sinus bradycardia alternating with rapid tachyarrhythmias). (13 minutes) (in color) **0916722**

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INTERPRETING EKGs: VENTRICULAR ARRYTHMIAS, with Peter Block, M.D., Assistant in Medicine, Massachusetts General Hospital, and instructor in Medicine, Harvard Medical School, Boston. Ventricular premature beats (VPB) and their complications are now known to produce alterations in hemodynamics . . . cardiac deterioration . . . and even sudden death. Dr. Block graphically demonstrates the differential diagnosis of VPBs, for the general physician.
(20 minutes) (in color) **0914816**

MANAGEMENT OF STROKE — PART I. Rudolph Kaelbling, M.D., Associate Professor of Psychiatry, Ohio State University College of Medicine, examines the common types of stroke and their effects on consciousness, mental acuity, speech, and physical coordination, as well as the impact of stroke on the patient's family. Rehabilitation is discussed also, and a speech therapist is shown working with patients.
(14 minutes). **1305506**

MANAGEMENT OF STROKE — PART II. The physical rehabilitation of the stroke patient — including rehabilitative appliances, exercise, and physiotherapy — is discussed and demonstrated by Ernest W. Johnson, M.D., Chairman, Department of Physical Medicine, Ohio State University College of Medicine.
(17 minutes). **1305607**

MEDICAL ASPECTS OF CONSTANT CORONARY CARE UNITS IN A GENERAL HOSPITAL
The problem of the rhythm death following myocardial infarction has provided the stimulus for development of cardiac resuscitation and cardiac monitoring units. The required equipment and techniques are discussed and demonstrated by Richard Watts, M.D., Head, Cardiovascular Section, Department of Medicine, Fairview General Hospital, Cleveland, Ohio.
(14 minutes). **1303923**

MILD-TO-SEVERE HYPERTENSION: TIPS FOR TREATMENT, with George N. Aagaard, M.D., Professor of Medicine and Head of the Division of Clinical Pharmacology, University of Washington School of Medicine, Seattle. As part of the national "Drug Spotlight Program", Dr. Aagaard presents a simple approach to treating patients with hypertension. He outlines the basic nonpharmacologic approach, the way in which to use oral diuretics, adrenergic inhibitors and smooth muscle dilators, and his method of handling refractory hypertension.
(14 minutes) (in color) **1317749**

THE NATIONAL HYPERTENSION TEST, with Harriet P. Dustan, M.D., Vice-Chairman, Research Division, The Cleveland Clinic; Edward D. Frohlich, M.D., George Lynn Cross, Research Professor and Director, Division of Hypertension, University of Oklahoma Health Sciences Center, Oklahoma City; Curtis G. Hames, M.D., Chairman, Evans County Health Department, Claxton, Georgia; Morton H. Maxwell, M.D., Clinical Professor of Medicine, UCLA, and Director, Hypertension Division, Cedars-Sinai Medical Center, Los Angeles; and Gerald E. Thomson, M.D., Professor of Medicine, Columbia University, and Director of Medicine, Harlem Hospital, New York City. How skilled are you in the diagnosis and treatment of various types of hypertension? How much do you know about the pathophysiology of this disease? Find out by taking this self-assessment test, produced in cooperation with the American Heart Association. The test considers hypertension as a hemodynamic abnormality and as a disease. Panelists present 8 patients with hypertension and ask questions to challenge your skills. You will learn the answer to each question and hear a discussion of the "right and wrong" alternatives of patient management during the test. To keep track of your score, an answer sheet is provided with the test. (68 minutes) (in color) **1422536**

PAUL D. WHITE: CARDIOLOGY IN MY TIME No physician is better equipped to describe the development of cardiology as a specialty than one of its pioneers. The telecast is filled with anecdotes from Dr. White's many associations through his many years of medical practice. (Courtesy, Eli Lilly and Company)
(28 minutes) (In color) **1608602**

PEDIATRIC CARDIOLOGY — PART I — CATHETERIZATION IN INFANTS. Donald R. Sperling, M.D., Assistant Professor of Pediatrics, California College of Medicine, University of California, explains the indications and technique for diagnosing congenital heart disease by means of the cardiac catheter. (14 minutes). **1605003**

PEDIATRIC CARDIOLOGY — PART II — DIVISION OF PATENT DUCTUS ARTERIOSUS. Joseph J. Verska, M.D., Associate Clinical Professor of Surgery, University of California, the California College of Medicine, and Director of Cardiac Surgery, White Memorial Medical Center, Los Angeles, operates to correct this congenital cardiac defect. The patent ductus in this infant patient was diagnosed (in PART I) by Donald R. Sperling, M.D., also of the California College of Medicine, using cardiac catheterization.
(19 minutes). **1605104**

PRE-HOSPITAL CORONARY CARE — A MODEL FOR YOUR COMMUNITY, with Eugene Nagel, M.D., Associate Professor, Department of Anesthesiology, University of Miami School of Medicine, and Clinical Director, Department of Anesthesiology, Jackson Memorial Hospital, Miami, Florida. How quickly can your community get optimum coronary care facilities to a patient in cardiac arrest or ventricular fibrillation? Dr. Nagel shows how the Miami Emergency Rescue Service is set up, demonstrates how quickly it works in a simulated rescue, and shows what is needed to implement a complete mobile emergency health care system.
(21 minutes) (in color) **1615438**

PHYSICAL MEASURES FOR ARTHRITIS, STROKE AND CARDIOVASCULAR REHABILITATION, with John A. Faulkner, Ph.D., Department of Physiology and Robert L. Joynt, M.D., George H. Koepke, M.D., Barry Miller, M.D., and Edwin M. Smith, M.D., all of the Department of Physical Medicine and Rehabilitation, University of Michigan Medical School, Ann Arbor, Mich. The panel discusses and demonstrates physical measures for treating arthritis, techniques to prevent contractures following stroke and methods to use in preserving maximal function of unaffected extremities, and also presents the basics for prescribing exercise for the patient with coronary heart disease. A Television Clinic of the American College of Physicians, 1973. Please inquire for special rental information.
(60 minutes) (in color) **ACP 2867370**

PRINCIPLES OF CORONARY CARE. Early surveillance and continuous monitoring of patients with acute myocardial infarction greatly enhance resuscitation probability in the event of arrhythmia or fibrillation. Stephen Wittenberg, M.D., Instructor in Medicine, and Roger Hand, M.D., Chief Medical Resident, New York University Medical Center, demonstrate monitoring and resuscitation procedures in the Intensive Care Unit of University Hospital.
(11 minutes). **1605920**

PROBLEMS AND PITFALLS IN PSYCHOSOMATIC MEDICINE: HYPERTENSION, with Roy R. Grinker, M.D., Associate Professor of Psychiatry and Neurology, and F. Theodore Reid, M.D., Associate Professor of Psychiatry and Neurology, both from the Michael Reese Hospital in Chicago, Illinois.

Doctors Grinker and Reid take up the problem of a young woman suffering from hypertension. Dr. Grinker analyzes the cause of the hypertension in interviews with the woman.
(15 minutes) (in color) **1612621**

PULMONARY EMBOLISM: A RATIONAL APPROACH TO MANAGEMENT, with William Hall, M.D., Director of the Pulmonary Function Unit at Strong Memorial Hospital, and Assistant Professor of Medicine, University of Rochester School of Medicine, Rochester, New York. The mortality rate for untreated pulmonary embolism patients is between 25 and 50 percent. Doctor Hall demonstrates that such gloomy results can be avoided through prompt and effective management, which includes anticoagulant therapy and the treatment of hypoxia. (This program is part of the Drug Spotlight Program of the American Society for Clinical Pharmacology and Therapeutics.) (17 minutes) (in color) **1619744**

RADIOISOTOPE ARTERIOGRAPHY
Richard Janeway, M.D., and C. Douglas Maynard, M.D., of the Departments of Neurology and Radiology at the Bowman Gray School of Medicine present a new aid to cerebrovascular disease.
(20 minutes). **1809503**

REPAIR OF A RUPTURED VENTRICULAR SEPTUM
A rupture of the ventricular septum is a comparatively uncommon phenomenon, most often occurring in patients who have suffered an occlusion, particularly of the left coronary artery involving the septal branch. The technique for repairing the rupture is demonstrated by Adrian Kantrowitz, M.D., Director of Surgery, Maimonides Medical Center, Brooklyn, and Professor of Surgery, State University of New York, College of Medicine. (16 minutes). **1803810**

RESECTING AN ANEURYSM OF THE ASCENDING AORTA
The demonstration illustrates the standard techniques for resection of an ascending aorta using pump oxygenator by-pass, continuous coronary perfusion, and replacement with dacron prosthesis — with Adrian Kantrowitz, M.D., Director of Surgery, Maimonides Medical Center and Professor of Surgery, State University of New York, College of Medicine. (16 minutes). **1803313**

SELECTION OF PATIENTS AND DRUGS FOR TREATMENT OF HYPERTENSION, with Albert N. Brest, M.D., Associate Professor of Medicine and Head, Section of Vascular Disease and Renology, Hahnemann Medical College and Hospital. (17 minutes). **1902801**

STASIS ULCERS OF THE ANKLE, with Robert A. Nabatoff, M.D., Chief, Vascular Surgery, Department of Obstetrics and Gynecology, at Mount Sinai Medical Center, New York City. How to treat these common, yet often puzzling, symptoms before surgery for the underlying vascular insufficiency. (14 minutes) (in color) **1918430**

SURGICAL TREATMENT OF HEART BLOCK
Robert Schramel, M.D., Associate Professor of Surgery, Tulane University School of Medicine, discusses and demonstrates surgical use of electronic pacemakers. (15 minutes). **1902823**

THE EXERCISE PRESCRIPTION, with Nanette K. Wenger, M.D., Professor of Medicine, Division of Cardiology, Emory University School of Medicine, Atlanta; and William L. Haskell, Ph.D., Co-Director of the Stanford University Cardiac Rehabilitation Program, Palo Alto, Calif. You can prescribe exercise as precisely as you do drugs. Here's how the results of an exercise stress test can guide you. (22 minutes) (in color) **0519324**

THE HEART. One in a series of Anatomical Relationships from Duke University Medical Center. The surface anatomy of the heart, presented by John L. Dobson, M.D., Department of Anatomy.
(19 minutes). **0800401**

THE MASTER TWO-STEP TEST — PART I. Arthur M. Master, M.D., Consulting Cardiologist, Mount Sinai Hospital, demonstrates the ultilization of the two-step test. (12 minutes). **1302312**

THE MASTER TWO-STEP TEST — PART II. Arthur M. Master, M.D., Consulting Cardiologist, Mount Sinai Hospital, presents patient data on negative and positive two-step tests.
(23 minutes). **1302413**

THE MASTER TWO-STEP: A REAPPRAISAL
Arthur M. Master, M.D., cardiologist and Emeritus Professor of Medicine at Mt. Sinai Hospital and Medical Center in New York, discusses the three major uses of the stress imposed by the double two-step test in conjunction with ECG tracings. (16 minutes). **1308914**

THE PRESENT STATUS OF CORONARY ARTERY BYPASS SURGERY, with Rolf M. Gunnar, M.D., Department of Medicine, Loyola University of Chicago, Stritch School of Medicine, Maywood, Ill. and Veterans Administration Hospital, Hines, Ill.; John M. Moran, M.D., Department of Surgery, Rimgaudas Nemickas, M.D., Deparment of Medicine, Roque Pifarré, M.D., Department of Surgery, and Patrick Scanlon, M.D., Department of Medicine, all of Loyola University of Chicago, Stritch School of Medicine, Maywood, Ill. The indications, contraindications, and ways to assess the prognosis of coronary artery surgery, including vein grafts, endarterectomy, and internal mammary artery grafts, are illustrated and discussed. In addition, postoperative complications are put in perspective and ways to evaluation postoperative results are discussed. A Television Clinic of the American College of Physicians, 1973. Please inquire for special rental information.
(60 minutes) (in color) **ACP 2837367**

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TIME BORROWERS IN SHOCK, with Leon I. Goldberg, Ph.D., M.D., Professor of Medicine and Pharmacology, and Director of Clinical Pharmacology, Emory University School of Medicine in Atlanta, Georgia. When the physician needs to "borrow time" while treating the underlying causes of shock, a cautious use of sympathomimetic amines is often a worthwhile temporary solution. (14 minutes) (in color) **2020737**

TRANSIENT ISCHEMIC ATTACK: PART I — THE HISTORY
(13 minutes) (in color) **2018829**

TRANSIENT ISCHEMIC ATTACK: PART II — THE PHYSICAL, with Clark H. Millikan, M.D., Senior Consultant and Professor of Neurology, The Mayo Clinic, Rochester, Minn. During this two-part program, you'll take a close look at how to make a diagnosis of TIA . . . an important challenge, as three of five major strokes may be preventable through proper diagnosis of TIA's and their subsequent treatment.
(21 minutes) (in color) **2018830**

TREATMENT OF CORONARY HEART DISEASE— ISCHEMIC HEART DISEASE. How can the physician in general practice determine if medical management of ischemic heart disease is no longer necessary and surgery is indicated? This program on coronary insufficiency is concerned with differential diagnosis of the patient afflicted for the first time with coronary heart disease. Surgical procedures for chronic occlusive disease are shown by Denton A. Cooley, M.D., Professor of Surgery; Grady L. Hallman, M.D., Associate Professor of Surgery; Robert D. Bloodwell, M.D., Assistant Professor of Surgery; and Robert D. Leachman, M.D., Professor of Cardiology, Baylor University College of Medicine, Houston, Texas.
(19 minutes) (in color) **2007215**

TREATMENT OF CORONARY HEART DISEASE — POSTINFARCTION COMPLICATIONS. Aortic aneurysm, ventricular septal defect, heart block . . . these complications to recovery of coronary patients are treatable, but require prompt evaluation and advanced surgical techniques. Denton A. Cooley, M.D., Professor of Surgery; Grady L. Hallman, M.D., Associate Professor of Surgery; Robert D. Bloodwell, M.D., Assistant Professor of Surgery; and Robert D. Leachman, M.D., Professor of Cardiology, Baylor University College of Medicine, delineate the management of these conditions. (21 minutes) (in color) **2007116**

TREATMENT OF SEVERE CORONARY ARTERY DISEASE: A TOTAL SURGICAL APPROACH
W. Dudley Johnson, M.D., Department of Cardiovascular and Thoracic Surgery and the Division of Surgery, Marquette School of Medicine, Milwaukee, Wisconsin, discusses and vividly describes the pedicle system of indirect revascularization, the vein by-pass technique in direct coronary surgery, and resection of aneurisms.
(19 minutes) (in color) **2009318**

U. C. GRAND ROUNDS:
THE HAND AND THE HEART
Mark E. Silverman, M.D., with the David Grant Hospital-Travis Air Force Base, California, presents visual evidence of the hand as a source of diagnosing cardiovascular disorders.
(20 minutes). **2108901**

UNCOVERING WHAT'S RIGHT WITH THE STROKE PATIENT, with Paul J. Corcoran, M.D., Associate Professor of Rehabilitation Medicine, Boston University Medical Center, Boston. Dr. Corcoran shows the non-rehabilitation specialist how to examine and evaluate the stroke patient during his first post-CVA days. Unlike most exams, attention is given to ability rather than disability. (22 minutes) (in color) **2117007**

VENOUS THROMBOSIS AND PULMONARY EMBOLISM PREVENTION, RECOGNITION, AND TREATMENT, with Harold A. Baltaxe, M.D., Department of Radiology; William Gay, M.D., Department of Surgery; James W. Hurley, M.D., and Susan A. Kline, M.D., Department of Medicine; all of Cornell University Medical College, New York City. Among topics considered: key signs and symptoms of venous thrombosis; preventive measures, including mini-heparinization; screening tests for the presence of emboli; and therapy, both surgical and medical, with special attention given to fibrinolytic agents.
(60 minutes) (in color) **ACP 2857481**

WHAT CAROTID ARTERIOGRAPHY CAN TELL YOU, with Michael D. F. Deck, M.D., Associate Attending Radiologist, Memorial Sloan-Kettering Cancer Center, and Associate Professor of Radiology, Cornell University Medical Center, New York City. Skull films and brain scans show bone metastases in a patient with inoperable lung cancer. Following irradiation, she has progressively severe headaches and slight dementia. Does she have additional metastases in the brain? Should she have more irradiation? With this patient, Dr. Deck demonstrates carotid arteriography and the value of the procedure.
(17 minutes) (in color) **2318909**

WHAT YOU AND YOUR PATIENT SHOULD KNOW ABOUT CORONARY ARTERIOGRAPHY, with F. Mason Sones, Jr., Director of the Department of Cardiovascular Disease and Cardiac Laboratory; and Donald B. Effler, M.D., Director of the Department of Cardiovascular and Thoracic Surgery, The Cleveland Clinic. What is coronary arteriography? Which patients are candidates? Which patients are not? What information does the consultant need? What happens during the procedure? These questions are answered during this program which includes a demonstration of coronary arteriography.
(24 minutes) (in color) **2318708**

WHEN SHOULD HYPERTENSION BE TREATED? with Edward Fries, M.D., Senior Medical Investigator, VA Hospital, Washington, D.C.; and Ray W. Gifford, Jr., M.D., Department of Nephrology, Cleveland Clinic. Dr. Fries, winner of the Lasker Award, and Dr. Gifford review, through patient interviews, the recent changes in criteria for intervention in hypertension.
(19 minutes) (in color) **2316203**

DERMATOLOGY

A CURRENT APPROACH TO POISON OAK/POISON IVY DERMATITIS, with William L. Epstein, M.D., Professor and Chairman, Department of Dermatology, University of California, San Francisco. Dr. Epstein will demonstrate various treatments for severe and mild cases of poison oak and poison ivy dermatitis, along with the problems of re-exposure, cross-sensitivity with other substances and other dermatological complications. He will also describe various de-sensitizing and hypo-sensitizing agents. This program will benefit the non-dermatologist.
(15 minutes) (in color) **0313364**

A PRACTICAL APPROACH TO ALLERGIC DERMATOSES IN CHILDREN, with Vincent J. Fontana, M.D., Professor of Clinical Pediatrics, New York University College of Medicine, New York City. Dr. Fontana demonstrates ways in which the general physician can arrive at positive diagnoses of both common and less-frequently encountered allergic dermatoses in children. He follows each diagnosis with its recommended treatment.
(18 minutes) (in color) **1614937**

ALL ABOUT WARTS, With Silas E. O'Quinn, M.D., Dermatologist and Dean of Medicine, Louisiana State University School of Medicine, New Orleans. Identification and three quick office techniques for removing these benign but unsightly growths are demonstrated.
(13 minutes) (in color) **0120539**

ALOPECIA IN DIAGNOSIS, with Norman Orentreich, M.D., Professor of Dermatology and Syphilology, New York University School of Medicine, New York City. Hair loss: an insult to vanity or a sign of systemic disease? This program shows how to recognize those cases that are more than just a cosmetic embarrassment.
(18 minutes) (in color) **0119938**

AN EFFECTIVE WAY TO CONTROL PSORIASIS, with Paul Lazar, M.D., Associate Professor of Dermatology, Northwestern University School of Medicine, Chicago. The treatment most likely to control psoriasis with emphasis on variations to suit patient age, motivation, occupation, and body area involvement.
(18 minutes) (in color) **0519825**

BURN: TREATMENT — PART I

The course of treatment of the burn patient is demonstrated by Charles L. Fox, Jr., M.D., Professor of Microbiology, Columbia University College of Physicians and Surgeons.
(15 minutes) (in color) **0207312**

COMMON SKIN DISORDERS IN THE FIRST YEAR OF LIFE, with David L. Cram, M.D., Chief of the Dermatology Clinic, University of California at San Francisco. How to distinguish among the variety of skin eruptions you may see in infants.
(15 minutes) (in color) **0322383**

CUTANEOUS SIGNS OF INTERNAL MALIGNANCY, with Irwin M. Braverman, M.D., Professor of Dermatology, Yale University School of Medicine, New Haven, Connecticut.

The signs can help—*if* you can read them. A dermatologist demonstrates the workup for a patient with a suspected underlying malignancy.
(16 minutes) (in color) **0323586**

DIAGNOSING COMMON SMALL SKIN LESIONS, with Robert Auerbach, M.D., Assistant Clinical Professor of Dermatology, New York University School of Medicine, New York City. Before the biopsy Dr. Auerbach shows you the subtle clinical differences between nevi and melanomas, and helps you sort out benign seborrheic keratoses, pre-malignant keratoses, and carcinoma.
(16 minutes) (in color) **0418345**

THE DIAGNOSIS OF SYSTEMIC LUPUS ERYTHEMATOSUS, with Naomi Rothfield, M.D., Professor of Medicine, and Chief, Arthritis Section, University of Connecticut School of Medicine, Farmington, Connecticut. Demonstrating the early detection of S.L.E. through clinical and laboratory findings. (19 minutes) (in color) **0419951**

DIAGNOSTIC CUTANEOUS SIGNS OF SYSTEMIC DISEASE, with Denny L. Tuffanelli, M.D., Assistant Clinical Professor of Dermatology, University of California at San Francisco. A noted dermatologist uses 13 examples to show how observation of the skin can lead to diagnosis of severe systemic disease. (19 minutes) (In color) **0413926**

DIAGNOSTIC DIFFICULTIES ON BLACK SKIN, with Betty Fischmann, M.B., B.S., M.R.C.P.Ed., Chief of Dermatology, Medical Service, Veterans Administration Hospital and Dermatologist at Howard University and Georgetown University.

Skin signs, particularly erythema, are hard to see on black patients. Further, black patients experience disorders of pigmentation, keloid formation and problems of hair growth more often than whites do. Using two patients and a number of slides, Dr. Fischmann shows the viewer how to reach a diagnosis based on skin signs in the black patient.
(19 minutes) (In color) **0417338**

THE HAND AS AN INDICATOR OF SYSTEMIC DISEASE, with Marguerite Lerner, M.D., Clinical Professor of Dermatology, Yale University School of Medicine, New Haven, Connecticut. Doctor Lerner investigates the cause of several hand complaints, including Raynaud's phenomenon, xanthomas, and telangiectasia.
(19 minutes) (In color) **0821337**

HERPES SIMPLEX: VIRAL DILEMMA, with Richard C. Gibbs, M.D., Associate Professor of Clinical Dermatology, New York University Medical Center, New York City. A clinical presentation, stressing some precautions in the differential diagnosis and treatment of the herpes simplex virus, Types I and II.
(13 minutes) (In color) **0820135**

HEXACHLOROPHENE: OPEN TO DEBATE, with Harold C. Neu, M.D., Associate Professor of Medicine and Chief, Division of Infectious Diseases; Stanley James, M.D., Professor of Pediatrics and Chairman of the American Academy of Pediatrics Committee on the Fetus and Newborn; Carl Nelson, M.D., Professor of Dermatology and President of the American Dermatological Association. All of the participants are on the faculty of the Columbia University College of Physicians and Surgeons, New York City.

Since December 15, 1971, bathing newborns with hexachlorophene, routine in most nurseries, has been banned by the F.D.A. and the American Academy of Pediatrics—or has it? Our panel looks at this problem from several angles and comes up with some interesting conclusions. (16 minutes) (In color) **0815520**

HOW I APPROACH ATOPIC DERMATITIS, with Marion B. Sulzberger, M.D., Professor Emeritus, New York University School of Medicine, and Clinical Professor of Dermatology, University of California in San Francisco. Atopic eczema is a perplexing problem for the dermatologist and the general practitioner. Dr. Sulzberger describes the "chain multiple factor" approach to the disease in terms of occurrence and treatment.
(15 minutes) (In color) **0813419**

LONG-TERM MANAGEMENT OF S.L.E., with Naomi F. Rothfield, M.D., Professor of Medicine and Chief, Arthritis Division, University of Connecticut School of Medicine, Farmington, Conn. Specific drugs and general life adjustments are important to the treatment course of systemic lupus erythematosus. However, the key to managing S.L.E., demonstrated here, is to identify symptoms and signs of impending flare-ups.
(17 minutes) (In color) **1220019**

OFFICE TREATMENT OF SKIN CANCER, with Rex A. Amonette, M.D., Chemosurgeon, Department of Dermatology, University of Tennessee College of Medicine, Memphis. Diagnosis and treatment of potential malignancy and skin cancer are demonstrated. Includes the use of fluorouracil, biopsy, curettage and electrodesiccation, total excision, cryosurgery, irradiation, and chemosurgery. (19 minutes) (In color) **1521010**

PARASITIC INFESTATION: LOOK FOR LICE, with Silas E. O'Quinn, M.D., Professor of Dermatology and Dean of Medicine; and Harold Trapido, Ph.D., Professor of Tropical Medicine and Medical Parasitology, both at Louisiana State University School of Medicine in New Orleans. A comprehensive look at what *may* be causing that terrible itch. Knowledge of your patients' hygiene does not rule out lice.
(11 minutes) (In color) **1620645**

PARASITIC INFESTATION: SCABIES, with Silas E. O'Quinn, M.D., Professor of Dermatology and Dean of Medicine; and Harold Trapido, Ph.D., Professor of Tropical Medicine and Medical Parasitology, both at Louisiana State University School of Medicine in New Orleans. Everything you always wanted to know about mites but (were too busy) (forgot) to ask.
(14 minutes) (In color) **1621348**

SKIN ERUPTIONS: DUE TO DRUGS?, with David L. Cram, M.D., Chief of the Dermatology Clinic, University of California at San Francisco. Is your patient's skin reaction due to drugs? Is it dangerous enough to warrant removing a necessary medication? Which of several drugs is the culprit?
(16 minutes) (In color) **1922140**

STASIS ULCERS OF THE ANKLE, with Robert A. Nabatoff, M.D., Chief, Vascular Surgery, Department of Obstetrics and Gynecology, at Mount Sinai Medical Center, New York City. How to treat these common, yet often puzzling, symptoms before surgery for the underlying vascular insufficiency. (14 minutes) (in color) **1918430**

SYSTEMIC LUPUS ERYTHEMATOSUS, with Werner F. Barth, M.D., Department of Medicine; and Harry M. Robinson, M.D., Department of Dermatology; both of University of Maryland School of Medicine; and Donald T. Lewers, M.D., Division of Nephrology, Maryland General Hospital.

Three patients illustrate the clinical variability of systemic lupus erythematosus, as well as differences in therapy. The immunopathology is discussed, with particular attention to recent concepts of pathogenesis. Factors adversely affecting prognosis and those warranting more aggressive therapy are underscored. Also discussed are recent developments using immunofluorescent examination of skin biopsy to distinguish systemic lupus erythematosus from discoid lupus and other connective tissue diseases. A Television Hospital Clinic of the American College of Physicians, 1972. Please inquire for special rental information.
(60 minutes) (in color) **ACP 2837247**

THE TREATMENT OF ACNE, with Paul Lazar, M. D., Associate Professor of Dermatology, Northwestern University Medical School, Chicago. When an adolescent with acne doesn't respond to topical therapy and a year of tetracycline treatment do you consider steroid therapy, topical or systemic, or acne surgery? Dr. Lazar recommends the progression to follow in treating acne. (19 minutes) (in color) **2020133**

WHAT'S CAUSING THE INCREASED INCIDENCE OF PHOTOSENSITIVE REACTIONS?, with John H. Epstein, M.D., Associate Clinical Professor of Dermatology, University of California at San Francisco, and Chief of Dermatology at Mount Zion Hospital and Medical Center in San Francisco. Increased public obsession with sunbathing and an ever increasing number of photosensitizers in our environment are reaching the stage where almost every physician can expect to see patients exhibiting phototoxic or photoallergic reactions. Diagnosis and treatment of the following are explored in this telecast: phototoxicity, photoallergy, exogenous photosensitizers, therapeutic and antibacterial agent reactions and plant-induced photosensitivity.
(15 minutes) (in color) **2313502**

ZOSTER: ITS COURSE AND TREATMENT, with Richard C. Gibbs, M.D., Associate Professor of Clinical Dermatology, and Philip A. Brunell, M.D., Director, Laboratory of Infectious Disease, Pediatrics Department; both of New York University Medical Center in New York City. Recommends treatment for the severe pain of this generally one-time, but unforgettable, illness.
(9 minutes) (in color) **2620201**

DRUG ABUSE

DOWN AND OUT IN THE E.R. Barbiturate overdose, accidental or intentional, is the number one drug abuse problem presenting in Emergency Rooms. Dr. George Gay of the Haight-Ashbury Free Medical Clinic in San Francisco and Dr. Eric Comstock, director of the Institute of Toxicology at Baylor University, join medical student John Rose of Baylor to demonstrate recommended procedures for the diagnosis and treatment of a barbiturate overdose crisis.
(26 minutes) (in color) **SAMA 2810859**

DRUG ABUSE: RECOGNIZING AND TREATING ACUTE REACTION TO AMPHETAMINES AND SEDATIVE HYPNOTICS, with David E. Smith, M.D., Instructor, Department of Pharmacology, University of California School of Medicine, San Francisco; and Medical Director, Haight-Ashbury Medical Clinic.

Why do most young drug users avoid family physicians when their drug experiences get them in trouble? Dr. Smith discusses this problem with several drug users. He also gleans detailed descriptions from the young users of drug "trips" and "bad trips." Finally, Dr. Smith describes the kinds of adverse reactions to amphetamines and sedative hypnotics, and outlines suggested therapy.
(19 minutes). **0411822**

DRUG ABUSE: RECOGNIZING AND TREATING ACUTE REACTIONS TO HALLUCINOGENS, with David E. Smith, M.D., Instructor, Department of Pharmacology, University of California School of Medicine, San Francisco; Medical Director, Haight-Ashbury Medical Clinic.

In this telecast, experienced young people candidly discuss "bad trips" and other adverse symptoms connected with drug abuse. The program concludes with a concise rundown of recommended therapy. (19 minutes). **0411923**

EARLY DIAGNOSIS OF ALCOHOLISM, with Marvin A. Block, M.D., Vice President of the AMA Society on Alcoholism, and Associate Professor, State University of New York at Buffalo. "There are all kinds of alcoholisms and all kinds of alcoholics." Dr. Block provides specific criteria by which you may judge the kind of alcoholism and the stage of the disease.
(24 minutes) (in color) **0516820**

GETTING A FIX ON HEROIN. Treatment of heroin-addicted patients is clouded by popular misconceptions and mythologies about heroin use and treatment. SAMA cameras take you to drug abuse treatment facilities in New York and San Francisco to explore the problems of the drug addict and the problems of treating him.

Speaking for SAMA is Dahlia Kirkpatrick, a third-year student at Yale who discusses the issues with Dr. Herbert Kleber, Director of the Drug Abuse Unit of the Connecticut Mental Health Center. (34 minutes) (in color) **SAMA 2810453**

IF THE DOCTOR'S INTO DRUGS . . . Studies show the incidence of drug abuse and alcoholism to be much higher among physicians than among the general population. Should this problem be chalked off as an occupational hazard? Or can it be controlled?

What might one do for a colleague developing dependence on drugs or alcohol? How does one avoid such dependence himself? Three psychiatrists who have studied and treated drug dependent and alcoholic physicians discuss these issues. (17 minutes) (in color) **SAMA 2810555**

MANAGEMENT OF ADOLESCENT SYMPTOMS. Methods of dealing with the problems of adolescents — particularly experimentation with sex and drugs — are demonstrated with groups of teenagers, by Steven R. Homel, M.D., Department of Pediatrics, Jefferson Medical College and Hospital of Philadelphia, Pa.
(18 minutes). **1308004**

MEDICAL PROBLEMS OF ADOLESCENT HEROIN ABUSE, with Michael I. Cohen, M.D., Director of the Division of Adolescent Medicine; and Iris Litt, M.D., Assistant Director. The Division is part of the Department of Pediatrics, Montefiore Hospital in New York City.

An in-depth interview is conducted with a 14-year-old heroin addict under treatment. The youth describes his life with drugs followed by comments on the interview by the physicians.
(14 minutes) (in color) **1311025**

METHADONE MAINTENANCE FOR THE HARD CORE HEROIN ADDICT, with Ray E. Trussell, M.D., Director of Beth Israel Medical Center in New York City; Harvey Gollance, M.D., Associate Director; and Harold L. Trigg, M.D., Unit Director, Methadone Maintenance Treatment Program at the Morris J. Bernstein Institute of Beth Israel.

For the acute heroin addict, Methadone may be the only answer to a normal existence. Physicians at the Beth Israel clinic describe their Methadone therapeutic program. The effectiveness of their program is dramatized by an interview with an addict in the program. The information in this telecast will interest general physicians and specialists. (21 minutes). **1311231**

NAME YOUR POISON: ALCOHOL. Many physicians and medical students still treat alcoholics as second-class patients. Because of ingrown cultural attitudes, early alcoholism often goes undetected and untreated. Med students meet with a recovered alcoholic, with Dr. Marvin Block of the State University of New York at Buffalo, and Dr. Frank Seixas, Medical Director of the National Council on Alcoholism, to discuss ways to detect and help incipient alcoholics using science to replace serendipity.
(37 minutes) (in color) **SAMA 2810251**

THE MULTIPHASIC TREATMENT OF ALCOHOLISM, with Albert N. Brown-Mayers, M.D., Director of the Alcoholic Service; Edward E. Seelye, M.D., Unit Administrator of the Alcoholic Service; and Leonard R. Sillman, M.D., Attending Psychiatrist, all of the Westchester Division of the New York Hospital-Cornell Medical Center, White Plains, N.Y. A new, formalized approach to the treatment of alcoholism is suggested. Presenters show how residential alcoholic treatment works and outline a complete program, many elements of which you'll find practical and effective in your own practice. (29 minutes) (in color) **1316946**

THE NEW HEROIN SCENE: A PERSPECTIVE FOR PHYSICIANS, with George R. Gay, M.D., Chief of the Medical Section and Director of the Heroin Clinic of the Haight-Ashbury Clinic, and Instructor in Toxicology at the University of California at San Francisco. A new group of drug abusers appears to be growing in numbers across the country — the "middle-class junkies" who are "dropping out" of society on heroin. The dimensions of the problem are explored by an authority on drug abuse. Dr. Gay also outlines treatment for the young abusers who are consumed by the chemical aspects of life rather than the human aspects.
(22 minutes). **1413530**

THRESHOLD TO ADDICTION, with Steven R. Homel, M.D., Department of Pediatrics, Jefferson Medical College and Hospital of Philadelphia. Dr. Homel discusses the reasons for drug addiction with two teen-agers and a young medical student. Their answers are candid, and offer insight into the attitudes of young people today. (23 minutes) (in color) **2010206**

TREATMENT OF ACUTE HEROIN TOXICITY, with David E. Smith, M.D., Director and Founder of the Haight-Ashbury Free Medical Clinic and Assistant Clinical Professor of Toxicology at the University of California in San Francisco; and George R. Gay, M.D., Chief of the Medical Section and Director of the Heroin Clinic with the Haight-Ashbury Clinic and Instructor in Toxicology at the University of California, San Francisco. With growing numbers of heroin abusers in middle-class America, physicians increasingly are going to face the problem of acute heroin overdose — a medical emergency. Two physician-experts put this problem into perspective through patient interviews, describe the "street" treatments that are in use — adding to risk of the complications — and finally present the treatment procedure they employ. (20 minutes). **2013422**

EMBRYOLOGY

EGG TRANSPORT IN MAMMALS, with Richard J. Blandau, M.D., Professor of Biological Structures, University of Washington School of Medicine. Discussion and cinemicrographic visualization of egg transport in rabbits, rats, and humans. (15 minutes). **0501404**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART I. Alvin F. Goldfarb, M.D., Assistant Professor of Obstetrics and Gynecology, Jefferson Medical College and Hospital, presents "Evaluating the Infertile Couple." (19 minutes). **0303118**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART II

"The Physio-Anatomic Basis of Fallopian Tube Function." The second program in this continuing series — with Luigi Mastroianni, Jr., M.D., Professor of Obstetrics and Gynecology, University of Pennsylvania School of Medicine. (17 minutes). **0303319**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART III

"Tubal Factor Treatment." Correcting distortion of the tubal ovarian relationships. With Celso-Ramon Garcia, M.D., Associate Professor of Obstetrics and Gynecology, University of Pennsylvania School of Medicine. (16 minutes). **0303620**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART IV

"The Cervix in Infertility." Evaluation of the cervical factor in cases of female infertility — with Kamran S. Moghissi, M.D., Associate Professor of Obstetrics and Gynecology, Wayne State University School of Medicine. (17 minutes). **0304421**

THE CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART V

"New Research," a discussion and demonstration of the use of frozen sperm for artificial insemination; immunologic aspects of infertility; and new laparoscopic techniques. (18 minutes). **0304622**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART VI — INDICES OF OVULATION

The tests which are available to determine the absence or occurrence of ovulation are described by Howard Balin, M.D., Chief, Gynecic Research Unit, Pennsylvania Hospital, Philadelphia. (26 minutes). **0304923**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS — PART VII — INDUCTION OF OVULATION. The several methods available for treating the anovulatory, infertile female are evaluated by Alvin F. Goldfarb, M.D., Assistant Professor of Obstetrics and Gynecology, and Abraham E. Rakoff, M.D., Professor of Obstetrics and Gynecology, Jefferson Medical College and Hospital, and Howard Balin, M.D., Chief, Gynecic Research Unit, Pennsylvania Hospital. (15 minutes). **0305024**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS — FINAL PROGRAM — THE SUMMATION. Alvin F. Goldfarb, M.D., Assistant Professor of Obstetrics and Gynecology, Jefferson Medical College and Hospital, presents a review and a summation of the seven programs in the series on human reproductive problems. (20 minutes). **0305325**

EMERGENCY MEDICINE AND TRAUMA

ACID-BASE IMBALANCE: THREE PROGRAMS. *"For decades, acid-base metabolism with its cabalistic terminology has intrigued and, incidentally, terrorized many clinicians. Actually, the whole subject of acid-base metabolism and its clinical application is just another facet of medical science that, once understood, presents itself in a straightforward, logical manner . . ." Hugh J. Carroll, M.D.*

The following three programs were produced in cooperation with the Office of Continuing Education of the State University of New York, Downstate Medical Center, Brooklyn, New York.

ACID-BASE IMBALANCE: PATHOGENESIS, with Hugh J. Carroll, M.D., Associate Professor of Medicine, Director of the Electrolyte and Hypertension Section, Department of Medicine, Downstate Medical Center. A presentation of the primary disturbances in metabolic and respiratory acid-base imbalances, their mechanisms of compensation, and the clinical situations in which they may occur.
(22 minutes) (in color) **0122444**

ACID-BASE IMBALANCE: RECOGNITION AND MANAGEMENT, with Hugh J. Carroll, M.D., Associate Professor of Medicine, Director of the Electrolyte and Hypertension Section, Department of Medicine, Downstate Medical Center. When blood gases and other clinical studies confirm the presence of an acid-base disturbance, it is the physician's part to reverse the imbalance directly or to treat the underlying disease so that the patient's own corrective mechanisms can restore the normal state.
(15 minutes) (in color) **0122445**

KETOTIC AND NONKETOTIC COMA: MECHANISMS AND TREATMENT, with Hugh J. Carroll, M.D., Associate Professor of Medicine, Director of the Electrolyte and Hypertension Section, Department of Medicine, Downstate Medical Center. Pathophysiology, diagnosis, and management in diabetic ketoacidosis and hyperosmolar, hyperglycemic, nonketotic coma.
(18 minutes) (in color) **1122406**

APPROACH TO GASTROINTESTINAL PAIN, with Howard M. Spiro, M.D., Chief of Gastroenterology, Yale-New Haven Hospital, and Professor of Medicine, Yale University School of Medicine, New Haven, Connecticut. Three carefully coached actors appear as patients seeking diagnoses. In this program Dr Spiro deals with abdominal emergencies. The viewer is invited to exercise his diagnostic skills alongside a leading gastroenterologist. (19 minutes) (in color) **0115226**

BEDSIDE PULMONARY ARTERY CATHETERIZATION, with T. Crawford McAslan, M.D., Associate Clinical Director, Maryland Institute for Emergency Medicine, and Professor of Anesthesiology, University of Maryland School of Medicine, Baltimore.

An introduction to the Swan-Ganz flow-directed, balloon-tipped catheter. Indications, method of insertion at bedside, and techniques for avoiding complications. (15 minutes) (in color) **0223621**

BURN — PART I. The course of treatment of the burn patient is demonstrated by Charles L. Fox, Jr., M.D., Professor of Microbiology, College of Physicians and Surgeons, Columbia University.
(15 minutes) (in color) **0207312**

BURN—PART II—SYSTEMIC TREATMENT. The guidelines for treatment of burns and the need for constant review of the physiologic status of the patient are presented by Charles L. Fox, Jr., M.D., Professor of Microbiology, College of Physicians and Surgeons, Columbia University. Dr. Fox also demonstrates results of topical treatment and grafting procedures.
(16 minutes) (in color) **0207413**

THE COMATOSE PATIENT: IMMEDIATE MANAGEMENT AND EVALUATION, with Jerome S. Resnick, M.D., Director of Neurology, Stamford Hospital, Stamford, Connecticut, and Clinical Associate Professor of Neurology, New York Medical College.

Problem: coma. History: unknown. Dr. Resnick shows how to find the cause of coma using your eyes and nose, a flashlight, ophthalmoscope, pin and blood pressure cuff. This program was produced in cooperation with the Department of Continuing Medical Education, New York Medical College.
(16 minutes) (in color) **0323990**

THE COMATOSE PATIENT: PATHOPHYSIOLOGY, with Jerome S. Resnick, M.D., Director of Neurology, Stamford Hospital, Stamford, Connecticut, and Clinical Associate Professor of Neurology, New York Medical College.

Definition, etiologies, and mechanisms of prolonged unconsciousness, presented in cogent, organized and practical style. This program was produced with the cooperation of the Department of Continuing Medical Education, New York Medical College. (16 minutes) (in color) **0323889**

COMMON PROFESSIONAL FOOTBALL INJURIES, MECHANICS AND EFFECTS, with James A. Nicholas, M.D., Associate Professor of Clinical Surgery (Orthopedic), Cornell University Medical College and Team Physician, the New York Jets.
(18 minutes). **0302330**

DIAGNOSING THE MALTREATMENT SYNDROME IN CHILDREN, with Vincent J. Fontana, M.D., Director of the Department of Pediatrics, St. Vincent's Hospital; and Medical Director, New York Foundling Hospital, both in New York City. In full agreement with a JAMA editorial stating that the "maltreated or battered child could be the leading cause of death in infants and children," Dr. Fontana describes the presenting signs that should make any physician suspect battered child or maltreatment syndrome — a diagnosis that may prevent future trauma to the child and may even save its life. (14 minutes) (in color) **0414329**

DIAGNOSIS AND TREATMENT OF HAND INJURIES. A key hint in making repairs of badly crushed hands is that if any connection exists at all, the injured part should not be severed, in the view of Ronald B. Berggren, M.D., Assistant Professor of Surgery and Director, Division of Plastic Surgery at Ohio State University College of Medicine. This and other aspects of hand injury are discussed among Dr. Berggren, and Robert J. Duran, M.D., Assistant Professor of Surgery, Division of Plastic Surgery and Co-Director, Hand Clinic, and Carl R. Coleman, M.D., Clinical Assistant Professor of Surgery, Division of Orthopedics, and Co-Director, Hand Clinic, Ohio State University College of Medicine.
(20 minutes). **0406808**

DIAGNOSTIC THORACENTESIS, with James W. Kilman, M.D., Associate Professor of Surgery, and Thomas E. Williams, Jr., M.D., Assistant Professor of Surgery, Ohio State University College of Medicine, Columbus. Thoracentesis for both diagnosis and emergency therapy is performed following a discussion of the causes, signs, and symptoms of pleural effusion.
(19 minutes) (in color) **0419248**

DOWN AND OUT IN THE E.R. Barbiturate overdose, accidental or intentional, is the number one drug abuse problem presenting in Emergency Rooms. Dr. George Gay of the Haight-Ashbury Free Medical Clinic in San Francisco and Dr. Eric Comstock, director of the Institute of Toxicology at Baylor University, join medical student John Rose of Baylor to demonstrate recommended procedures for the diagnosis and treatment of a barbiturate overdose crisis.
(26 minutes) (in color). **SAMA 2810859**

DRINKERS IN CRISIS, with Henry D. Abraham, M.D., Harvard Medical School, and Chief, Marlborough-Westborough Unit, Westborough State Hospital, Westborough, Massachusetts; and John A. Renner, M.D., Director of the Alcoholism Clinic, Massachusetts General Hospital, Boston. The Scene: The psychiatric emergency department at Mass. General. The Players: Walk-in alcoholics seeking help. The Plot: How to use an alcoholic's time of crisis to set the stage for short- and long-term care.
(16 minutes) (in color) **0421255**

EMERGENCY CARDIAC CARE, with William J. Grace, M.D., Department of Medicine, New York University School of Medicine, and Joseph G. Hayes, M.D., Cornell University Medical College, New York City; Costas T. Lambrew, M.D., Department of Medicine, Meadowbrook Hospital, East Meadow, New York; and Joseph Schluger, M.D., Department of Medicine, Long Island College Hospital, Brooklyn, New York. A personal plan of action is proposed to help the physician deal with patients showing early signs of myocardial infarction. Optional emergency cardiac care systems adaptable to different community needs are described, and recently modified CPR techniques are demonstrated. A Television Clinic of the American College of Physicians, 1974. Please inquire for special rental information.
(60 minutes) (in color) **ACP 2867482**

EMERGENCY CLOSED TUBE THORACOSTOMY, with James W. Kilman, M.D., Associate Professor of Surgery, and Thomas E. Williams, M.D., Assistant Professor of Surgery, Ohio State University College of Medicine, Columbus. The causes, consequences, symptoms, and signs of pneumothorax are reviewed and an emergency closed tube thoracostomy is performed.
(20 minutes) (in color) **0519123**

EMERGENCY ORTHOPEDIC MANAGEMENT. Paul R. Meyer, Jr., M.D., Department of Orthopedics, Tulane University School of Medicine, demonstrates emergency splinting at the scene of an accident. (7 minutes). **0501308**

EMERGENCY TRANSVENOUS CARDIAC PACING, with Doris J. W. Escher, M.D., Attending Physician in the Division of Medicine and Physician-in-Charge of Cardiac Catheterization, Montefiore Hospital and Medical Center, Bronx, New York. The technique for emergency transvenous cardiac pacemaker insertion at the patient's bedside is shown. Intracardiac electrogram patterns are correlated with catheter position, from high right atrium to the target position at the apex of the ventricle. (19 minutes) (in color) **0520426**

FRACTURED FACES — THE LOWER FACE. From the First Annual Symposium on Maxillofacial Trauma, Washington, D.C., Lester M. Cramer, M.D., Professor and Chairman, Section of Plastic Surgery, Temple University Health Sciences Center, demonstrates operative and fixation procedures for fractures of the mandible.
(15 minutes). **0605608**

FRACTURED FACES — THE MIDDLE FACE. Lester M. Cramer, M.D., Professor and Chairman of the Section of Plastic Surgery, Temple University Health Sciences Center, reviews the structure of the middle face, the types of fractures which occur in that area, and the various forms of treatment including internal fixation.
(18 minutes). **0605509**

INTERNAL JUGULAR VEIN CATHETERIZATION, with T. Crawford McAslan, M.D., Associate Clinical Director, Maryland Institute for Emergency Medicine, and Professor of Anesthesiology, University of Maryland School of Medicine, Baltimore.

The internal jugular vein . . . an attractive alternative to the basilic with its high failure rate and the subclavian with its high incidence of pneumothorax.

(11 minutes) (in color) **0923735**

LEGAL PROBLEMS IN THE EMERGENCY ROOM. When can a minor be treated without parental consent? What special precautions should be taken with victims of criminal violence? Points to know about the law to protect a patient's rights and safeguard physician liability are discussed by William Mangold, M.D., J.D., resident in surgery at Bexar County Hospital, San Antonio, and member of the Texas bar; and Crawford Morris, Esq., trial lawyer specializing in medical malpractice cases for Arter & Hadden Associates, Cleveland.

(18 minutes) (in color) **SAMA 2811063**

MAINTAINING THE AIRWAY, with Richard Zepernick, M.D., Instructor in Anesthesiology, Department of Surgery, Tulane University School of Medicine. How to keep the patient breathing in an emergency. (16 minutes). **1301201**

MANAGEMENT OF ACUTE HEAD INJURIES, with William E. Hunt, M.D., Professor and Director of the Division of Neurosurgery; and W. George Bingham, Jr., M.D., Assistant Professor, Division of Neurosurgery, both with the Ohio State University College of Medicine, in Columbus. Two neurosurgeons demonstrate the diagnostic procedure they follow in cases of acute head injuries. They also show the complications that can arise during the hours immediately after the injuries, and the neurosurgical emergencies that can develop. (15 minutes) **1314140**

MANAGEMENT OF ACUTE POISONING, with Jay M. Arena, M.D., Director, Poison Control Center of the Duke University Medical Center, Durham, N.C., and former President, American Academy of Pediatrics. Dr. Arena shows how to treat common and uncommon poisoning episodes on an emergency basis.

(22 minutes) (in color) **1317648**

MANAGEMENT OF ACUTE SPINAL INJURIES, with William E. Hunt, M.D., Professor and Director of the Division of Neurosurgery; W. George Bingham, Jr., M.D., Assistant Professor, Division of Neurosurgery; and Stephen Natelson, M.D., Senior Resident in Neurosurgery; all with the Ohio State University College of Medicine in Columbus. Three specialists describe the precautions that are necessary in handling patients with spinal injuries through the stages in the emergency room, in radiology and during the period of rehabilitation. (18 minutes) (in color) **1314038**

MANAGEMENT OF ASPIRIN POISONING, with Jay M. Arena, M.D., Director, Poison Control Center, Duke University Medical Center, Durham, N. C., and former President, American Academy of Pediatrics.

Dr. Arena draws on 35 years' experience to give a practical approach to handling the most common cause of poisoning in children.

(13 minutes) (in color) **1317347**

MANAGEMENT OF THE BATTERED CHILD SYNDROME, with C. Henry Kempe, M.D., Professor and Chairman of Pediatrics; Brandt F. Steele, M.D., Professor of Psychiatry; and Helen Alexander, Medical Social Worker, Supervisor of Lay Therapists, Battered Child Program. All three are with the University of Colorado Medical Center. The reasons for abuse become apparent in an unrehearsed conversation with the mother of a patient. Three experts offer some practical advice on coping with parents once child abuse has been diagnosed and the underlying problems identified.

(18 minutes) (in color) **1314441**

MANAGEMENT TIPS FOR SOFT TISSUE INJURIES IN CHILDREN, with Thomas S. Morse, M.D., Associate Professor of Surgery, Ohio State University College of Medicine, Columbus, Ohio. The surgical technique for repairing a laceration in a child is about the same as that used for adults, but there are ways to make it easier. In this program, special attention is given to dressings, restraints and slings, as Dr. Morse shares his "little tricks" that help make it easier to deal with children. (18 minutes) (in color) **1319552**

MEDICAL PROBLEMS ENCOUNTERED WITH BASEBALL PLAYERS, with Joseph T. Coyle, M.D., Clinical Associate, Bone and Joint Surgery, Stritch School of Medicine, Loyola University and Team Physician, the Chicago White Sox. (14 minutes). **1302426**

THE MILITARY DOCTOR, with Commander Richard M. Escajeda, M.D. Report from Vietnam on military and civilian care.

(21 minutes). **1302133**

NEAR DROWNING: WATCH THE BLOOD GASES, with Norman L. Fine, M.D., Chief, Respiratory Services, The Griffin Hospital, Derby, Conn., and Assistant Clinical Professor of Medicine, Yale University Medical School, New Haven. The model of the fatally-drowned person is no longer relevant in treating the survivor of *near* drowning. This program brings you up to date.
(15 minutes) (in color) **1422940**

OFFICE ORTHOPAEDICS: AFTER THE FALL, with Robert E. Leach, M.D., Professor and Chairman, Department of Orthopaedic Surgery, Boston University Medical Center, Boston.

Dr. Leach demonstrates casting and wrapping procedures for common orthopaedic injuries—dislocated shoulder, tibia and fibula fractures, ankle sprains and fractures.
(18 minutes) (in color) **1517208**

PRE-HOSPITAL CORONARY CARE — A MODEL FOR YOUR COMMUNITY, with Eugene Nagel, M.D., Associate Professor, Department of Anesthesiology, University of Miami School of Medicine, and Clinical Director, Department of Anesthesiology, Jackson Memorial Hospital, Miami, Florida.

How quickly can your community get optimum coronary care facilities to a patient in cardiac arrest or ventricular fibrillation? Dr. Nagel shows how the Miami Emergency Rescue Service is set up, demonstrates how quickly it works in a simulated rescue, and shows what is needed to implement a complete mobile emergency health care system. (21 minutes) (in color) **1615438**

PRIMARY EVALUATION OF THE MULTIPLY INJURED PATIENT from the Ohio State University College of Medicine, with Larry C. Carey, M.D., Professor and Chairman of the Department of Surgery, and William E. Hunt, M.D., Professor and Director of the Division of Neurologic Surgery. The first 90 seconds in the emergency room are crucial. Evaluating the nature and extent of the patient's injuries in a proper sequence greatly increases the patient's chances for survival. This program was produced in cooperation with the Center for Continuing Medical Education, Ohio State University. (16 minutes) (in color) **1623049**

PRIMARY TREATMENT OF SOFT TISSUE INJURIES, with Ronald B. Berggren, M.D., Professor and Director, Division of Plastic Surgery, Ohio State University College of Medicine, Columbus. The three Ds of sound management, Diagnosis, Debridement and Definitive Care, are demonstrated. Special emphasis is given to ways to avoid particular deformities.
(19 minutes) (in color) **1619443**

RESPIRATORY DISTRESS SYNDROME OF THE ADULT: TREATMENT WITH PEEP, with Robert M. Rogers, M.D., Professor of Medicine, Associate Professor of Physiology, and Chief of the Pulmonary Disease Section, University of Oklahoma Health Sciences Center, Oklahoma City. How a reasonable therapeutic program can significantly reduce high mortality from RDSA, the major pulmonary complication from trauma, hemorrhage, surgery, septicemia, and shock.
(16 minutes) (in color) **1821131**

SAVING LIVES: IMMEDIATE CARE FOR COMMON EMERGENCIES. Could you save a life if your neighbor's baby was choking? Could you effectively stop massive bleeding? When accidents happen, friends of victims are likely to turn to you assuming you know more about emergency medical treatment. Do you? Hugh Hill of Medical College of Virginia and Marcia Silver of Johns Hopkins join David Pilcher, M.D., Associate Professor of Surgery at the University of Vermont School of Medicine, and C. Earl Gettinger, Coordinator of Emergency Medical Services for the Vermont State Health Department, to discuss and demonstrate emergency treatment in cases of respiratory failure, massive bleeding and common household poisonings.
(23 minutes) (in color) **SAMA 2811461**

SECONDARY AND TERTIARY EVALUATION OF THE MULTIPLY INJURED PATIENT, from the Ohio State University College of Medicine, with Larry C. Carey, M.D., Professor and Chairman, Department of Surgery, and William E. Hunt, M.D., Professor and Director, Division of Neurologic Surgery, Columbus. When the multiply injured patient arrives in the emergency room, various examinations help identify the extent of injury. This program outlines each examination. The program was produced in cooperation with the Center for Continuing Medical Education, Ohic State University.
(14 minutes) (in color) **1923142**

SKELETAL TRACTION — THE TEAM APPROACH — PART I — ADMISSION AND EMERGENCY CARE. The emergency management of a fractured femur is demonstrated by William McCloud, M.D., Resident in Orthopedic Surgery, Ohio State University Center for Continuing Medical Education, for his departmental team, which includes an R.N., orthopedic orderly, physiotherapist, and occupational therapist. Discussing the rationale for the procedures, he reviews the structure of the femur and the surrounding muscles and how the anatomy and type of fracture dictate the appropriate management. (25 minutes). **2701332**

SNAKEBITE, with Findlay E. Russell, M.D., Ph.D., Professor of Neurology, Physiology and Biology, and Director, Laboratory of Neurological Research, University of Southern California School of Medicine, Los Angeles. How to distinguish the dangerous bites, recommend first aid, and apply emergency and supportive treatment.
(24 minutes) (in color) **1920636**

SPECIAL PROBLEMS OF FACIAL TRAUMA SURGERY — PART I. Examination and repair of lacerations of the soft tissue of the face are discussed and demonstrated by Ross H. Musgrave, M.D., Clinical Associate Professor of Surgery (Plastic), University of Pittsburg School of Medicine. (26 minutes) (in color) **1905412**

TECHNIQUES IN EMERGENCY CARE, with Stephen E. Goldfinger, M.D., Director of Community Programs, Department of Continuing Education and James Dineen, M.D., Fellow in Continuing Education, Harvard Medical School and Massachusetts General Hospital, Boston, Massachusetts.

The procedures and armamentarium of the community hospital emergency room are constantly improving. Physicians who have not served as house officers recently will benefit from this program. Current emergency care is demonstrated with a patient in coma, another with unexplained fever, a third with trauma and, finally, several malingerers and depressives.
(16 minutes) (in color) **2010904**

THE ASPHYXIATING PATIENT: HOW AND WHY I INTUBATE, with Richard J. Ward, Professor, Department of Anesthesiology, University of Washington School of Medicine, Seattle. The steps involved in endotracheal intubation are reviewed and the procedure then performed on a surgical candidate.
(13 minutes) (in color) **0117933**

THE EMERGENCY ROOM: A GROWING PUBLIC UTILITY, with Stephen E. Goldfinger, M.D., Director of Community Programs, Department of Continuing Education; and James Dineen, M.D., Fellow in Continuing Education, Harvard Medical School and Massachusetts General Hospital, Boston, Massachusetts.

Physicians have trained their patients to seek medical attention from emergency rooms after office hours. This common practice has placed staggering demands on hospital emergency facilities. How can the hospital meet these demands? Who will staff the emergency facilities? What technical advances have been introduced to assist ER personnel? Doctors Goldfinger and Dineen present answers to these dilemmas.
(19 minutes) (in color) **0511009**

THERMAL INJURIES: MEDICAL, SURGICAL, AND PSYCHIATRIC CARE, *with an introduction by T. G. Blocker, Jr., M.D., Professor of Surgery and President Emeritus, The University of Texas Medical Branch at Galveston. The following three programs were produced under the supervision of Stephen R. Lewis, M.D., Chief of Plastic Surgery and Director of Continuing Education, UTMB-Galveston.*

KEEPING THE BURN PATIENT ALIVE, with Duane L. Larson, M.D., Professor of Plastic Surgery, UTMB-Galveston, and Director, Shriners Burns Institute, Galveston. A 1-2-3 approach to emergency care for critically-burned patients plus the steps to take in assuring the patient's survival during transportation to a hospital.
(16 minutes) (in color) **1122205**

HOSPITAL BURN CARE: MINIMIZING DEFORMITIES AND OTHER COMPLICATIONS, with Hugo Carvajal, M.D., Pediatric Nephrologist, Shriners Burns Institute, and Assistant Professor of Pediatrics, UTMB-Galveston; and Duane L. Larson, M.D., Director, Shriners Burns Institute, and Professor of Plastic Surgery, UTMB-Galveston. The definitive care of burn patients. Emphasis is placed on daily wound care, fluid replacement and nutrition, and procedures to minimize contracture and hypertrophic scarring before and after grafting.
(22 minutes) (in color) **0822239**

THERMAL INJURY: EMOTIONAL AND PHYSICAL STRESS, with Mary S. Knudson, Ph.D., Chief, Division of Behavioral Sciences, Shriners Burns Institute; Duane L. Larson, M.D., Professor of Plastic Surgery, UTMB-Galveston, and Director, Shriners Burns Institute; and Robert B. White, M.D., Professor of Psychiatry, UTMB-Galveston. Practical methods to combat the physical pain, toxic delirium, helplessness, and regression seen in patients with major burns. (12 minutes) (in color) **2022240**

TIME BORROWERS IN SHOCK, with Leon I. Goldberg, Ph.D., M.D., Professor of Medicine and Pharmacology, and Director of Clinical Pharmacology, Emory University School of Medicine in Atlanta, Georgia. When the physician needs to "borrow time" while treating the underlying causes of shock, a cautious use of sympathomimetic amines is often a worthwhile temporary solution. (14 minutes) (in color) **2020737**

TRANSFUSION THERAPY: THE GROWING IMPACT OF FROZEN BLOOD, with Charles Huggins, M.D., Director of the Blood Bank and Transfusion Service, Massachusetts General Hospital, Boston. Blood transfusion therapy as it is practiced in a major medical center, with guidelines to the use of fresh and freshly frozen components.
(14 minutes) (in color) **2022139**

TRAUMA TO THE LIVER: PRIMARY CARE AND DIAGNOSIS, with Gordon F. Madding, M.D., Associated Clinical Professor of Surgery, University of California School of Medicine, and Associate in Surgery, Stanford University School of Medicine; and Paul A. Kennedy, M.D., Assistant Clinical Professor of Surgery, Stanford University School of Medicine. In the opinion of Drs. Madding and Kennedy, there are ten specific steps that should be taken when a patient presents with traumatic injury to the abdomen. These measures lead most rapidly to effective therapeutic actions.
(18 minutes) (in color) **2014924**

TRAUMA TO THE LIVER: OPERATIVE APPROACH AND SURGICAL PROCEDURE, with Gordon F. Madding, M.D., Associate Clinical Professor of Surgery, University of California School of Medicine and Associate in Surgery, Stanford University School of Medicine; and Paul A. Kennedy, M.D., Assistant Clinical Professor of Surgery, Stanford University School of Medicine. Anatomically and surgically this large, vital organ is not widely understood. Drs. Madding and Kennedy demonstrate a few basic principles which can make emergency treatment of damaged liver safer and surer. (17 minutes) (in color) **2015025**

TRAUMATIC HAND INJURIES: ASSESSMENT AND EMERGENCY MANAGEMENT, with Martin A. Posner, M.D., hand surgeon and Chairman of the Hand Service, Hospital for Joint Diseases, New York City. An organized approach to the evaluation and care of patients with lacerations, crushing wounds, and other traumatic hand injuries. (16 minutes) (in color) **2021535**

TREATMENT OF ACUTE HEROIN TOXICITY, with David E. Smith, M.D., Director and Founder of the Haight-Ashbury Free Medical Clinic and Assistant Clinical Professor of Toxicology at the University of California in San Francisco; and George R. Gay, M.D., Chief of the Medical Section and Director of the Heroin Clinic with the Haight-Ashbury Clinic and Instructor in Toxicology at the University of California, San Francisco. With growing numbers of heroin abusers in middle-class America, physicians increasingly are going to face the problem of acute heroin overdose — a medical emergency. Two physician-experts put this problem into perspective through patient interviews, describe the "street" treatments that are in use — adding to risk of the complications — and finally present the treatment procedure they employ.
(20 minutes). **2013422**

TREATMENT OF RESPIRATORY FAILURE, with Robert M. Rogers, M.D., Assistant Professor of Medicine, and Director of the Respiratory Intensive Care Unit, Hospital of the University of Pennsylvania. The selection and application of appropriate emergency procedures in treating respiratory failure are fully explored.
(19 minutes) (In color) **2010217**

ENDOCRINOLOGY

ADULT DIABETES: OUTGUESSING THE NEXT 24 HOURS, with Leo P. Krall, M.D., Director, Education Division, and Lecturer in Medicine, Joslin Diabetes Foundation, Inc., and New England Deaconess Hospital, and Lecturer, Harvard Medical School, Boston. Management of diabetes mellitus starts with defining treatment goals and understanding why they are often difficult to obtain. Dr. Krall discusses the general management of different types of patients. This program was produced with the cooperation of the Council on Scientific Assembly of the American Medical Association. (25 minutes) (in color) **0123046**

CAN YOU TREAT OBESITY IN CHILDREN? with Platon J. Collipp, M.D., Chief Pediatrician, Nassau County Medical Center, and Professor of Pediatrics, State University of New York, Stony Brook, Long Island, N. Y. Long-standing obesity, a health hazard in later life, can be stemmed in childhood and adolescence. Dr. Collipp shows how diet, group treatment, challenge and support have successfully removed "60 tons of Long Island baby fat."
(18 minutes) (in color) **0318076**

CELLULAR DISTURBANCES:
A NEW CONCEPT OF OBESITY

Some extremely obese patients may be incapable of losing weight and maintaining the loss because they may have acquired an excessive number of fat cells early in life. This is one of the findings of Jules Hirsch, M.D., Professor and Senior Physician to The Hospital, Rockefeller University, New York. (19 minutes). **0308704**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART I. Alvin F. Goldfarb, M.D., Assistant Professor of Obstetrics and Gynecology, Jefferson Medical College and Hospital, presents "Evaluating the Infertile Couple."
(19 minutes). **0303118**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART II

"The Physio-Anatomic Basis of Fallopian Tube Function." The second program in this continuing series — with Luigi Mastroianni, Jr., M.D., Professor of Obstetrics and Gynecology, University of Pennsylvania School of Medicine.
(17 minutes). **0303319**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART III

"Tubal Factor Treatment." Correcting distortion of the tubal ovarian relationships. With Celso-Ramon Garcia, M.D., Associate Professor of Obstetrics and Gynecology, University of Pennsylvania School of Medicine.
(16 minutes). **0303620**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART IV
"The Cervix in Infertility." Evaluation of the cervical factor in cases of female infertility — with Kamran S. Moghissi, M.D., Associate Professor of Obstetrics and Gynecology, Wayne State University School of Medicine.
(17 minutes) **0304421**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART V
"New Research," a discussion and demonstration of the use of frozen sperm for artificial Insemination; immunologic aspects of infertility; and new laparoscopic techniques.
(18 minutes). **0304622**

CLINICAL MANAGEMENT OF HUMAN REPRODUCTIVE PROBLEMS: PART VI — INDICES OF OVULATION
The tests which are available to determine the absence or occurrence of ovulation are described by Howard Balin, M.D., Chief, Gynecic Research Unit, Pennsylvania Hospital, Philadelphia. (26 minutes). **0304923**

CURABLE HYPERTENSION, with Ralph E. Peterson, M.D., Professor of Medicine and Director, Division of Endocrinology, New York Hospital-Cornell University Medical Center, New York City. Forty million adult Americans have blood pressures higher than 150/90; in half of them, hypertension is secondary to another disease process. Among this latter group, surgery to remove the underlying cause is effective in five per cent, or one million cases. Dr. Peterson shows how to determine if a hypertensive patient is among the curable million. (16 minutes) (in color) **0315270**

CURRENT DIAGNOSTIC AND TREATMENT PROBLEMS IN THYROID DISEASE, with Gerald Burke, M.D., Department of Medicine; Leslie J. DeGroot, M.D., Department of Medicine; Alexander Gottschalk, M.D., Department of Radiology; Edward Paloyan, M.D., Department of Surgery; and Samuel Refetoff, M.D., Department of Medicine, all of the Pritzker School of Medicine of the University of Chicago. In a clinic setting, the panelists discuss diagnostic and therapeutic problems including management of possible thyroid carcinoma, clinical thyrotoxocosis with "normal" lab data, exophthalmos, and congenital goiter. Clinical physiology and related research are emphasized. A Television Clinic of the American College of Physicians, 1973. Please inquire for special rental information.
(60 minutes) (in color) **ACP 2827366**

CURRENT MANAGEMENT OF THE ADULT DIABETIC, with Peter H. Forsham, M.D,, Director of Metabolic Research Unit, University of California School of Medicine, San Francisco.
Dr. Forsham outlines the treatment of adult diabetes. In addition to explaining his use of diet and insulin therapy, he thoroughly examines the oral hypoglycemic agents, including phenformin hydrochloride and the sulfonylureas, explaining how they work, their side-effects, and the most effective regimen. (22 minutes) (in color) **0316673**

CUSHING'S SYNDROME: FORTY YEARS LATER, with Ralph Peterson, M.D., Professor of Medicine and Director, Division of Endocrinology, New York Hospital-Cornell Medical Center, New York City. Today physicians know much more than did Harvey Cushing about the syndrome that bears his name. Dr. Peterson reviews and updates its mechanisms and diagnosis.
(19 minutes) (in color) **0315069**

DIABETIC ACIDOSIS. Practical rules of thumb for the treatment of diabetic acidosis, with Daniel B. Stone, M.D., and Joseph D. Brown, M.D., Department of Internal Medicine, University Hospital, University of Iowa College of Medicine. (29 minutes). **0400403**

THE DIABETIC FOOT: HANDLE WITH CARE, with H. Howard Goldstein, M.D., Senior Physician, The Joslin Clinic, Division of the Joslin Diabetes Foundation, and the New England Deaconess Hospital, Boston. Once problems occur in the diabetic foot, they tend to be progressive. Dr. Goldstein teaches how to examine the feet of the diabetic patient and recommends appropriate preventive care and treatment. This program was produced in cooperation with the Council on Scientific Assembly of the American Medical Association.
(17 minutes) (in color) **0423261**

DIABETIC RETINOPATHY: ATTEMPTS TO HOLD THE IMAGE, with Raymond Pilkerton, M.D., Associate Professor of Ophthalmology and Director, Retina Service, Georgetown University Medical Center, Washington, D.C. The relationship between the duration of diabetes and the stages of diabetic retinopathy. How treatment may temporarily stop the advance of this complication. This presentation was produced in cooperation with the Council on Scientific Assembly of the American Medical Association.
(16 minutes) (in color) **0423160**

INBORN ERRORS OF METABOLISM: TREATMENT

Charles R. Scriver, M.D., of the DeBelle Laboratory for Biochemical Genetics at the Montreal Children's Hospital in Montreal, Canada, places the problems of hereditary disorders in their proper perspective. Dr. Scriver divides his therapeutic discussion into "genetic engineering" and "environmental engineering."

(15 minutes) (in color) **0909605**

INBORN ERRORS OF METABOLISM: MECHANISM AND DIAGNOSIS

Charles R. Scriver, M.D., of the DeBelle Laboratory for Biochemical Genetics at Montreal Children's Hospital in Montreal, P.Q., Canada, describes the current techniques for managing genetic diseases. (13 minutes) (in color) **0909804**

HOW I EVALUATE THE THYROID, with W. Lester Henry, Jr., M.D., Professor and Chairman of the Department of Medicine, Howard University School of Medicine, Washington, D.C. How to examine the thyroid from the anterior position (instead of the more usual posterior) with tips on the diagnosis of thyroid enlargement. Henry emphasizes the physical exam as well as the use of radioactive scans.

(14 minutes) (in color) **0816827**

HYPERCALCEMIA: A DIFFERENTIAL DIAGNOSIS, with John T. Potts, Jr., M.D., Chief of Endocrinology, Massachusetts General Hospital, and Associate Professor of Medicine, Harvard Medical School, Boston, Massachusetts. Hypercalcemia can be a life-threatening situation. Dr. Potts shows how a diagnosis of hypercalcemia can be established, and what action can be taken for the various possible causes.

(16 minutes) (in color) **0813218**

NEWEST DIAGNOSTIC AND THERAPEUTIC APPROACHES TO THYROID DISEASE, with Manfred Blum, M.D., Charles S. Hollander, M.D., Herbert H. Samuels, M.D., and Louis Shenkman, M.D., all of the Department of Medicine, New York University Medical Center School of Medicine, New York City. Recent diagnostic and therapeutic approaches to thyroid disease are examined in the context of clinical case presentations of T3 toxicosis, hypothalamic hypothyroidism, and autonomous adenoma. Topics discussed include the clinical usefulness of radioassay procedures for thyroxine, triiodothyronine, thyrotropin, free thyroxine, the value of thyrotropin-releasing hormone in thyroid diagnosis, and the diagnostic utility of thyroid echography. A Television Clinic of the American College of Physicians, 1974. Please inquire for special rental information.

(60 minutes) (in color) **ACP 2837479**

RECOGNIZING ROLES IN JUVENILE DIABETES, with Donnell D. Etzwiler, M.D., Director, Diabetes Education Center, and Pediatrician, St. Louis Park Medical Center, Minneapolis. A pediatrician gives guidelines for early diagnosis and management of juvenile diabetes and shows which responsibilities of good control should be assigned to physician, health professional, and patient. This presentation was produced with the cooperation of the Council on Scientific Assembly of the American Medical Association.

(16 minutes) (in color) **1822934**

SHORT STATURE IN CHILDREN, with Maurice D. Kogut, M.D., Director, Clinical Research Center, Children's Hospital of Los Angeles, Los Angeles, California.

Three standard growth deviations are defined, and those conditions which are responsible for growth retardation — where no obvious disease is present — are described by Dr. Kogut.

(17 minutes) (in color) **1911705**

SOME ORGANIC CAUSES OF CHILDHOOD OBESITY, with Platon J. Collipp, M.D., Chief of Pediatrics, Nassau County Medical Center, and Professor of Pediatrics, State University of New York, Stony Brook, Long Island, N. Y. Don't discount "glandular" causes of obesity without a long, hard look. Dr. Collipp presents patients with the more common of these rare diseases associated with overweight.

(15 minutes) (in color) **1518209**

THE DIABETES PUZZLE: A PRACTICAL APPROACH, with Rachmiel Levine, M.D., Professor and Chairman of the Department of Medicine at New York Medical College in New York City.

Most of the three million cases of diabetes in the U.S. are considered mild from the metabolic viewpoint. However, because of the link to many forms of cardiovascular, renal and other diseases it becomes important to discover and treat the mild forms. Dr. Levine describes the classical symptoms of diabetes and when to suspect the disease in the absence of the typical signs. He classifies therapy, relates diabetes to other diseases and describes its pathogenesis and inherited factors. (17 minutes). **0410805**

THE DIABETIC IN COMA/BRITTLE DIABETES/ THE YOUNG DIABETIC, with Rachmiel Levine, M.D., Professor and Chairman of the Department of Medicine at New York Medical College in New York City.

Coma may occur in a person with diabetes for the same reasons as it would occur in the nondiabetic. For that reason it is important to differentiate between the two comas. Dr. Levine describes ketoacidosis, hyperglycemic coma, lactacidosis, hypoglycemia. Dr. Levine also discusses "brittle" diabetes and the prognosis of childhood diabetes. (20 minutes). **0410904**

TREATMENT OF INSULIN-DEFICIENT DIABETES, with Peter H. Forsham, M.D., Director, Metabolic Research Unit, University of California School of Medicine, San Francisco. Insulin has been available for 50 years, but its use in diabetic therapy can be improved. To achieve better results, says Dr. Forsham, physicians must simulate as closely as possible the release of insulin by normal persons. (21 minutes) (in color) **2016427**

FAMILY PRACTICE

AFTER THE SPONTANEOUS ABORTION: COUNSELING BY THE FAMILY PHYSICIAN, with William C. Rigsby, M.D., Assistant Professor, Department of Obstetrics and Gynecology, and Adolph Hass, M.D., Clinical Associate Professor, Department of Psychiatry, Ohio State University School of Medicine. Using an actual case of spontaneous abortion in a much wanted pregnancy, Drs. Rigsby and Hass — and the patient in question — delineate areas in which the physician can support and reassure his patient in the time of crisis.
(19 minutes) (in color) **0116129**

FAMILY PRACTICE: A NEW SPECIALTY FOR THE GENERALIST, with Eugene S. Farley, M.D., M.P.H., Director of Family Medicine Program, Highland Hospital, University of Rochester, Rochester, N.Y. The formalization of Family Practice as a medical specialty raises questions: What is this specialist's role in the community? What training does he require? Answering these questions and others, Dr. Farley—a pioneer in this growing new specialty—shows us a modern family practice and describes the type of paramedical assistance required for its efficient operation. (20 minutes) (in color) **0614413**

FAMILY PRACTICE AND COMMUNITY HEALTH, with Eugene S. Farley, Jr., M.D., M.P.H, Director of Family Medicine Program, Highland Hospital, University of Rochester, Rochester, N. Y.
Family Practice is distinguished from general practice by its emphasis on prevention, a bridge with the community and, often, group structure. Dr. Farley takes us into his family practice to show how it serves the community as well as the changing needs of the practice's staff. (16 minutes) (in color). **0614514**

THE GENERAL PRACTITIONER AND COMMUNITY RESOURCES AVAILABLE FOR THE DEVELOPMENTALLY DELAYED, with William Gibson, M.D., Director, the Ohio State University Hirschel W. Nisonger Center for Mental Retardation, and Associate Professor of Physical Medicine at the Ohio State University College of Medicine; Cary W. Perkins, with the Ohio Association for Retarded Children, Inc.; and Donald Cavin, Ed.D., Chief of Special Education at the Nisonger Center in Columbus, Ohio. The general practitioner can play an invaluable role in guiding families of the mentally retarded and the developmentally disabled to community resources and treatment centers. This telecast acquaints the physician with recent federal legislation providing for new resources, and how he can learn of their availability in his community.
(16 minutes) (in color) **0714015**

GASTROENTEROLOGY

ABDOMINAL ARTERIOGRAPHY, with Robin Caird Watson, M.D., Chairman of the Department of Diagnostic Radiology, Memorial Sloan-Kettering Cancer Center, and Associate Professor of Radiology, Cornell University Medical Center, New York City. When to order an arteriogram and what it can tell you. Dr. Watson demonstrates the technique he uses to investigate a patient with a mass in the area of the pancreas, vague upper GI symptoms and equivocal X-rays.
(20 minutes) (in color) **0118435**

ACUTE REGIONAL ENTERITIS: A CLINICAL PATHOLOGICAL CONFERENCE, with Floyd M. Beman, M.D., Professor of Medicine; J. David Dunbar, M. D., Assistant Professor of Radiology; Dante G. Scarpelli, M.D., Professor of Pathology; and William Pace, M.D., Assistant Dean of the College of Medicine; all from the Ohio State University College of Medicine in Columbus, Ohio.
Four physician-educators critically explore a case of severe, progressive regional enteritis.
(23 minutes). **0110703**

ADVANCES IN GASTROSCOPY
The value of gastroscopy is discussed by Charles S. Winans, M.D., Instructor in the Department of Medicine at the University of Chicago Pritzker School of Medicine, and demonstrated by Seibi Kobayashi, M.D., Research Fellow at the same institution. (19 minutes) (in color) **0108905**

APPLICATION OF DIAGNOSTIC CYTOLOGIC TECHNIQUES TO GASTROINTESTINAL ENDOSCOPY AND LIVER BIOPSY, with Sidney J. Winawer, M.D., Director, Diagnostic Gastrointestinal Laboratory, Memorial Sloan-Kettering Cancer Center and Clinical Associate Professor of Medicine, Cornell University Medical College; and and Paul Sherlock, M.D., Chief, Gastroenterology Service, Memorial Sloan-Kettering Cancer Center and Associate Professor of Medicine, Cornell University Medical College. Directed brush cytology and directed pulsatile lavage cytology through the upper Gastrointestinal Panendoscope and Colonoscope are demonstrated. Pulsatile lavage through the standard sigmoidoscope for rectocolonic cytology is also shown. This presentation was made at the 1973 annual meeting of the American Society for Gastrointestinal Endoscopy. Please inquire for special rental information.
(41 minutes) (in color) **ASGE 2800076**

APPROACH TO GASTROINTESTINAL PAIN: CASES I & II, with Howard M. Spiro, M.D., Professor of Medicine, Yale University School of Medicine, New Haven, Connecticut. Dr. Spiro is confronted by two "patients"—actors who have been carefully coached by another physician in symptoms and histories of particular gastrointestinal disorders. The "patients" do not know their diagnoses, nor does Dr. Spiro who arrives at his conculsions on camera. An opportunity to match diagnostic skills with a leading gastroenterologist. (18 minutes) (in color) **0115024**

APPROACH TO GASTROINTESTINAL PAIN: CASES III & IV, with Howard M. Spiro, M.D., Professor of Medicine, Yale University School of Medicine, New Haven, Connecticut. Dr. Spiro encounters "patients" unknown to him who have been carefully coached in symptoms of specific G.I. disorders. You can match diagnosis with him as he reaches his conclusions on camera.
(14 minutes) (in color) **0115125**

APPROACH TO GASTROINTESTINAL PAIN: CASES V, VI & VII, with Howard M. Spiro, M.D., Chief of Gastroenterology, Yale-New Haven Hospital, and Professor of Medicine, Yale University School of Medicine, New Haven, Connecticut. Three carefully coached actors appear as patients seeking diagnosis. In this program Dr. Spiro deals with abdominal emergencies. The viewer is invited to exercise his diagnostic skills alongside a leading gastroenterologist.
(19 minutes) (in color) **0115226**

APPROACH TO GASTROINTESTINAL PAIN: CASES VIII & IX, with Howard M. Spiro, M.D., Professor of Medicine, Yale University School of Medicine, and Chief of Gastroenterology, Yale-New Haven Hospital, New Haven, Conn. Two "patient" actors present with radiating pain. Dr. Spiro listens to their stories and elucidates: one rare but not difficult to diagnose, the other fairly common but not easy. Compare your own findings. (14 minutes) (in color) **0115327**

APPROACH TO UPPER GI BLEEDING, with Robert M. Lowman, M.D., Professor and Acting Chairman, Department of Radiology; and Howard M. Spiro, M.D., Chief of Gastroenterology Division, Department of Medicine, Yale University School of Medicine, New Haven, Conn. Endoscopy . . . selective arteriography . . . contrast media studies . . . gastric aspiration. Which of these aids should you employ and in what order for a patient with upper GI bleeding? Drs. Spiro and Lowman review the relevant history of two patients and then show the results of several examinations including arteriography and endoscopy used for the diagnoses.
(19 minutes) (in color) **0118234**

CROHN'S DISEASE: ONCE YOU HAVE THE DIAGNOSIS, with Henry D. Janowitz, M.D., Clinical Professor of Medicine and Head of the Division of Gastroenterology at Mt. Sinai Hospital and School of Medicine, New York City. Surgery or medicine . . . the critical decision in treating patients with Crohn's disease. Doctor Janowitz offers a rational approach to selecting the appropriate therapy. (16 minutes) (in color) **0322082**

DIAGNOSING DYSPHAGIA, with Robert M. Lowman, M.D., Department of Radiology, and Howard M. Spiro, M.D., Chief of the Gastroenterology Division, Department of Medicine, Yale University School of Medicine, New Haven, Conn. Drs. Lowman and Spiro outline the order in which to use barium swallow X-rays, esophagoscopy, bouginage and motility studies to differentiate psychogenic, obstructional and physiological dysphagia. They illustrate their discussion with barium swallow X-rays and films of esophagoscopy.
(20 minutes) (in color) **0417741**

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DIAGNOSIS OF OCCULT INTRA-ABDOMINAL NEOPLASMS, with Herbert B. Greenlee, M.D., Department of Surgery, Loyola University of Chicago, Stritch School of Medicine, Maywood, Ill., and Veterans Administration Hospital, Hines, Ill.; Erwin M. Kammerling, M.D., Department of Medicine, University of Health Sciences, The Chicago Medical School, and Louis Weiss Memorial Hospital, Chicago; Sumner C. Kraft, M.D., Department of Medicine, University of Chicago; and Armand Littman, M.D., Department of Medicine, University of Illinois, College of Medicine, Chicago, and Veterans Administration Hospital, Hines, Ill. The panel presents cases illustrating some of the difficulties and solutions involved in tracking down a diagnosis of intra-abdominal neoplasms. Emphasis is given to the tests (some old, some new, some under-utilized) which are helpful in deciding to perform exploratory laparotomy. A Television Clinic of the American College of Physicians, 1973. Please inquire for special rental information.
(60 minutes) (in color) **ACP 2847368**

DIAGNOSING PEPTIC ESOPHAGITIS, with Robert M. Lowman, M.D., Professor and Acting Chairman, Department of Radiology, and Howard M. Spiro, M.D., Chief of the Gastroenterology Division, Department of Medicine, Yale University School of Medicine, New Haven, Conn. Drs. Lowman and Spiro demonstrate how to determine the cause of heartburn and to reveal related serious diseases using barium swallow X-rays, endoscopy and the Bernstein acid perfusion test.
(16 minutes) (in color) **0417842**

DIAGNOSING THE INFLAMED BOWEL, with Marvin M. Schuster, M.D., Director of Gastroenterology, Baltimore City Hospitals, Associate Professor of Medicine and Assistant Professor of Psychiatry, Johns Hopkins University School of Medicine, Baltimore. When the signs point to inflammatory bowel disease, here is the four-stage workup recommended to differentiate ulcerative colitis from granulomatous disease of the colon. (17 minutes) (in color) **0419449**

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THE DIAGNOSTIC CHALLENGE OF CHEST PAIN, with moderator Alfred Soffer, M.D., Professor of Cardiology at the University of Health Sciences—Chicago Medical School, Chicago, and Editor-in-Chief of *Chest*, the Journal of the American College of Chest Physicians. Participants are: R. D. Henderson, M.B., F.R.C.S. (C)., Staff Surgeon, Toronto General Hospital and Assistant Professor of the Department of Surgery at the University of Toronto, Canada; Barry William Levine, M.D., Assistant Professor of Medicine, Harvard Medical School, and Chief, Outpatient Pulmonary Services, Massachusetts General Hospital, Boston; Linda D. Lewis, M.D., Assistant Professor of Neurology, College of Physicians and Surgeons of Columbia University, and Chief of the Neurology Clinic, Neurological Institute of Columbia Presbyterian Medical Center, New York City, Morton E. Tavel, M.D., Associate Professor of Medicine at the Indiana University School of Medicine, Indianapolis.

Pinpointing the cause of your patient's chest pain poses an urgent diagnostic challenge. Join Dr. Soffer and four specialists on these clinical grand rounds as they reach a diagnosis on five patients. (32 minutes) (in color) **0423362**

DIFFERENTIAL DIAGNOSIS OF DISEASES OF THE ILEUM AND COLON. The dilemma of differentiating between regional ileitis, ulcerative colitis and granulomatous colitis may be no more than a confusion of terms, yet may lead to improper management of these diseases. In this presentation, Burrill B. Crohn, M.D., Professor Emeritus, Department of Gastroenterology, Mount Sinai Hospital and Medical Center, New York, illustrates his own diagnostic techniques and methods of handling these clinical entities.
(16 minutes). **0407915**

Many NCME programs have self-assessment quizzes, one copy of which will accompany each videocassette. You may duplicate the quiz as your needs require. Upon completion, return the quizzes to NCME, and we will maintain a record of participation (not the score) for each physician. These records are keyed to the physician's Social Security numbers: please take care that the numbers are clearly written. Physicians may request their NCME activity records at any time.

ESOPHAGEAL DISORDERS AND CHEST PAIN—(EVALUATION AND MANAGEMENT OF ESOPHAGEAL REFLUX), with Thomas R. Hendrix, M.D., Department of Medicine; Theodore M. Bayless, M.D., Department of Medicine, Martin W. Donner, M.D., Department of Radiology; Francis D. Milligan, M.D., Department of Medicine; and David B. Skinner, M.D., Department of Surgery: all of the Johns Hopkins University School of Medicine.

Clinical, radiologic, and special procedures used in evaluation of gastroesophageal reflux are presented, including acid perfusion, esophagoscopy, cine radiology, esophageal motility, and pH probe measurement. The relation of reflux to esophagitis and to reflux symptoms are defined. Among topics discussed: medical therapy for reflux; surgical options; relation of hiatal hernia to reflux; and management of complications of reflux, stricture, and esophageal ulcer.

A Television Hospital Clinic of the American College of Physicians, 1972. Please inquire for special rental information.

(59 minutes) (in color) **ACP 2817243**

FROM TOP TO BOTTOM: A FIBEROPTIC VIEW OF THE BODY, with Howard J. Eddy, M.D., Attending Surgeon, Community Hospital, Doctors Hospital and St. John's Hospital, Long Island, New York; and Richard S. McCray, M.D., Associate Director of the Clinical Gastrointestinal Unit, St. Luke's Hospital, and Associate in Medicine, Columbia University College of Physicians and Surgeons, New York City.

Fiberoptic instruments now available permit the clinician to inspect, biopsy, and even treat lesions in the esophagus, stomach, duodenum and colon. Dr. McCray, a gastroenterologist, takes viewers on a trip through the upper GI tract. Dr. Eddy, a colon and rectal surgeon, shows lesions of the lower tract and removes a benign polyp using the instrument.

(30 minutes) (in color) **0616715**

GASTROINTESTINAL CYTOLOGY — A VALUABLE DIAGNOSTIC PROCEDURE: PART I

"Application and Results." Confirming a diagnosis without surgery through the use of Papanicolaou staining of cells from the gastrointestinal tract — with Charles Norland, M.D., Assistant Professor of Medicine, University of Chicago School of Medicine. (16 minutes). **0703303**

GASTROINTESTINAL CYTOLOGY: PART II

"Techniques and Methods of Interpretation." Demonstration of the techniques of tubular intubation, stomach washing, and slide staining utilized in this diagnostic procedure — with Charles Norland, M.D., Assistant Professor of Medicine, University of Chicago School of Medicine. (17 minutes). **0703404**

HANDLING THE GALLSTONE PATIENT, with J. L. Thistle, M.D., Gastroenterologist, Department of Gastroenterology, Mayo Clinic, Rochester, Minnesota. Deciding which of your patients will benefit from surgery is not always easy. Dr. Thistle looks at two patients and points out the important elements in making that decision.

(15 minutes) (in color) **0823043**

HOW I DO SIGMOIDOSCOPY, with Ralph E. L. Hertz, M.D., Associate Attending Surgeon, Department of Colon and Rectal Surgery, Memorial Hospital for Cancer and Allied Diseases, New York City. Dr. Hertz demonstrates the steps involved in inspection of the perianal tissues, sigmoidoscopy and anoscopy, and provides tips for making the procedure easier for you and more comfortable for the patient.

(21 minutes) (in color) **0817329**

IMMUNOLOGIC TESTS FOR VIRAL HEPATITIS, with Carroll M. Leevy, M.D., Professor of Medicine and Director, Division of Hepatology and Nutrition, College of Medicine and Dentistry of New Jersey, Newark. The tests for Hepatitis A and B and what they can do for you in practice.

(16 minutes) (in color) **0923132**

JAUNDICE: IS IT MEDICAL OR SURGICAL?, with Carroll M. Leevy, M.D., Professor of Medicine and Director, Division of Hepatology and Nutrition, the College of Medicine and Dentistry of New Jersey, Newark. An up-to-date look at this diagnostic puzzle.

(15 minutes) (in color) **1023202**

MALABSORPTION SYNDROME

Victor W. Groisser, M.D., Clinical Professor of Medicine, New Jersey College of Medicine, and Director of Gastroenterology, Mountainside Hospital, Montclair, New Jersey, comments on the frequency of malabsorption syndrome, on the possibility of misdiagnosis and the need for clearing up the confusion surrounding malabsorption. (19 minutes). **1309602**

PROBLEMS AND PITFALLS IN PSYCHOSOMATIC MEDICINE: PEPTIC ULCER, with Roy R. Grinker, M.D., Associate Professor of Psychiatry and Neurology; and F. Theodore Reid, M.D., Associate Professor of Psychiatry and Neurology, both from the Michael Reese Hospital in Chicago, Illinois.

Most physicians are confronted with patients suffering from psychosomatic illnesses. Many of these patients are treated as "second class citizens," largely because of the frustrations in treating them. This program shows the nonpsychiatric physician the way to handle psychosomatic conditions by citing a peptic ulcer patient as an example. Particular attention is given to differentiating illnesses with etiologic origins and emotional disturbances.

(16 minutes) (in color) **1612522**

RADIOLOGIC DIAGNOSIS OF THE ACUTE ABDOMEN

Several radiological techniques are available in diagnosing patients presenting with acute abdominal pain. Robert D. Moseley, Jr., M.D., Professor and Chairman of the Department of Radiology, University of Chicago Pritzker School of Medicine, summarizes these techniques and considers their importance in clinical findings.

(15 minutes) (in color) **1808804**

RECTAL BLEEDING: FINDING THE CAUSE, with Robert M. Lowman, M.D., Professor and Acting Chairman, Department of Radiology, and Howard M. Spiro, M.D., Chief of Gastroenterology Division, Department of Medicine, Yale University School of Medicine, New Haven, Conn. Drs. Lowman and Spiro review the procedures involved in diagnosing minimal, moderate and massive rectal bleeding and outline the order in which sigmoidoscopy, colonoscopy, barium enema and selective arteriography should be employed. (18 minutes) (in color) **1817927**

A TELEVISION DISCUSSION BY THE CLINICAL PATHOLOGICAL FACULTY. Featured are Floyd M. Beman, M.D., Professor of Medicine, J. David Dunbar, M.D., Assistant Professor of Pathology, Dante G. Scarpelli, M.D., Professor of Pathology, and William Pace, Assistant Dean, Ohio State University College of Medicine, who discuss a case of severe, progressive regional enteritis, the causes of death, and the steps that might have been taken to prolong the patient's life.

(58 minutes). **2800006**

TRAUMA TO THE LIVER: OPERATIVE APPROACH AND SURGICAL PROCEDURE, with Gordon F. Madding, M.D., Associate Clinical Professor of Surgery, University of California School of Medicine; and Associate in Surgery, Stanford University School of Medicine; and Paul A. Kennedy, M.D., Assistant Clinical Professor of Surgery, Stanford University School of Medicine. Anatomically and surgically this large, vital organ is not widely understood. Drs. Madding and Kennedy demonstrate a few basic principles which can make emergency treatment of damaged liver safer and surer. (19 minutes) (in color) **2015025**

TRAUMA TO THE LIVER: PRIMARY CARE AND DIAGNOSIS, with Gordon F. Madding, M.D., Associated Clinical Professor of Surgery, University of California School of Medicine, and Associate in Surgery, Stanford University School of Medicine; and Paul A. Kennedy, M.D., Assistant Clinical Professor of Surgery, Stanford University School of Medicine. In the opinion of Drs. Madding and Kennedy, there are ten specific steps that should be taken when a patient presents with traumatic injury to the abdomen. These measures lead most rapidly to effective therapeutic actions.

(18 minutes) (in color) **2014924**

GENETICS

ABNORMAL SEX DIFFERENTIATION, with Maurice D. Kogut, M.D., Director, Clinical Research Center; and Jordan J. Weitzman, M.D., pediatric surgeon, both of Children's Hospital of Los Angeles.

The factors of determining sexual ambiguities in the newborn are clearly demonstrated in this telecast. Emphasis is also placed on early treatment and establishing an unambiguous sex of rearing. (17 minutes) (in color) **0111601**

AN OUTLINE OF CYTOGENETICS

A survey of the field of human cytogenetics, covering the development of the techniques which make chromosome studies practical — with LCDR Thomas R. Birdwell, MC, U.S.N., Head, Cytogenetics Division, Department of Pathology, U.S. Naval Hospital, San Diego, Calif.

(16 minutes). **1503403**

CHROMOSOMES BY MAIL

As an alternative to the time-consuming and costly practice of transporting the patient to the testing center, there is now a practical method for shipping blood specimens over long distances for the leukocyte chromosome culture and chromosome analysis. LCDR Thomas R. Birdwell, MC, U.S.N., Head, Cytogenetics Division, U.S. Naval Hospital, San Diego, Calif., demonstrates the technique.

(11 minutes). **0303810**

CYSTIC FIBROSIS: DIAGNOSIS AND MANAGEMENT, with Paul R. Patterson, M.D., Director, Cystic Fibrosis Unit, Albany Medical Center, Albany Medical College, Union University, New York.

Attention is drawn to cystic fibrosis as a model genetic disease. Dr. Patterson describes, with many examples, C/Fs mode of inheritance, incidence, symptoms, differential diagnosis, detection of carrier state, current status of manageability and prospects for palliative or corrective therapy. (19 minutes) (in color) **0310961**

DIAGNOSIS AND MANAGEMENT OF HUNTINGTON'S CHOREA, with Charles Markham, M.D., Professor of Neurology, University of California, Los Angeles School of Medicine, and Milton Wexler, Ph.D., President, California Chapter of The Committee to Combat Huntington's Disease. Huntington's Chorea — an invariably fatal disorder —is transmitted by a dominant gene; thus the carriers' children have a 50 percent chance of inheriting it. There are thought to be more than 100,000 people afflicted with Huntington's Chorea in the U.S. and Canada — most of them undiagnosed or mis-diagnosed. Unless physicians can detect the disease in these unknown thousands and effectively counsel them, a genetic time-bomb is being fused against future generations. (17 minutes) (in color) **0415331**

EPIDEMIC GENETIC DISEASE, with Claude Laberge, M.D., of the Human Genetics Laboratory, Universite Laval, Quebec.

Tyrosinemia is an hereditary disease which can be traced back to a French couple who migrated to North America more than 300 years ago. One in 24 to one in 41 in a particular region in Quebec are carriers of the disease trait, and this is spreading. (No trace of the disease has been recorded in the United States.) Dr. Laberge charts the diagnosis and treatment of this disease to describe the dynamics of genetic diseases, how the diseases are approached today and how they may be overcome in the future. (16 minutes) (in color) **0512310**

GENETIC COUNSELING FOR CHROMOSOME ABNORMALITIES: AUTOSOMAL CHROMOSOMES, with Frederick Hecht, M.D., Associate Professor of Medical Genetics and Pediatrics, Crippled Children's Division, University of Oregon Medical Center, Portland, Oregon.

The parents of children with non-sexual chromosomal abnormalities are interviewed by several people who are involved in training physicians in genetic counseling at the University of Oregon. This program is of particular value to general physicians.
(13 minutes) (in color) **0712706**

GENETIC COUNSELING FOR CHROMOSOME ABNORMALITIES: SEX CHROMOSOMES, with Frederick Hecht, M.D., Associate Professor of Medical Genetics and Pediatrics, Crippled Children's Division, University of Oregon Medical Center, Portland, Oregon.

The general physician learns eight characteristics of effective genetic counseling from this telecast. Particular attention is given to several different sex chromosomal abnormalities.
(17 minutes) (in color) **0712605**

GENETIC MEDICINE: A MEDICAL FACILITY OF THE FUTURE, with Claude Laberge, M.D., Human Genetics Laboratory, Faculte de Medecine, Universite Laval, Quebec.

Dr. Laberge shows the steps that are now being taken to use the advances in molecular biology and biochemical genetics for curing human hereditary diseases. The advances include determining the structure of genetic material, understanding protein synthesis and deciphering the genetic code. Dr. Laberge demonstrates how this information will lead to a new branch of medicine; and how it will go far beyond the present use of merely counseling patients. (16 minutes) (in color) **0712407**

GROWTH AND DIFFERENTIATION OF HUMAN LYMPHOCYTES: PART I
"Nucleic Acid Synthesis." Arnold D. Rubin, M.D., Associate in Medicine, Mount Sinai School of Medicine, demonstrates the techniques for extracting nucleic acid from lymphocytes, focusing on RNA metabolism. (19 minutes). **0703912**

GROWTH AND DIFFERENTIATION OF HUMAN LYMPHOCYTES: PART II, ABNORMAL NUCLEIC ACID SYNTHESIS IN LEUKEMIA
Arnold D. Rubin, M.D., Associate in Medicine, Mount Sinai School of Medicine, New York, conducts an investigation of abnormal growth patterns of lymphocytes taken from patients with lymphoproliferative disorders.
(20 minutes). **0704713**

HUMAN CYTOGENETICS
"Indications for Cytogenetic Testing" — LCDR Thomas R. Birdwell, MC, U.S.N., Head, Cytogenetics Division, U.S. Naval Hospital, San Diego, Calif., presents some of the indications for primary amenorrhea, investigation of female and male sterility, differential diagnosis of congenital defects in the newborn, differential diagnosis of chronic myelocytic leukemia, investigation of intersex, family counseling, and genetic prognosis. (14 minutes). **0803707**

INDENTIFICATION AND GROUPING OF CHROMOSOMES
LCDR Thomas R. Birdwell, MC, U.S.N., Head, Cytogenetics Division, Department of Pathology, U.S. Naval Hospital, San Diego, Calif., discusses the features of the model chromosome and then demonstrates the technique of identifying and grouping chromosomes in karyotypes.
(16 minutes). **0903501**

INBORN ERRORS OF METABOLISM: MECHANISM AND DIAGNOSIS
Charles R. Scriver, M.D., of the DeBelle Laboratory for Biochemical Genetics at Montreal Children's Hospital in Montreal, P.Q., Canada, describes the current techniques for managing genetic diseases. (13 minutes) (in color) **0909804**

INBORN ERRORS OF METABOLISM: TREATMENT
Charles R. Scriver, M.D., of the DeBelle Laboratory for Biochemical Genetics at the Montreal Children's Hospital in Montreal, Canada, places the problems of hereditary disorders in their proper perspective. Dr. Scriver divides his therapeutic discussion into "genetic engineering" and "environmental engineering."
(15 minutes) (in color) **0909605**

NEW GENETIC COUNSELING FOR THE 70s, with Carlo Valenti, M.D., Associate Professor, Department of Obstetrics and Gynecology, Downstate Medical Center in Brooklyn, New York.

New uses of amniocentesis give the physician greater opportunities for genetic counseling. Dr. Valenti shows how the procedure can be used to define chromosomal aberrations, effects of drugs — specifically LSD — and how it can be employed to identify sex and define fetal maturity. (17 minutes) (in color) **1410806**

NEW TECHNIQUES IN AMNIOCENTESIS, with Carlo Valenti, M.D., Associate Professor, Department of Obstetrics and Gynecology, State University of New York, Downstate Medical Center in Brooklyn, New York.

Removing amniotic fluid from a pregnant woman to determine Rh abnormalities is one of the major advances in obstetrics and gynecology during the past 10 years. Amniocentesis is now successfully used at early stages of pregnancy to predict other fetal anomalies. The procedure, with sonograms and instruments, is illustrated in detail. (16 minutes) (in color) **1410728**

OBTAINING AND CULTURING LEUKOCYTES FOR CHROMOSOME ANALYSIS

Demonstrating a simple method of obtaining chromosomes for study and analysis — with LCDR Thomas R. Birdwell, MC, U.S.N., Head, Cytogenetics Division, Department of Pathology, U.S. Naval Hospital, San Diego, Calif.

(15 minutes). **1503402**

PRENATAL DIAGNOSIS OF HEREDITARY DISORDERS, with Carlo Valenti, M.D., Professor of Obstetrics and Gynecology, State University of New York, Downstate Medical Center, Brooklyn, New York. A practical look at the indications for amniocentesis. Dr. Valenti demonstrates the procedure, which many primary care physicians are performing themselves.

(21 minutes) (in color) **1617942**

SICKLE-CELL ANEMIA: MANAGEMENT, with Roland B. Scott, M.D., Professor and Head of the Department of Pediatrics, Howard University, and Chief Pediatrician at Freedmen's Hospital in Washington, D.C.

There is no curative treatment for sickle-cell anemia, according to Dr. Scott. However, early diagnosis of the disease, which afflicts more than 50,000 black Americans, can ameliorate the most disturbing symptoms. Dr. Scott describes the therapeutic program he follows to enhance survival until the patient reaches puberty — when the natural course of the disease process appears to become attenuated.

(14 minutes) (in color) **1911506**

SICKLE-CELL ANEMIA: SUSPICION AND DIAGNOSIS IN INFANTS AND CHILDREN, with Roland B. Scott, M.D., Professor and Head of the Department of Pediatrics, Howard University, and Chief Pediatrician at Freemen' s Hospital in Washington, D.C. Also V. Bushan Bhardwaj, M.D., Assistant Professor of Pediatrics, Howard University, and Pediatric Hematologist, Freedmen's Hospital.

Sickle-cell anemia afflicts more than 50,000 Americans of African descent. Perhaps another two million Americans carry the trait.

Until recently, it was believed that little could be done for the disease. Now relief from the symptoms and a prolonging of life are possible. This telecast features the characteristics of the disease, and the laboratory procedure followed to establish a conclusive diagnosis.

(20 minutes) (in color) **1911407**

SPHINGOLIPIDOSIS: GENETICS

The increasing incidence of sphingolipid disease, such as Tay-Sachs, Gaucher's, and Niemann-Pick, is examined genetically by Stanley M. Aronson, M.D., Professor of Pathology, State University of New York Downstate Medical Center, and Attending Neuropathologist, Isaac Albert Research Institute, Jewish Chronic Disease Hospital, Brooklyn.

(15 minutes). **1904914**

SPHINGOLIPIDOSIS — PART I — BIOCHEMICAL ASPECTS. The chemical compositions of gangliosides, sphingomyelins, sulfatides, glycolipids, and cerebrosides, as they are found in the various sphingolipid diseases, are analyzed by Abraham Saifer, Ph.D., Chief of the Biochemistry Department, Isaac Albert Research Institute of the Jewish Chronic Disease Hospital, Brooklyn.

(21 minutes). **1905015**

SPHINGOLIPIDOSIS — PART II — PATHOLOGY. Several pathologic manifestations — such as amaurotic family idiocy (Tay-Sachs disease), hepatosplenomegaly (Niemann-Pick disease), and others — grouped under the general category of sphingolipidosis, are examined and defined by Bruno W. Volk, M.D., Director of the Isaac Albert Research Institute, of the Jewish Chronic Disease Hospital, and Clinical Professor of Pathology, State University of New York Downstate Medical Center, Brooklyn. (21 minutes). **1905016**

SPHINGOLIPIDOSIS — PART III — CLINICAL ASPECTS. The specific physiologic manifestations of the Tay-Sachs and Niemann-Pick diseases and amaurotic idiocy—such as cherry red macula, clonus, severe contractions, the "frog" position of the legs, and lack of macrocephaly — are demonstrated with young patients by Larry Schneck, M.D., of the Albert Isaac Research Institute of the Jewish Chronic Disease Hospital, Downstate Medical Center, Brooklyn, New York. (13 minutes). **1905217**

WHO SPEAKS FOR THE BABY? A baby is born mongoloid, with a defective heart and duodenal atresia. The parents, with three other children at home, ask that lifesaving intestinal surgery not be performed. The pediatrician, trained to preserve life, seeks a court order to operate. Is such a decision within the physician's province? Should this child live or die? A presentation by physicians and medical students of points of view on infant euthanasia.
(20 minutes) (In color) **SAMA 2810352**

GERIATRICS

AGING AS A FACTOR IN DIFFERENTIAL DIAGNOSIS, with Isadore Rossman, M.D., Medical Director, Home Care Department, Montefiore Hospital, and Associate Professor of Community Medicine, Albert Einstein College of Medicine, in New York City. Normal changes in the geriatric patient modify usual considerations in doing differential diagnosis. The changes that produce the involutional phenomena are indicated, and their effect on a true diagnosis is emphasized. (14 minutes). **0110306**

ARTHRITIS: SURGICAL INDICATIONS, PART I — EARLY, PROPHYLACTIC

John L. Sbarbaro, Jr., M.D., Assistant Professor of Orthopedic Surgery at the University of Pennsylvania School of Medicine in Philadelphia, Pa., indicates that if suppressive drug and physical therapy cannot control advancing arthritis, extirpation might. (19 minutes) (In color) **0108416**

ARTHRITIS: SURGICAL INDICATIONS, PART II — LATE, RECONSTRUCTIVE

The development of non-reactive metals, improved design of molds and prosthesis and improved surgical techniques have caused a recent resurgence of interest in the surgical reconstruction of deformed arthritic joints. John L. Sbarbaro, Jr., M.D., Assistant Professor of Orthopedic Surgery, University of Pennsylvania School of Medicine, demonstrate some of the new techniques and shows the results of surgical repair.
(16 minutes) (In color) **0108517**

DEALING WITH THE TERMINALLY ILL PATIENT

Elizabeth Kubler-Ross, M.D., Assistant Professor of Psychiatry, University of Chicago, demonstrates the stages a patient goes through once he becomes aware he is dying. Dr. Ross also discusses the reaction of people around the patient.
(16 minutes) **0409401**

LEARNING TO LIVE WITH DYING. Dying is perhaps the most difficult and least understood phase of life. Terminall y ill patients and their families turn to their physicians for support and guidance that goes beyond clinical care.

Medical students Sam Cullison and Mike O'Neal join The Reverend Barry Wood, M.D. and William Fischer, M.D., both of Roosevelt Hospital, New York City, and Robert Neale, Th.D., of Union Theological Seminary, New York City, to discuss the management of terminally ill patients and their families.
(39 minutes) (In color) **SAMA 2810656**

MANAGEMENT OF THE TERMINALLY ILL: THE FAMILY

Elizabeth Kubler-Ross, M.D., Assistant Professor of Psychiatry at the University of Chicago, offers practical help to physicians in dealing with the dying patient and his family.
(16 minutes). **1309708**

PRACTICAL MANAGEMENT OF DISABILITY IN THE AGED. Geriatric patients with neuromuscular and orthopedic disabilities demonstrate that, when properly motivated and emotionally supported, they can adapt to their physical disabilities as mechanical obstacles to be overcome. Leo Dobrin, M.D., Clinical Instructor, Rehabilitative Medicine, New York University Medical Center, and Director of Physical Medicine and Rehabilitation, The Jewish Home and Hospital for Aged, and Frederic Zeman, M.D., Chief of Medical Services at The Jewish Home and Hospital for Aged, offer clinical commentary.
(18 minutes). **1605918**

TOTAL HIP REPLACEMENT, with John J. Gartland, M.D., James Edwards Professor of Orthopaedic Surgery, Jefferson Medical College of Thomas Jefferson University, Philadelphia, Pennsylvania. Since the mid-sixties thousands of American men and women have obtained relief from crippling hip disease through the implantation of total hip arthroplasties. Dr. Gartland employs the technique advanced by Charnley and Muller to replace the right hip of a middle-aged man who had his left hip similarly replaced five months previously.
(19 minutes) (In color) **2015126**

HEMATOLOGY

ACUTE MYELOGENOUS LEUKEMIA: THE DIAGNOSIS, with Monroe Dowling, Jr., M.D., Assistant Attending Physician, Hematology Service, Memorial Sloan-Kettering Cancer Center, New York City. Tips for making an earlier diagnosis in patients suspected of having acute leukemia.
(13 minutes) (In color) **0121941**

ACUTE MYELOGENOUS LEUKEMIA: THE TREATMENT, with Monroe Dowling, Jr., M.D., Assistant Attending Physician, Hematology Service, Memorial Sloan-Kettering Cancer Center, New York City. Treating the patient with acute leukemia can be difficult and dangerous. To enable you to handle your patients more safely, Doctor Dowling outlines the principles and precautions of the management of this disease.
(21 minutes) (in color) **0121942**

AN OUTLINE OF CYTOGENETICS
A survey of the field of human cytogenetics, covering the development of the techniques which make chromosome studies practical — with LCDR Thomas R. Birdwell, MC, U.S.N., Head, Cytogenetics Division, Department of Pathology, U.S. Naval Hospital, San Diego, Calif.
(16 minutes). **1503403**

ANTIBODY AFFINITY AND BIOLOGICAL ACTIVITY: PART I
"Measurement of Antibody Affinity." A discussion of the binding affinity of antibodies for the antigenic determinant, and a description of the two methods used for measuring affinity: equilibrium dialysis and fluorescence quenching. With Gregory W. Siskind, M.D., Assistant Professor of Medicine, New York University Medical Center.
(15 minutes). **0103613**

ANTIBODY AFFINITY AND BIOLOGICAL ACTIVITY: PART II
"Measurement of Antibody Affinity." Gregory W. Siskind, M.D., Assistant Professor of Medicine, New York University Medicine Center, discusses fluorescence quenching for measuring the binding affinity for antibodies for the antigenic determinant. (14 minutes). **0103714**

ANTIBODY AFFINITY AND BIOLOGICAL ACTIVITY: PART III
"Biological Significance of Antibody Affinity." Gregory W. Siskind, M.D., Assistant Professor of Medicine, New York University Medical Center, discusses the heterogeneity of the immune response with respect to antibody affinity and the effects of affinity on in vivo antibody activity. (16 minutes). **0103815**

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BLOOD CELL NEOMORPHOLOGY IN HEMATOLOGIC DISEASE, with Wallace N. Jensen, M.D., Lawrence S. Lessin, M.D., and Dennis S. O'Leary, M.D., all of the Department of Medicine, George Washington University School of Medicine, Washington, D.C. Advanced microscopic and cinematographic techniques have allowed reinterpretations of some older morphologic findings and afforded new observations concerning the reasons for changes in shapes and size of blood cells. The panel reviews the newer findings and offers a current interpretation of their significance. A Television Clinic of the American College of Physicians, 1974. Please inquire for special rental information.
(60 minutes) (in color) **ACP 2847480**

BLOOD COMPONENT THERAPY — FACTOR VIII — RICH CRYOPRECIPITATE. The rational treatment of classical hemophilia using cryoprecipitate is discussed in this presentation by Carol Kasper, M.D., and Judith Pool, Ph.D., of the American Association of Blood Banks Component Therapy Workshop Program. The discovery of cryoprecipitate, dosage schedules, and clinical effectiveness are among the topics covered.
(20 minutes). **0206106**

BLOOD COMPONENT THERAPY — PLATELETS FOR TRANSFUSION. The importance of platelet transfusions in leukemia, in intensive cancer chemotherapy, and their usefulness in other disease states: a report by Gerald S. Gilchrist, M.B., M.C.H., Assistant Professor of Pediatrics, University of Southern California School of Medicine, and Director of the Platelet Donor Center, Children's Hospital of Los Angeles.
(16 minutes). **0206408**

BLOOD COMPONENT THERAPY — THE CLINICAL USE OF GAMMA GLOBULIN. Although safe and effective in many situations, gamma globulin may be contraindicated in others. Both of these areas are discussed by Paul Wehrle, M.D., Hastings Professor of Pediatrics, University of Southern California, and Chief Physician, Children's Division of Los Angeles County General Hospital; Frans Peetoom, M.D., Director of Immunology, Hyland Laboratories; and E. R. Jennings, M.D., Clinical Professor of Pathology, University of California at Irvine, and Director of Pathology, Memorial Hospital of Long Beach.
(20 minutes). **0206307**

CHROMOSOMES BY MAIL

As an alternative to the time-consuming and costly practice of transporting the patient to the testing center, there is now a practical method for shipping blood specimens over long distances for the leukocyte chromosome culture and chromosome analysis. LCDR Thomas R. Birdwell, MC, U.S.N., Head, Cytogenetics Division, U.S. Naval Hospital, San Diego, Calif., demonstrates the technique.

(11 minutes). **0303810**

CONTROL OF HEMOGLOBIN SYNTHESIS IN THE CHICK BLASTODERM — PART I — Preparation of the experimental model with Richard D. Levere, M.D., Assistant Professor of Medicine, Downstate Medical Center, State University of New York. (17 minutes). **0302946**

CONTROL OF HEMOGLOBIN SYNTHESIS IN THE CHICK BLASTODERM — PART II — "The Effect of Delta Aminolevulinic Acid" — with Richard D. Levere, M.D., Assistant Professor of Medicine, Downstate Medical Center, State University of New York. (15 minutes). **0303047**

CONVERSATION WITH GEORGES MATHÉ: IMMUNOLOGICAL APPROACH TO THE TREATMENT OF LEUKEMIA, with Professor Georges Mathé, Director, Institut de Cancérlogie et d'Immunogénétique, Hôpital Paul-Brousse, Villejuif, France; and Ernest H. Rosenbaum, M.D., Director Medical Cancer Service, and Director, Immunological Research of Mt. Zion Hospital in San Francisco.

NCME has videotaped several hours of candid conversation with French professor Georges Mathé, noted for his innovations in treating leukemia. In this telecast, Professor Mathé describes how his active immunotherapy differs from conventional leukemic therapy.

(17 minutes). **0312948**

FINGERPRINTING MYOCARDIAL INFARCTION SERUM ENZYMES. How serum enzyme analysis is used to increase diagnostic accuracy and what prognostic data are furnished are demonstrated by John S. Ladue, M.D., Ph.D., Assistant Professor of Clinical Medicine, Cornell University School of Medicine, New York.

(15 minutes). **0608104**

GROWTH AND DIFFERENTIATION OF HUMAN LYMPHOCYTES: PART I

"Nucleic Acid Synthesis." Arnold D. Rubin, M.D., Associate in Medicine, Mount Sinai School of Medicine, demonstrates the techniques for extracting nucleic acid from lymphocytes, focusing on RNA metabolism. (19 minutes). **0703912**

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Arnold D. Rubin, M.D., Associate in Medicine, Mount Sinai School of Medicine, New York, conducts an investigation of abnormal growth patterns of lymphocytes taken from patients with lymphoproliferative disorders.

(20 minutes). **0704713**

HEMODYNAMICS — AN INSTRUCTIONAL DEVICE. A mechanical model of the cardiovascular system is demonstrated and discussed by Simon Rodbard, M.D., Chief of Cardiology, City of Hope Medical Center, Duarte, California. Designed by Dr. Rodbard as a teaching aid, the machine simulates normal blood flow, and, by opening or closing the appropriate valves, demonstrates cardiovascular dysfunction, as well.

(15 minutes). **0805303**

HOME TRANSFUSION FOR HEMOPHILIA PATIENTS, with S. Frederick Rabiner, M.D., Director of the Clinical Hematology Unit, Michael Reese Hospital and Medical Center, Chicago; and Associate Professor of Medicine at the University of Chicago Pritzker School of Medicine. Hemophilia patients can be administered anti-hemophiliac factor by trained relatives in the home, on vacation — almost anywhere. This is the experience of a three-year program at Michael Reese Hospital. The program, the training for relatives and the results are discussed and shown in this telecast. (20 minutes) (In color) **0813117**

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(16 minutes). **0903501**

IN VITRO STUDIES OF HUMAN LYMPHOCYTES — PART I — "Lymphocyte Culture and Response to Antigens" — with Kurt Hirschhorn, M.D., Professor of Pediatrics and Chief, Division of Medical Genetics, Mount Sinai School of Medicine.

(19 minutes). **0903112**

IN VITRO STUDIES OF HUMAN LYMPHOCYTES: PART II

"Mixed Lymphocyte Culture and Histocompatibility" — with Kurt Hirschhorn, M.D., Professor of Pediatrics and Chief, Division of Medical Genetics, Mount Sinai School of Medicine.

(15 minutes). **0903213**

MULTIPLE MYELOMA: A CONTROLLABLE DISEASE, with Raymond Alexanian, M.D., Associate Professor of Medicine, University of Texas, M.D. Anderson Hospital and Tumor Institute, Houston. In three patients, Doctor Alexanian points out the clinical and laboratory abnormalities in multiple myeloma along with the tests needed to confirm the diagnosis.

(17 minutes) (in color) **1322055**

OBTAINING AND CULTURING LEUKOCYTES FOR CHROMOSOME ANALYSIS

Demonstrating a simple method of obtaining chromosomes for study and analysis — with LCDR Thomas R. Birdwell, MC, U.S.N., Head, Cytogenetics Division, Department of Pathology, U.S. Naval Hospital, San Diego, Calif.

(15 minutes). **1503402**

REGULATION OF BLOOD FLOW BY THE CAPILLARY. A challenge to the theory that blood flow is regulated by the smooth muscle of the arterioles is presented by Simon Rodbard, M.D., Chief of Cardiology, City of Hope Medical Center, Duarte, California. By means of the "capillaron," a mechanical device that he has constructed to show a "packet" of cells supplied by one or more capillaries, he offers the point of view that regulation of blood flow and perfusion of the tissues are controlled at the capillary level.

(13 minutes). **1805208**

RH DISEASE — PART I — PREVENTION. Anti D gamma globulin, an antigen which has proved successful in preventing Rh sensitization in pregnant women, is described by Edward T. Bowe, M.D., Instructor, Obstetrics and Gynecology, College of Physicians and Surgeons, Columbia University, New York.

(15 minutes). **1805618**

RH DISEASE — PART II — MANAGEMENT — AMNIOCENTESIS. Edward T. Bowe, M.D., Instructor, Obstetrics and Gynecology, College of Physicians and Surgeons, Columbia University, discusses the etiology, incidence, and diagnosis of Rh immunization, with particular emphasis on the usefulness of amniocentesis in management of the disease. (15 minutes). **1805719**

RH DISEASE — PART III — MANAGEMENT — INTRAUTERINE TRANSFUSION. The intraperitoneal infusion of O neg. red cells to an Rh positive fetus is "not an occasional procedure" and demands an obstetric team skilled in the technique, according to Edward T. Bowe, M.D., Instructor, Obstetrics and Gynecology, College of Physicians and Surgeons, Columbia University. The intrauterine procedure is demonstrated in this telecast, and Dr. Bowe also covers the criteria for selecting patients, the risk to the fetus, and the over-all rates of success of the procedure compiled from several U.S. hospitals.

(10 minutes). **1805820**

SICKLE-CELL ANEMIA: MANAGEMENT, with Roland B. Scott, M.D., Professor and Head of the Department of Pediatrics, Howard University, and Chief Pediatrician at Freedmen's Hospital in Washington, D.C.

There is no curative treatment for sickle-cell anemia, according to Dr. Scott. However, early diagnosis of the disease, which afflicts more than 50,000 black Americans, can ameliorate the most disturbing symptoms. Dr. Scott describes the therapeutic program he follows to enhance survival until the patient reaches puberty — when the natural course of the disease process appears to become attenuated.

(14 minutes) (In color) **1911506**

SICKLE-CELL ANEMIA: SUSPICION AND DIAGNOSIS IN INFANTS AND CHILDREN, with Roland B. Scott, M.D., Professor and Head of the Department of Pediatrics, Howard University, and Chief Pediatrician at Freedmen's Hospital in Washington, D.C. Also V. Bushan Bhardwaj, M.D., Assistant Professor of Pediatrics, Howard University, and Pediatric Hematologist, Freedmen's Hospital.

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This telecast features the characteristics of the disease, and the laboratory procedure followed to establish a conclusive diagnosis.

(20 minutes) (In color) **1911407**

STRUCTURE AND FUNCTION OF IMMUNOGLOBULINS, with Edward C. Franklin, M.D., Assistant Professor of Medicine, New York University Medical Center. (15 minutes). **1900919**

THE SYMPTOM, ANEMIA: A DIFFERENTIAL DIAGNOSIS, with Herbert C. Lichtman, M.D., Professor of Medicine at the Downstate Medical Center, Brooklyn, New York. Dr. Lichtman and two third-year medical students participate in a teaching session in which one patient case history is traced. Anemia is a concern in this program only as it pertains to the process of arriving at a differential diagnosis. Dr. Lichtman offers the pattern he follows in arriving at a diagnosis and warns against approaches that will void a diagnosis. (21 minutes). **1910624**

TRANSFUSION THERAPY: THE GROWING IMPACT OF FROZEN BLOOD, with Charles Huggins, M.D., Director of the Blood Bank and Transfusion Service, Massachusetts General Hospital, Boston, Blood transfusion therapy as it is practiced in a major medical center, with guidelines to the use of fresh and freshly frozen components.
(14 minutes) (in color) **2022139**

HOSPITAL MEDICINE

A NEW HOSPITAL APPROACH TO ADOLESCENT CARE, with Michael I. Cohen, M.D., Director of the Division of Adolescent Medicine; and Iris Litt, M.D., Assistant Director. The division is part of the Department of Pediatrics, Montefiore Hospital in New York City.

What advantages are there to an adolescent center within a hospital? With the help of young patient, Dr. Cohen and Dr. Litt show many benefits of establishing an adolescent unit as a distinct service of a general hospital.
(17 minutes) (in color) **1411107**

CLINICAL PHARMACY: THE PHYSICIAN'S VIEWPOINT, with Padraig Carney, M.D., Chief of Staff, Memorial Hospital Center of Long Beach, California, and William E. Smith, Jr., Pharm. D., Director, Pharmacy and Central Services, Memorial Hospital Center of Long Beach, California. Although Clinical Pharmacy has only recently gained wide attention, it has been operating at Long Beach since 1959. Dr. Carney gives a candid evaluation of the Clinical Pharmacist as a member of the patient-care team, against a background of specific demonstrations provided by Dr. Smith and his staff.
(21 minutes) (in color) **0315672**

LEGAL PROBLEMS ON THE WARDS. What constitutes a proper informed consent for medical treatment? In what circumstances can information be legally withheld from a patient? What are the consequences of altering a patient's medical record? Points to know about the law to protect a patient's rights and safeguard physician liability are discussed by William Mangold, M.D., J.D., resident in surgery at Bexar County Hospital, San Antonio, and columnist for The New Physician; and Crawford Morris, Esq., trial lawyer specializing in medical malpractice cases for Arter & Hadden Associates, Cleveland.
(23 minutes) (in color) **SAMA 2810962**

MEDICAL ADVANCES INSTITUTE: AN NCME REPORT. MAI, an organization of physicians in Ohio advocating a system of health care review, is providing guidance to physicians in that state who are attempting to establish Professional Standards Review Organizations. Neither the MAI system nor any other has yet been totally accepted by HEW as a model system for PSRO.
(17 minutes) (in color) **1321454**

MEDICAL ASPECTS OF CONSTANT CORONARY CARE UNITS IN A GENERAL HOSPITAL
The problem of the rhythm death following myocardial infarction has provided the stimulus for development of cardiac resuscitation and cardiac monitoring units. The required equipment and techniques are discussed and demonstrated by Richard Watts, M.D., Head, Cardiovascular Section, Department of Medicine, Fairview General Hospital, Cleveland, Ohio. (14 minutes). **1303923**

NEW I.V. TECHNIQUE FOR TOTAL NUTRITIONAL SUPPORT. Maintaining patients on I.V. feeding for long periods of time with satisfactory results can now be accomplished with an indwelling catheter. The technique and its use on patients are demonstrated by Stanley J. Dudrick, M.D., and Douglas W. Wilmore, M.D., Department of Surgery, and Harrison Department of Surgical Research, University of Pennsylvania School of Medicine, Philadelphia, Pa.
(20 minutes). **1406708**

PREVENTION OF HOSPITAL INFECTIONS, with Harold C. Neu, M.D., Associate Professor of Medicine and Chief, Division of Infectious Diseases, Columbia University College of Physicians and Surgeons, New York City. Dr. Neu takes us into an intensive care unit to demonstrate not only the physician's role in preventing nosocomial infections but also his sometimes unwitting role in causing them. (19 minutes) (in color) **1615840**

PSRO: THE ISSUE OF 1974, with Senator Wallace F. Bennett (R-Utah); James L. Henry, M.D., President, Ohio State Medical Association; Robert B. Hunter, M.D., member, AMA Board of Trustees; and J. Lewis Schricker, Jr., M.D., President, Utah State Medical Association. Edmund D. Pellegrino, M.D., Chancellor for Health Sciences, University of Tennessee, is moderator. Senator Bennett's controversial PSRO amendment to Public Law 92-603, the Social Security Act, is outlined and examined. Dr. Pellegrino challenges panelists with major questions surrounding the legislation. Topics include PSRO's cost, effect on malpractice liability, and possible interference in the practice of medicine.
(22 minutes) (in color) **1620947**

THE IMPORTANCE OF PEER REVIEW, with Ralph S. Emerson, M.D., Chairman, Ad Hoc Committee on peer review, Medical Society of the State of New York. Peer review is no longer a professional persuasion, it is a government mandate under the Medicare and Medicaid laws. Dr. Emerson points out the benefits of a properly established and executed peer review committee. He discusses the benefit of review committees to the public; and to physicians as an organized voice in policy making. (13 minutes). **0910603**

THE PROBLEM-ORIENTED MEDICAL RECORD, with Paul Y. Ertel, M.D., Associate Professor of Pediatrics, Ohio State University College of Medicine, Columbus, Ohio.

This Special Workshop offers a comprehensive picture of what physicians and other health professionals need to know to initiate and maintain Problem-Oriented Medical Records. This unique interactive Workshop combines television instruction and workbooks which include POMR forms for the participants to use as they work along with the videotape. Please inquire for special rental information.
(50 minutes) (in color) **2800048**

UNDERSTANDING STATISTICS — PART I. Dr. Vernon E. Weckwerth, Associate Professor, Program in Hospital Administration, University of Minnesota, shows how the probability distributions used by the statistician are directly analogous to the use of experience by administrators as a guide in making decisions.
(15 minutes). **2700103**

UNDERSTANDING STATISTICS — PART II. "Accounting." Robert E. Linde, Director of Finance, American Hospital Association, explains the significance of reports prepared by the accountant to assist the administrator in decision-making.
(15 minutes). **2700204**

A Workshop Conference on Computer Applications to Hospital Dietetic Information. A series of 11 lectures, demonstrations, and discussions taped during two workshops cosponsored by Ohio State University, Division of Medical Dietetics, College of Medicine, and the Health Facilities Planning and Construction Service (Hill-Burton Program), United States Public Health Service, Department of Health, Education, and Welfare, and held at Columbus, Ohio, during the summer of 1967.

Although not produced by NCME the Network makes these tapes available through special arrangements with the Public Health Service.

Please note that these are not conventional television programs, but rather videotape recordings made at portions of a live workshop. Because of the value placed on the videotapes by the Public Health Service, NCME has agreed to make these programs available through its library.

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART A. John P. Casbergue, Associate Professor, Ohio State University, Columbus, Ohio. Introduction and Orientation to the Conference and Evolution of Electronic Data Processing. Mr. Casbergue sets the scene for the four-day conference. **2800015**

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART B. George Christakis, M.D., Associate Professor, Community Medicine (Nutrition), Mt. Sinai School of Medicine, New York. Dietetics and Computers, Their Role in Community Health. Dr. Christakis presents the rationale for the marriage of dietetics and computers for community health. **2800016**

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART C. John P. Casbergue, and James Griesen, Systems Analyst, School of Allied Medical Services, Ohio State University. Basic Computer Concepts in Review. The lecturers describe what is meant by electronic data processing and systems design, using relevant dietary concepts. **2800017**

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART D. Jordan Baruch, D.Sc., Director, MEDINET, Watertown, Massachusetts. Medical Information Systems and Their Implications for Patient Medical and Dietetic Care. Dr. Baruch discusses medical information systems and their implications for patient, medical, and dietetic care. **2800018**

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART E. James Griesen. Systems Analysis and the Role of the Dietitian in Planning the Use of Electronic Data Processing. Mr. Griesen answers the question, "What does electronic data processing mean to the hospital dietitian?" **2800019**

APPLICATIONS TO HOSPITAL DIETETIC INFOR- A WORKSHOP CONFERENCE ON COMPUTER MATION — PART F. John P. Casbergue. Planning a Dietary Information System: Goals and Data Requirements. Mr. Casbergue lists the kinds of information required to meet preestablished system goals. **2800020**

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART G. John P. Casbergue. Demonstration of a Model Dietary Information System. Data are put into the computer and information is printed out in this live demonstration. **2800021**

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART H. Paul Konnersman, Consultant, MEDINET, Watertown, Mass. Planning Considerations in the Use of Electronic Data Processing Systems. A discussion and demonstration of the mathematical foundations of computer-assisted menu planning and how C.A.M.P. assists in writing the menu. This lecture supplements that given by Dr. Baruch in PART D. **2800022**

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART I. John P. Casbergue. The Role of Education for the Professional and Nonprofessional in Planning the Use of Electronic Data Processing. The need for in-service education is stressed. **2800023**

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART J. Joseph L. Balinfly, D.Eng., Professor, Computer System Research, School of Business Administration, Tulane University; and Ronald L. Gue, Ph.D., Director of Research Division, Center for Health and Administration, University of Florida. A Demonstration and Discussion of Computer-Assisted Menu Planning. A discussion and demonstration showing how the computer can assist in writing the menu. **2800024**

A WORKSHOP CONFERENCE ON COMPUTER APPLICATIONS TO HOSPITAL DIETETIC INFORMATION — PART K. John P. Casbergue. Second Conference: Final Discussion and Summary. A lively and varied topic discussion among the participants and the staff of the second conference. Each conference group stressed different needs in their discussion. **2800025**

INFECTIOUS AND PARASITIC DISEASE

ANTIBIOTIC MISADVENTURE: "THE CASE OF OVERKILL," with Harold C. Neu, M.D., Chief of Infectious Diseases, Columbia University College of Physicians and Surgeons, New York City. Test your prescribing ability by following the day-by-day reports on a 70-year-old male patient admitted to the hospital with shaking chills, pleuritic pain, headache, fever, rapid respirations and pulse. This program is part of the "Drug Spotlight Program" sponsored by the American Society for Clinical Pharmacology and Therapeutics.
(8 minutes) (In color) **0118636**

ANTIBIOTIC MISADVENTURE: "THE CASE OF SUPERINFECTION, PAR EXCELLENCE," with Harold C. Neu, M.D., Chief of Infectious Diseases, Columbia University College of Physicians and Surgeons, New York City. See if you can find all of the prescribing mistakes made in this case, which started as a relatively simple problem—a 71-year-old woman complaining of fatigue and nausea, with abdominal mass, elevated body temperature and white count. (This program was presented as part of the American Society for Clinical Pharmacology and Therapeutics' Drug Spotlight Program.)
(13 minutes) (in color) **0118737**

ANTIMICROBIAL TOXICITIES: FROM OFFICE TO HOSPITAL, with Harold C. Neu, M.D., Associate Professor of Medicine and Head, Division of Infectious Diseases, Columbia University College of Physicians and Surgeons, New York City. Help manage a patient with chronic urinary tract infection. As the case unfolds, you select the most effective drug, manage various unexpected complications, and alter or stay with your choice given a variety of clinical situations.
(20 minutes) (in color) **0122343**

ANTIMICROBIAL TOXICITIES: THE INNOCUOUS SETTING, with Harold C. Neu, M.D., Associate Professor of Medicine and Head, Division of Infectious Diseases, Columbia University College of Physicians and Surgeons, New York City. Which antibiotics are effective and least toxic for the patient who has staphylococcal cellulitis . . . vaginitis . . . otitis media and externa? To test your skills in prescribing, help manage a patient with these problems.
(13 minutes) (in color) **0121840**

BACTERIAL MENINGITIS: RAPID ASSESSMENT AND TREATMENT, with Paul F. Wehrle, M.D., Professor of Pediatrics, University of Southern California School of Medicine, and Allen W. Mathies, Jr., M.D., Professor of Pediatrics, University of Southern California School of Medicine, Los Angeles. An estimated 15,000 Americans contract acute bacterial meningitis every year. Between 10 and 15 percent of these die, almost always within twenty-four hours of the onset of symptoms. Drs. Wehrle and Mathies review what the physician must know and do to institute therapy with the speed and precision which can beat the 24-hour deadline.

(18 minutes) (in color) **0215315**

BUGS vs. DRUGS: CAN WE COMBAT BACTERIAL RESISTANCE?, with Harold C. Neu, M.D., Associate Professor of Medicine and Chief, Division of Infectious Diseases, Columbia University College of Physicians and Surgeons. Dr. Neu employs semi-animated graphic art to answer the title's question with a qualified "Yes." He illustrates several of the mechanisms by which bacteria develop resistance and suggests ways in which knowledge of those mechanisms can be used against resistant strains.

(15 minutes) (in color) **0215716**

CHOLERA, 1971: RISK, DIAGNOSIS AND MANAGEMENT, with David J. Sencer, M.D., Director of the Center for Disease Control, Department of Health, Education and Welfare in Atlanta, Georgia; Philip S. Brachman, M.D., Chief of the CDC Epideminology Program; and Eugene J. Gangarosa, M.D., Deputy Chief, Bacterial Diseases Branch, CDC Epidemiology Program.

Cholera, feared since biblical times, is spreading throughout the world. As more tourists travel abroad, concern grows over contacting the disease and infecting the American continent. Three experts from CDC discuss the very small risk to America, and describe the simple steps that can be taken to diagnose and treat cholera among returning travelers.

(17 minutes) (in color) **0312909**

COUNSELLING THE VD PATIENT. The sexual overtones of venereal disease expose VD patients to a special complication . . . a value judgment of their behavior. Such judgment, however unintentional, can detract from the treatment of the disease. Dr. Mary Howell of the Somerville Women's Health Project in Somerville, Massachusetts, Rev. Thomas Mauer of the University of Minnesota Human Sexuality Program and three medical students use role playing to illustrate these difficulties and discuss their solution. (22 minutes) (in color) **SAMA 2810860**

CURRENT STATUS OF THE PROBLEM OF VENEREAL DISEASE, with Frank M. Calia, M.D., Department of Medicine, and R. C. Vail Robinson, M.D., Division of Dermatology, both of University of Maryland School of Medicine; and Richard Hahn, M.D., Department of Medicine, the Johns Hopkins University School of Medicine. The panelists discuss the epidemiology, diagnosis and treatment of syphilis and gonorrhea. Special emphasis given to methods of diagnosis in asymptomatic infections, extragenital manifestations of gonorrhea, increasing penicillin resistance of the gonococcus, use of single dose therapy in gonorrhea, dermatologic manifestations of syphilis, and problems related to the serologic diagnosis of syphilis.

A Television Hospital Clinic of the American College of Surgeons, 1972. Please inquire for special rental information.

(57 minutes) (in color) **ACP 2857242**

DID YOUR PATIENT GET HIS DISEASE ABROAD? with Kevin M. Cahill, M.D., D.T.M. & H. (Lond.), Director of the Tropical Disease Center at Lenox Hill Hospital, New York, and Professor of Tropical Medicine, Royal College of Surgeons in Ireland. Your patients may present you with amebiasis or malaria if they travel, have been in Vietnam, have had contact with those who have been abroad or if they experiment with drugs. Dr. Cahill shows you how to diagnose and treat these diseases, and offers advice on prevention.

(19 minutes) (in color) **0417540**

DRUG INTERACTION: "THE CASE OF THE PUSHY ANTIBIOTIC," with Harold C. Neu, M.D., Head of Infectious Diseases, and Associate Professor of Medicine, Columbia University College of Physicians and Surgeons, New York City. When can the right selection of antibiotics be wrong? In four clinical situations, says Dr. Neu in this "Drug Spotlight Program," presented in cooperation with the American Society for Clinical Pharmacology and Therapeutics.

(9 minutes) (in color) **0419146**

DRUGS vs. BUGS: CHOOSING THE RIGHT ANTIBIOTIC, with Harold C. Neu, M.D., Associate Professor of Medicine and Chief, Division of Infectious Diseases, Columbia University College of Physicians & Surgeons, New York. The best way to choose an antibiotic is to match its antibacterial action to the organism's susceptibility. Dr. Neu uses lively graphics to demonstrate the metabolic effects of several commonly used antibiotics, and offers some practical advice on choosing the right drug for the bug.

(15 minutes) (in color) **0415632**

GONORRHEA: ELUSIVE EPIDEMIC, with Frank M. Calia, M.D., Chief of Infectious Diseases, Loch Raven Veterans Administration Hospital, and Associate Professor of Medicine, University of Maryland School of Medicine, Baltimore, Maryland.

An aggressive approach to this seemingly unbeatable venereal disease. Shows screening procedure for catching the silent carrier, and how to handle new oriental strains of GC.

(20 minutes) (in color) **0717417**

GONORRHEA: A PLAGUE OUT OF CONTROL

The incidence of gonorrhea is increasing at a rate of 10 to 15 per cent a year. Although many people jump to the conclusion that this is due to relaxed morals and increased liberality in sex, the National Communicable Disease Center in Atlanta, Ga., feels differently. Discussing the problem, new diagnostic techniques and ways to eradicate the disease are William J. Brown, M.D., and Leslie C. Norins, M.D., Chief of the Center's Venereal Disease Research Laboratory.

(12 minutes) (in color) **0709909**

HERPES SIMPLEX: VIRAL DILEMMA, with Richard C. Gibbs, M.D., Associate Professor of Clinical Dermatology, New York University Medical Center, New York City. A clinical presentation, stressing some precautions in the differential diagnosis and treatment of the herpes simplex virus, Types I and II.

(13 minutes) (in color) **0820135**

HEXACHLOROPHENE: OPEN TO DEBATE, with Harold C. Neu, M.D., Associate Professor of Medicine and Chief, Division of Infectious Diseases; Stanley James, M.D., Professor of Pediatrics and Chairman of the American Academy of Pediatrics Committee on the Fetus and Newborn; Carl Nelson, M.D., Professor of Dermatology and President of the American Dermatological Association. All of the participants are on the faculty of the Columbia University College of Physicians and Surgeons, New York City. Since December 15, 1971 bathing newborns with hexachlorophene, routine in most nurseries, has been banned by the F.D.A. and the American Academy of Pediatrics—or has it? Our panel looks at this new problem from several angles and comes up with some interesting conclusions.

(16 minutes) (in color) **0815520**

IMMUNIZATION FOR FOREIGN TRAVEL. Although Federal law requires certain immunization shots for Americans going abroad, physicians can recommend additional protection for their traveling patients by knowing what major diseases are endemic to particular areas of the world and prescribing the appropriate shots. Discussing this proposal is E. L. Buescher, M.D., Chief, Department of Virus Diseases, Walter Reed Army Institute of Research, Washington, D.C.

(15 minutes) **0905702**

INFECTIOUS MONONUCLEOUS, with James C. Niederman, M.D., Associate Clinical Professor of Epidemiology and Medicine, Yale University School of Medicine, New Haven, Conn. Diagnosis and treatment of an important clinical entity—plus some of the burgeoning research implications. (16 minutes) (in color) **0919525**

KEEPING UP ON IMMUNIZATIONS, with Samuel L. Katz, M.D., Professor and Chairman of the Department of Pediatrics, Duke University Medical School, and Chairman of the Committee on Infectious Disease of the American Academy of Pediatrics, Durham, North Carolina. Here's a quiz on preventive practice. Six cases point up some problems in "routine" office immunization.

(18 minutes) (in color) **1121804**

THE LABORATORY IN DIAGNOSIS OF PNEUMONIA. Pneumonia continues to account for 45,000 deaths each year. Treating a patient with drugs which may suppress but fail to eradicate the infecting organism may place him in jeopardy, and provides less than optimal care. Discovery of the etiologic agent is determined in the laboratory by such efforts as microscopic, cultural, serological and antimicrobial sensitivity tests, according to Robert Austrian, M.D., John Herr Musser Professor and Chairman, Department of Research Medicine, University of Pennsylvania School of Medicine.

(16 minutes). **1207201**

"THE NATIONAL ANTIBIOTIC THERAPY TEST." The National Antibiotic Therapy Test consists of a seventy-five minute videotape workshop with the objective of self-assessment and learning about the proper use of antibiotics in medical practice. The practicing physician is confronted with patient problems requiring decision on the use or nonuse of antibiotics.

NATT was also designed for a broad range of medical specialties including the family physician, the internist, the pediatrician, the otolaryngologist, the urologist, the obstetrician/gynecologist, and the general surgeon. The test scores of the participating physician can be compared with those of other physicians throughout the country. The test is self-administered, and self-scored.

Please call NCME for special price information which includes scoring folders.

(75 minutes) (in color) **2800075**

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THE NATIONAL ANTIBIOTIC THERAPY TEST: FIRST RESULTS, with Alan L. Goldberg, M.D., family physician, Bronx, New York; Harold C. Neu, M.D., Head, Infectious Diseases, Columbia University College of Physicians and Surgeons, New York City; and Edmund D. Pellegrino, M.D., Professor of Medicine and Chancellor for Health Sciences, University of Tennessee. "First Results" on the National Antibiotic Therapy Test include 4,513 scores of physicians taking the examination. The national averages and how various specialty groups scored are among the results presented on this program. Five of the questions, the most difficult on the test, are reviewed. (17 minutes) (in color) **1420834**

PARASITIC INFESTATION: LOOK FOR LICE, with Silas E. O'Quinn, M.D., Professor of Dermatology and Dean of Medicine; and Harold Trapido, Ph.D., Professor of Tropical Medicine and Medical Parasitology, both at Louisiana State University School of Medicine in New Orleans. A comprehensive look at what *may* be causing that terrible itch. Knowledge of your patients' hygiene does not rule out lice.
(11 minutes) (in color) **1620645**

PARASITIC INFESTATION: SCABIES, with Silas E. O'Quinn, M.D., Professor of Dermatology and Dean of Medicine; and Harold Trapido, Ph.D., Professor of Tropical Medicine and Medical Parasitology, both at Louisiana State University School of Medicine in New Orleans. Everything you always wanted to know about mites but (were too busy) (forgot) to ask.
(14 minutes) (in color) **1621348**

PREVENTION OF HOSPITAL INFECTIONS, with Harold C. Neu, M.D., Associate Professor of Medicine and Chief, Division of Infectious Diseases, Columbia University College of Physicians and Surgeons, New York City. Dr. Neu takes us into an intensive care unit to demonstrate not only the physician's role in preventing nosocomial infections but also his sometimes unwitting role in causing them. (19 minutes) (in color) **1615840**

Each program is accompanied by a review card. NCME asks that the person for whom the program was ordered fill out and return this card. Because reevaluation of Master Library videocassettes is a continual process, return of the program review card is essential in helping NCME determine which programs remain useful as resources for continuing medical education.

PROBLEMS OF BACTERIAL INFECTION — PART I. With David H. Smith, M.D., Assistant Professor of Pediatrics, Harvard Medical School, and Children's Hospital Medical Center, Boston, Massachusetts.

How do bacteria become resistant to antibiotics and what does this mean to physicians in the care of their patients?

This problem is becoming increasingly difficult for physicians whose patients may suddenly cease to respond to a medication or may suddenly develop "hospital-based" infections.

In this presentation, Dr. Smith shows how these problems are being met. He demonstrates the selection process of drug resistance development in bacterial populations and illustrates hospital-by-hospital, ward-by-ward differences in resistant pathogens. Dr. Smith emphasizes the need to determine the pattern of multiple infectious drug resistance of bacteria in a hospital environment, so that the most appropriate and effective drug can be prescribed.
(16 minutes). **1607025**

PROBLEMS OF BACTERIAL INFECTION — PART II. The need for physicians to understand the pattern of drug resistance that exists in hospital wards . . . and what to do with these vital data . . . is pointed up by David H. Smith, M.D., Assistant Professor of Pediatrics, Harvard Medical School, and Children's Hospital Medical Center, Boston, Massachusetts. (16 minutes). **1607126**

ROCKY MOUNTAIN SPOTTED FEVER, with Theodore E. Woodward, M.D., Professor and Head, Department of Medicine, University of Maryland School of Medicine and Hospital, Baltimore. Using three patients with diagnoses in doubt, Doctor Woodward helps you arrive at the proper diagnosis, noting the distinguishing features of spotted fever and its mimicking diseases.
(19 minutes) (in color) **1820329**

RUBELLA. Albert McKee, M.D., Department of Microbiology, University of Iowa School of Medicine, discusses the isolation of the rubella virus and the problems that the disease causes in pregnancy. (26 minutes) **2800002**

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