propagation of a defective short-lived cell line. Recovery from aplasia however, suggests the emergence of precursor cells with "new" mitotic competence. This may result from biochemical "recovery" of the injured cells through the development of alternate metabolic pathways or the emergenc of a genetically different line of stem cells. This genetically different line of stem cells may be "normal" or may differentiate into an autonomous leukemic cell population.

In the issue of the *Journal* Brauer and Dameshek record 3 examples of acute myeloblastic leukemia developing in patients who had aplastic anemia that followed chloramphenicol therapy.[5] As pointed out by the authors, aplastic anemia without known cause may be premonitory of acute leukemia; accordingly, any postulated relation between chloramphenicol and the leukemia in the 3 cases recorded must be regarded as conjectural. On the other hand, it is possible that acute leukemia would be seen much more frequently in patints with chloramphenicol-induced bone-marrow aplasia if they survived longer; most of these patients succumb to their disease within seven months from its onset. At present it is reasonable to consider any agent that is potentially myelotoxic as being also potentially leukemogenic. However, little can be said in this regard until more is known about the basic mechanisms by which chloramphenicol and other drugs injure the bone marrow.

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[5] See article, "Hypoplastic Anemia and Myeloblastic Leukemia Following Chloramphenicol Therapy," by Dr. Brauer and Dr. Dameshek, p. 2402, supra.

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CHLORAMPHENICOL OPTIC NEURITIS

APPARENT PROTECTIVE EFFECTS OF VERY HIGH DAILY DOSES OF PYRIDOXINE AND CYANOCOBALAMIN

(By Maj. Joseph G. Cocke, Jr., MC, USA, Fort Sam Houston, San Antonio, Tex.)

Over the last several years, there has been increasing recognition of an apparent deleterious effect of chloramphenicol on vision in the form of an optic neuritis. Recognition of this entity has not produced any uniform suggestion for treatment or prevention of the neuritis other than minimal total dosage or withdrawal of chloramphenicol once toxic eye signs are noted.

Experience with a previously reported [1] 12-year-old girl with cystic fibrosis who developed an episode of chloramphenicol optic neuritis (CON) following prolonged use of the drug has led to the suggestion that pyridoxine (B_6) or cyanocobalamin or both in very high daily doses may be of significant value in prevention of optic neuritis while the patient is receiving sustained chloramphenicol treatment.

REPORT OF A CASE

A 12-year-old girl with proved moderately severe cystic fibrosis (CF), developed an optic neuritis in January 1964 following total dosage of 135 gm chloramphenicol. From near total blindness associated with contricted visual fields, large central scotomata, papilledema, and retinal hemorrhages, her visual acuity improved to 20/50 for both eyes while receiving treatment. Also, visual fields widened except for small central scotomata, and fundus changes resolved save for minimal residual disc pallor. Therapy consisted of stopping chloramphenicol administration and administering large doses ascorbic acid, thiamine, pyridoxine,

NOTE.—Numbered footnotes at end of article, p. 2656.