outcome rates. Thus, among the remaining 151 patients who had drug-attributed marrow depression, three received a diagnosis of leukemia *after* submission of the initial registry report (cases 1 through 3).

Among these three cases (all from the chloramphenicol series), only one had characteristics which would suggest a causal relationship between drug intake and leukemia. In case 1, a complete hemogram performed five months after the start of intermittent chloramphenicol therapy was entirely normal. Eight months after therapy began, the diagnosis of aplastic anemia was made from bone marrow examination; serial blood counts showed persistence of this condition until 2½ years following the start of therapy when an abrupt conversion took place to acute myelogenous leukemia.

Patient 2 did not have a clearly defined sequence of drug ingestion—marrow hypoplasia—leukemia. The diagnosis of "aregenerative anemia" was made three months after therapy with chloramphenicol; however, granulocytic hyperplasia of the marrow was present throughout the course of the pancytopenia. The diagnosis of chronic myelogenous leukemia was not made until death, 3½ years after drug ingestion, but it seems possible that this patient was in an early phase of leukemia at the onset of the drug-attributed blood dyscrasia. In case 3, the "latent period" between start of chloramphenicol therapy and diagnosis of leukemia was relatively short (five months), and the reporting physician could not exclude the possibility that leukemia was actually present at the time of the initial leukopenia.

Other Sequelae.—The following diseases subsequently developed in nine patients who had recovered from hematotoxic effects of chloramphenicol: hepatitis in two; renal failure in two; and cirrhosis, systemic lupus erythematosus, lung cancer, hypernephroma, and Gaucher's disease in one each. In addition, two patients who were still under care for chloramphenicol-attributed marrow depression had a history of hemolytic anemia during treatment of the hypoplastic marrow with prednisone and testosterone. In one case the hemolysis was associated with a positive Coomb's test reaction and subsided without additional therapy; in the other case, the hemolysis was successfully treated by splenectomy. (These cases are distinct from the two patients with toxic effects from chloramphenicol, noted in Table 2, who were still receiving care for paroxysmal nocturnal hemoglobinuria.)

Of four patients who recovered from toxic reactions to phenylbutazone, cirrhosis, lupus erythematosus, aseptic necrosis of the femoral head, and gout developed in one each. Sequelae were also described in the two patients who recovered from hematotoxicity associated with the administration of both drugs; one patient had a transient granulocytic leukemoid reaction, and a disorder resembling Weber-Christian disease developed in the other. As with leukemia, it is possible that some of these diseases, although diagnosed subsequent to the blood dycrasia, were actually present at the time of drug administration.