earlier age, attacks particularly the arteries of the legs and frequently results in gangrene.

The disease often lasts up to a year; it seems to be self-limited, and is rarely fatal. No specific organism has been discovered to be causative, nor does drug allergy appear to play any part, as seems so probable in periarteritis. From experience in one case, penicillin appears to be without effect. Cook *et al.* (1946) used nicotinamide in their cases, but were not impressed with its effect. I have wondered whether the use of the anti-coagulants heparin or dicoumarol early in the disease might not reduce the risk of permanent damage, especially in the brain and retina.

ADDENDUM

Since writing this note I have had the opportunity of studying a number of papers by American writers (Gordon and Thurber, 1946; Dantes, 1946; Shannon and Solomon, 1945; Hoyt, Perera, and Kauvar, 1941; Chasnoff and Vorzimer, 1944; Post and Sanders, 1944), and it has been striking how closely the clinical and pathological findings have approximated to one another in all the descriptions, thus confirming my impression that the syndrome is sufficiently characteristic to suggest the diagnosis to physicians who are confronted with it. All the writers agree as to age incidence, a prolonged subacute phase with low fever, progressive secondary anaemia, mild leucocytosis, and high sedimentation rate, during which the main symptoms, already named, are prominent, followed by a protracted stage of slow recovery, with reduction of symptoms and improving blood picture. All agree that the sedimentation rate falls relatively slowly and may still be abnormal after the main symptoms have disappeared. All agree, too, that the tendency is towards spontaneous recovery, and that mortality is low. Most writers note that the disease appears more often in women.

Shannon and Solomon (1945) record a case with permanent bilateral loss of vision, as was present in my second case. Hoyt, Perera, and Kauvar (1941) note the symptom of pain on moving the jaw. This symptom appears not infrequently, and was prominent in my third case. Post and Sanders (1944) stress the expressionless facies, and refer to a Parkinsonian appearance. This was very striking in Cases I and II, above. Many writers have described reduction in pain after arterial biopsy, though Dantes (1946) noted but little improvement in his case. Relief following biopsy was unquestionable in the two of my cases in which this was carried out. All writers agree that the aetiology remains obscure, and no useful therapy apart from arterial resection has been suggested. I can find no reference to the attempted use of anticoagulants.

I wish to thank Drs. Proverbs, Carling, Bowers, and Barnett, who referred to me the four cases here described; also Drs. Wrigley, Gleave, and Edwards, who were responsible for the pathological investigations.

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ANASTOMOTIC ULCER 12 MONTHS AFTER SOMERVELL OPERATION

BY

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The following case, in which an anastomotic ulcer occurred twelve months after a Somervell (1942) operation and gastro-enterostomy, may be of interest.

Case Report

A man aged 44 began to suffer from painful indigestion in 1942. In April, 1944, he was discharged from the Army with a diagnosis of duodenal ulcer and pyloric stenosis. I saw him in November, 1945, when a clinical diagnosis of duodenal ulcer and recurrent appendicitis was made. The barium meal showed a small ulcer deformity at the incisura angularis, and the pyloric canal was displaced to one side of the duodenal cap—consistent with an old duodenal ulcer. The fractional test meal showed that the highest total acid and free acid were 50 and 40 respectively (Fig. 1). Operation was advised because of the history

FIG. 1.—The pre- and post-operative acidity curves.

of having recently vomited food which he had taken the previous day, indicating impending stenosis.

First Operation.—On Jan. 1, 1946, the Somervell operation of ligature of all the gastric vessels and a posterior gastro-enterostomy was performed. The ligatures included the left coronary artery, the pyloric artery, and all the branches of the right and left gastro-epiploic arteries from the pylorus to the lower pole of the spleen (Fig. 2). He made a good recovery.

FIG. 2.—Somervell operation, Jan. 1, 1946. Ligature of gastric vessels and gastro-enterostomy.

Two months later he stated that he was feeling better but was afraid to eat freely on account of the pain it caused. He was putting on weight. A second fractional test meal showed that the highest total acid and free acid were 60 and 45 respectively, in contrast to the pre-operative figures of 50 and 40. In March he started work, his weight had increased by 2 lb. (0.97 kg.), and he was feeling well. When seen on Aug. 16, 1946, he complained of pain in the left iliac fossa, although it was not quite so severe as before operation. His abdomen felt sore. A barium meal showed a gastro-enterostomy deformity of the stomach, and the crater of a gastric ulcer just above the stoma. Operation was advised because of the pain from the increasing

The Council of the International Organization for Standardization held its first meeting in Zürich on June 17-20 under the presidency of Mr. Howard Coonley, of the U.S.A. The Council consists of representatives from the national standards bodies of Britain, Australia, Belgium, Brazil, China, France, India, Norway, Switzerland, the U.S.A., and the U.S.S.R. Mr. Coonley is president of the I.S.O., and the Council appointed M. Henri St. Leger general secretary. It was recommended that the International Electro-technical Commission should affiliate with the I.S.O.

acidity, and especially because, in my experience, if a patient has any sort of discomfort after a gastro-enterostomy he has almost certainly a stomal ulcer, and the sooner the anastomosis is undone the better. He declined this.

He returned in October, 1946, when he was "not feeling too good." He still had the pain, and his appetite was only fair. A third fractional test meal showed that the highest total acid and free acid were 70 and 52 respectively in contrast to the previous figures of 60 and 45 (see Fig. 1). Operation was again advised, and this time it was accepted.

Second Operation, November, 1946.—An enormous gastric ulcer was found, beginning on the stoma and passing widely up on to the lesser curvature, perforating into the pancreas as shown in Fig. 3, which is taken from the artist's painting of the

FIG. 3.—Findings at re-operation, Nov. 19, 1946. Lines A—B and C—D indicate area of stomach excised. The size of the ulcer is also shown.

specimen. It was approximately 3 by 3½ in. (7.6 by 8.9 cm.). A partial gastrectomy was performed (Fig. 4) and he made a good recovery.

FIG. 4.—Operation (Nov. 19, 1946) completed.

Comment

The points I wish to present in reporting this case are: (1) This is the largest anastomotic ulcer I have seen in a personal series of 40 cases and certainly the quickest to develop after a gastro-enterostomy (twelve months). (2) The progressive increase in the acid content of the gastric juice is the opposite to that for which the operation is designed. (3) I have performed the Somervell operation for several patients with unimpressive results, and therefore offer the conclusion that, for London patients at any rate, the Somervell method offers no advantage. (4) Finally, in my experience, if a patient after a gastro-enterostomy is not completely comfortable, but needs to diet or to take powders, then the sooner the gastro-enterostomy is undone the better. A stomal ulcer is likely to be present, and in my patients it was seldom shown by the radiographs.

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THE COMBINED ACTION OF PHENOBARBITONE AND EPANUTIN

BY

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The two most important drugs in the modern treatment of epilepsy are phenobarbitone (5-phenyl-5-ethylbarbituric acid, "luminal," "gardenal") and "epanutin" (soluble phenytoin, sodium diphenylhydantoinate, "soluble dilantin"). It is only when the pharmacology, toxicity, etc., of these drugs are appreciated that they can be used with the maximum efficiency; I have dealt with this matter fully elsewhere (Blair, 1940). For the present it suffices to say that the chief disadvantage of the former is the drowsiness it may produce in adequate anticonvulsant doses, and of the latter the toxic effects (particularly those of a neurological, mental, or dermatological nature) when administered in doses of over three or four capsules (1½ gr.—0.1 g.—each) daily. A number of authors have tried these two drugs in combination in the hope that a synergistic action might occur.

The reports published to date (see Bibliography) have varied in their findings; some claim that in nearly all cases the use of epanutin and phenobarbitone together is more efficient than when these drugs are administered separately, but others find that in combination they are seldom better than when one or other is given alone. Moreover, little has been said regarding the frequency of toxic symptoms when they are combined, the optimum dosage in which they may be administered, etc.

In my experience there is a definite place in anti-convulsant therapy for the combined use of epanutin and phenobarbitone, and the present work was undertaken* to try to throw some light on the proportion of cases in which the combination of the drugs is more efficient than either administered separately (a) in controlling the number of fits and (b) in improving behaviour; the optimum dosage in which the two drugs may be combined; what toxic effects or other complications occur during their administration; and their pharmacological action.

Method and Results of Investigation

All patients concerned in this series were epileptic psychotics, mostly of long standing. For purposes of description the work may best be considered in three different categories: (1) the effect of adding epanutin in patients previously on phenobarbitone; (2) the effect of adding phenobarbitone in patients previously on epanutin; (3) a clinical trial of the two drugs combined in capsule form (referred to throughout this article as "E and P capsules").

Patients Previously on Phenobarbitone

In this group 30 chronic psychotics (whose ages ranged from 32 to 67 and averaged 42 years) had been on phenobarbitone for at least twelve months previously. Epanutin was added and the results observed. In a few cases previously on 3 or 4 gr. (0.2 or 0.26 g.) of phenobarbitone the dosage was reduced for control purposes to 2 gr. (0.13 g.) after epanutin had been combined with the former dose for several months.

* At Cane Hill Hospital in 1940-1 and not previously published owing to my absence on service in the R.A.M.C.