I wish to thank Dr. Wrigley, of Messrs. Roche Products, for procuring a copy of Gopalan's article and sending it out to me in Greece; and Dr. A. A. Weinstein, of Atlanta, Ga., for refreshing my memory of details of the "fits" episode. The food figures are taken from the "Nutritive Values of Wartime Foods," No. 14 of the M.R.C. war memoranda, or from Bicknell and Prescott.

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TEMPORAL OR GIANT-CELL ARTERITIS

BY

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Since 1932 reports have been published from time to time describing what appears to be an unusual form of arterial inflammation. This condition seems to have a particular predilection for the superficial cranial arteries, and so frequently have the temporal arteries been involved that it has been known as "temporal arteritis." More recently Cooke *et al.* (1946), in a masterly review of the condition and its literature which includes a full description of seven of their own cases, suggest that the condition is probably less rare than the literature suggests—only forty cases had been described up to 1946—and that its effects are not confined to the cranial vessels but may be widespread throughout the body.

Their histopathological findings differed little from those of other writers, and they describe a subacute inflammatory process which, starting in the adventitia, subsequently spreads axially to the media, and is accompanied by areas of necrosis in the media and interna elastica of such a nature as to suggest an obliterative process in the vasa vasorum. They confirm the finding of giant cells, which give to the condition one of its names. The appearance of these follows necrosis, differing in this respect from tuberculosis, in which they precede necrosis. There is nothing to support a tuberculous hypothesis when considering aetiology.

My observations support the thesis, so ably presented by these workers, that the condition is by no means rare and that its recognition is difficult unless the possibility is borne in mind, but easy when once seen or the clinical picture appreciated. I believe that the diagnosis will be made with ever-increasing frequency and that neurologists particularly may find it useful in answering a number of baffling problems. For these reasons I feel that at the present time reports should be made of cases as they appear, even if only individual cases can be recorded.

Case I

In July, 1945, I was asked to see a man aged 65 who had shown an unexplained temperature for over a month. Two years previously he had suffered from intermittent claudication in the left leg. This had not been severe, and had been in abeyance for over a year. Apart from this the patient, who

had been an Army instructor in physical training, presented an excellent medical record. About six weeks before I saw him he had begun to experience lassitude, depression, and widespread pains about his body. He had lost his appetite, and after ten days retired to bed. His condition deteriorated, and sweating at night became pronounced, while severe headache made its appearance. At this stage he asked his doctor to visit him. Physical examination was negative, but it was found that fever up to 101° F. (38.3° C.) was present. This condition continued, and headache became very severe, preventing sleep. The Widal reactions were investigated and were found to be negative.

When first seen on July 29 his appearance was striking. He appeared ill and looked older than his age. His face was expressionless and suggested profound dejection, which was confirmed in conversation. Physical examination was unproductive, but it was noted that there was no pulsation in the left dorsalis pedis artery and that the left temporal artery and both facial arteries were thrombosed. Furthermore, they were exceedingly tender to the touch, as was the right temporal artery though it was pulsating. There was, in addition, tenderness in the occipital region and the patient explained that his head had been so sore that it was sometimes impossible to lay it on the pillow. The only other findings were an absence of the abdominal reflexes and exaggerated deep reflexes. The plantar responses were flexor. Heart and lungs seemed normal and the blood pressure was 150/90. The fundi were within normal limits. A tentative diagnosis of periarteritis nodosa was suggested and he was admitted next day to hospital.

In hospital further investigations were carried out. A skiagram of the chest showed no abnormality in the heart or lungs. Electrocardiograms were normal. The urine showed a trace of albumin, a few red cells and epithelial casts, and was sterile. The blood count revealed 5,000,000 red cells, 80% haemoglobin, and 6,600 white cells, with a normal differential count apart from an eosinophilia of 7%. The blood sedimentation rate showed a fall of 40 mm. in the first hour when estimated by Wintrobe's method. The blood urea was 42 mg. per 100 ml.; the Wassermann reaction was negative. Agglutinations for enteric and *Br. abortus* were negative. The cerebrospinal fluid was normal, except that 5 cells per c.mm. were present; the Wassermann reaction and the gold-sol curve were negative. Blood culture was sterile.

An inch (2.5 cm.) of the anterior branch of the right temporal artery was removed under local analgesia, and the microscopical appearances here were consistent with a diagnosis of temporal arteritis. The operation produced a remarkable alleviation of headache in this part of the head. During his stay in hospital the fever persisted, but there was a gradual improvement in his general condition. Headache was reduced, sweating became less severe, appetite improved, and he became rather less depressed. His haemoglobin level fell to 63% and his sedimentation rate continued high. Apart from symptomatic treatment he was given nicotinamide by mouth. He was discharged home to bed, where his improvement was maintained.

He was readmitted to the surgical ward at the end of November with gangrene of a toe on the left foot. I did not see him then, but the fact that his sedimentation rate was still high, though falling, suggests that the condition was active. He was apyrexial. A left femoral periarterial sympathectomy was carried out in an attempt to save the toe, but it was not successful and amputation became necessary later. Though no sections were cut off the arteries at the site of the operation, it seems likely that the gangrene was due to arteriosclerosis obliterans and not to giant-cell arteritis. It will be recalled that he had suffered from intermittent claudication two years previously and that there had been no tenderness of the dorsalis pedis artery.

By June, 1946, the patient seemed perfectly well, and his wife assured us that he had suffered no ill effects as a result of his long illness. Furthermore, his blood picture was normal and the sedimentation rate only 4 mm. During his convalescence from the operative treatment there had been a period of mental confusion lasting several days. We did not see him in this state, and it is difficult to be sure whether or not this was

related to the drugs he was receiving or to cerebral ischaemia. Mental symptoms have from time to time been recorded (Cooke *et al.*, 1946).

Case II

On June 27, 1946, a man aged 65 was led into my consulting-room at the hospital; at once something in his general appearance recalled the case which has just been described. He was expressionless, dejected, and ill-looking. He had been well until April, when symptoms of lassitude, failing strength and appetite, muscular pains, sweating at night, and especially severe and increasing pains in his head, had appeared. There had also been an unproductive cough and he had lost weight. He told us that his head had been so painful that it was often impossible to tolerate contact with the pillow, and, in his own words, "the veins" of his head had been very tender to the touch. Three days before the consultation he had lost the sight of his right eye and within a couple of hours the left eye had shared the same fate.

On examination both temporal and both facial arteries were thrombosed and very tender, and there was tenderness on palpation over the occipital region. His fundi showed bilateral thrombosis of branches of the central retinal arteries with fragmentation of the blood stream, well seen in other branches. His abdominal reflexes were absent, but no further physical signs were discovered. The blood pressure was within normal limits.

He was admitted to hospital at once for fuller investigation. He was found to be running a temperature up to 100° F. (37.8° C.). Blood examination showed 4,800,000 red cells; haemoglobin 84%; white cells 9,000, with a normal differential count; blood urea 35 mg. per 100 ml.; blood sedimentation rate 43 mm. in the first hour (Wintrobe); Wassermann reaction negative. The urine showed a trace of albumin, a few red cells, and an occasional hyaline cast. The cerebrospinal fluid revealed no abnormality, and the Wassermann reaction and gold-sol curve were negative. Blood culture was sterile and the usual agglutinations were negative. A skiagram of the chest and skull revealed no abnormality and the E.C.G. tracing was normal.

An inch (2.5 cm.) of the right temporal artery was removed under local analgesia—a portion was cultured but grew nothing—and the histological appearances of the sections made were quite characteristic of temporal arteritis.

While in hospital he developed an area of superficial inflammation of the scalp along the distribution of the left temporal artery. This area was tender and persisted for about three weeks. His right plantar response became extensor for about a week and there seemed to be an exaggeration of both knee-jerks. His general condition altered very little; he remained febrile, quiet, and uncomplaining except when questioned; it was, however, clear that head pain remained severe, though it had been relieved in the region of the biopsy. He was given 3,000,000 units of penicillin in a week, but so far as one could say this had no effect upon his condition. At the end of a month in the ward fever remained at about the same level, his haemoglobin had fallen to 58%, and his blood sedimentation rate remained high.

When last seen on Aug. 28 he was showing considerable improvement. His headache was much less severe, his appetite was returning, and he was more inclined to talk. There had been no return of vision, and his sedimentation rate had fallen a little, to 35 mm. I have not seen him again, but I understand that he has continued to improve in every way except that he remains blind.

Case III

In September, 1946, I was asked to see a woman of 73 who had been in hospital for three weeks with an unexplained fever. This had remained between 100° and 101° F. (37.8° and 38.3° C.), and in spite of a very thorough investigation was still unexplained. She had been hypertensive for a long time, and had complained of severe cephalalgia for some three months. There had also been pain on moving the jaw, general lassitude, depression, and anorexia. The only positive finding in her investigation was a very high sedimentation rate. On examination she had tender facial, temporal, and occipital

arteries, though pulsation was present in all these vessels. No biopsy was possible, but I have little doubt as to the diagnosis, and her subsequent slow but steady progress towards recovery fits in well with the condition under discussion.

Case IV

A man aged 67 was first seen early in 1946 suffering from symptoms characteristic of gastric ulcer. He had had similar attacks in the past, and the present symptoms cleared up well on routine treatment. Some three months later he was seen again on account of severe headache, pain in the right shoulder, and general weakness. There had been no recurrence of his dyspepsia. Physical examination was negative, but there was tenderness in the occipital region and over the course of the temporal arteries, though pulsation seemed normal. Investigations were not possible, and a tentative diagnosis of temporal arteritis was suggested. His symptoms altered little during the next six weeks, but at the end of this time he developed a complete right twelfth cranial-nerve palsy.

Mr. McKeith could find little amiss in his throat, and skiagrams of the skull and neck were normal. Two weeks later, by which time his twelfth cranial-nerve palsy had recovered very considerably, he was seen by Mr. Pennybacker, who quite independently suggested temporal arteritis as a possible diagnosis, but in view of a sedimentation rate only a little raised he did not feel able to be dogmatic. A month later the patient suffered an acute perforation of a gastric ulcer high up in the lesser curve. Before operation it was noticed that his left radial pulse was absent. He did not survive the operation, and at a post-mortem examination the left subclavian artery was found to be thrombosed. There had been no recurrence of gastric symptoms before the catastrophe. Unfortunately no histological examination of the thrombosed artery was undertaken, nor was the arterial supply of the stomach in the region of the perforation investigated. It seems possible that an ischaemic process here may have resulted in ulcer formation and perforation.

Discussion

In a period of little more than a year four cases have been seen in which a diagnosis of temporal or giant-cell arteritis has seemed justified. In two of these biopsy produced incontrovertible evidence, while the other two were clinically most suggestive.

I feel that the clinical picture is sufficiently characteristic to suggest the diagnosis. The patient is always in the latter part of life—no case under 55 has been reported. He may have been in good health prior to the gradual onset of a subacute illness in which fever, severe cephalalgia, anorexia, depression, and general toxæmia figure prominently, and arthralgia, myalgia, scattered lesions in the central nervous system, and interference with vision are often seen. Physical signs depend upon the situation of the disease, but tender thrombosing arteries about the skull are an almost constant finding. A low-grade secondary anaemia is common; a slight increase in white cells, sterile blood culture, normal blood urea, negative Wassermann reaction, but almost invariably raised blood sedimentation rate complete the picture. Cooke and his fellow workers (1946) do not stress the high sedimentation rate, but record that it was noted by Bowers (1940), Sprague and Mackenzie (1940), and Murphy (1942). In three of my cases the figures recorded are very high and did not return to normal until a considerable time after symptomatic cure.

In considering diagnosis, the age of the patient is against the possibility of periarteritis nodosa, in which condition younger individuals are attacked and in which the visceral vessels suffer severely. A high blood urea is almost constant and mortality is high. In temporal arteritis, though visceral vessels are by no means immune, they suffer less constantly, nitrogen retention is unusual, and mortality is relatively low. Buerger's disease, appearing again at an

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.