- Ellingson, R. J. (1964). "Studies of the Electrical Activity of the Developing Human Brain" in *The Developing Brain*, edited by W. A. Himwich and H. E. Himwich. Elsevier, Amsterdam.
- Faladé, S. (1955). *Le Développement psychomoteur du jeune Africain originaire du Sénégal au cours de la première année*. Foulon, Paris.
- Geber, M. (1956). *Courrier*, 6, 17.
- and Dean, R. F. A. (1957). *Lancet*, 1, 1216.
- (1964). *Courrier*, 14, 425.
- Grafstein, B. (1964). "Postnatal Development of the Corpus Callosum in the Cat," in *Neurological and EEG Correlative Studies in Infancy*, edited by P. Kellaway and I. Petersen. Grune and Stratton, New York.

- Pampiglione, G. (1956). *Proc. electrophysiol. Technol. Ass.*, 7, 80.
- (1963). *The Development of Cerebral Function in the Dog*. Butterworth, London.
- Purpura, D. P., Shofer, R. J., Housepian, E. M., and Noback, C. R. (1964). "Comparative Ontogenesis of Structure-function Relations in Cerebral and Cerebellar Cortex," in *Growth and Maturation of the Brain*, edited by D. P. Purpura and J. P. Schade. Elsevier, Amsterdam.
- Schade, J. P., and Pascoe, E. G. (1964). "Maturational Changes in Cerebral Cortex," in *The Developing Brain*, edited by W. A. Himwich and H. E. Himwich, p. 132. Elsevier, Amsterdam.

Medical Memoranda

Acute Massive Gangrene of Tongue

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Gangrene of the tongue with sloughing of the gangrenous area due to atherosclerotic arterial occlusion appears to have been reported only once previously in the British literature—by Bergan (1959). The rareness of the condition is because of the rich arterial blood supply of the tongue. The chief blood supply is by the lingual artery; however, the ascending pharyngeal artery and the external facial artery contribute deep branches to the tongue.

CASE REPORT

The patient, a woman aged 72 years, was seen on 6 December 1962. She complained of aching pains in the temporal areas bilaterally for four weeks. The pains appeared to radiate into the jaws and were associated with vague aches in the posterior cervical area; they were intensified by chewing. There were no significant findings on physical examination; the mouth could be opened fully. The temporal arteries were pulsating, palpable, but not tender. The blood-pressure was 140 mm. of mercury systolic and 80 mm. of mercury diastolic.

The patient was a known epileptic of long standing under control with phenacemide (Phenurone). In 1954 she developed mild exertional angina. In 1962 she was treated for sub-thyroidism with thyroxine.

The pains were thought to be due to a mild temporo-mandibular arthritis and treatment was started with local heat and oral salicylates. No improvement occurred and she was admitted on 14 December 1962 to St. Luke's Hospital for further investigations.

After admission she developed tenderness over the bifurcation of the carotid artery on the right side. Investigations showed a normal skull x-ray and radiographs of the temporor-mandibular region were also normal. Examination of the blood showed a haemoglobin of 12.1 g./100 ml.; white cell count 6,700 per c.mm. with a normal distribution. The erythrocyte sedimentation rate was 54 mm. in one hour (Wintrobe).

The patient had a low-grade pyrexia for the first four days after admission with a maximum reading of 99.2° F. (37.3° C.). She was treated with oxyphenbutazone 25 mg. six-hourly, with no significant improvement.

On 22 December she complained of difficulty in speaking and her speech was found to be slurred. It was felt that this was due

to a minor cerebral ischaemic episode. Muscle power, sensation, and co-ordination were normal, as were the deep nervous reflexes. This episode lasted a few hours and cleared completely.

During the subsequent week the patient was reluctant to chew as the pain intensified in the jaws and in the temples. On 28 December she complained of a sudden intolerable pain in the tongue and face; her speech became unclear. On examination there was cyanosis and coldness of the skin of the centre of the mandible; the lower lip, upper lip, and tip of the tongue were deeply cyanosed. The tongue began to swell rapidly. The pulsation of the temporal arteries had disappeared and the facial arteries could not be felt. Examination of the central nervous system was normal, the blood-pressure was unchanged. The pain was eased by intramuscular pethidine and the tongue became numb.

A diagnosis of vascular occlusion involving the lingual artery was made with reluctance, in view of the known vascularity of the tongue and lips. A bilateral stellate ganglion block was performed; intramuscular papaverine hydrochloride and intravenous heparin were started.

Twelve hours after the infarction good circulation was present in the lips and mandible; however, the tongue was mottled and grossly oedematous. A pulse could be felt in the temporal arteries. It was impossible to inspect the pharynx but the airway was clear. It was decided that carotid exploration was not indicated.

The patient was kept on oral fluids, oral antiseptics, Hibitane lozenges, intramuscular papaverine and penicillin, and intravenous heparin 5,000 units every four hours.

By the fourth day the tongue was grey but less oedematous. The patient was able to speak with difficulty and to swallow some fluids. She was free of pain. There was profuse salivation which made her mop her lips frequently. At the end of 10 days heparin was discontinued and phenindione therapy started. The prothrombin index was maintained at about 30%. The haemoglobin was 11.8 g./100 ml. and white cell count 7,100 per c.mm. The erythrocyte sedimentation rate was 34 mm. in one hour (Wintrobe).

The tongue was now a light brown colour, healthy vascular tissue could be recognized at its root, and posteriorly the infarcted area extended to within 1 cm. of the circumvallate papillae. The infarcted area started to slough from the edges (Fig. 1) and a gross oral foetor developed. The slough separated on the twentieth day aided by minor surgery, after which the tongue oozed blood and a blood transfusion of one pint (0.6 l.) of blood was given and anti-coagulants were discontinued.

The slough (Fig. 2) measured 4.5 cm. in maximum diameter and 4.3 cm. in maximum width. Microscopically complete necrosis was present.

Progress was subsequently uneventful. The patient could tolerate a denture two weeks after removal of the slough and she was discharged on 6 February 1963. A photograph (Fig. 3) taken three months after discharge shows the remaining stump of the tongue.

She has been closely followed for two years after the infarction, during which time she has remained in good health.

COMMENT

Acute ischaemic necrosis of the tongue with gangrene due to atherosclerosis appears to have been described only once previously in the British literature by Bergan (1959). His patient was a woman aged 80 years, who developed severe pain in the left side of the neck six weeks before developing pain in the tongue. Gangrene developed in a fashion similar to the

FIG. 1.—Sloughing areas shown lifted from underlying viable tongue.

FIG. 2.—Sloughed area of tongue.

FIG. 3.—Remaining tongue shown three months after patient's discharge.

present case and the gangrenous area was removed by surgery. Clinical examination suggested that there was a unilateral thrombosis of the external carotid artery.

Cases of sloughing of the tongue from any cause are very rare; thus a few cases have been reported associated with carcinoma of the tongue or the floor of the mouth, or due to acute inflammation with abscess formation. Bergan (1959) has reviewed these cases. Schönherr and Rumrich (1953—quoted by Bergan) reported a case of incomplete infarction of the tongue after arterial embolism in auricular fibrillation with mitral stenosis.

Howard and Cremin (1959) reported a case of "acute parenchymatous glossitis with gangrene of the tongue" in a woman aged 74 years. High fever and a grossly swollen tongue were part of the clinical picture, suggesting parenchymatous infection as the cause of the condition; however, Wise (1959) in a published letter suggested temporal arteritis as the basis of her gangrene.

Temporal arteritis appeared to be the cause of infective gangrene of the tongue reported by Brearley and McDonald (1961) in an 82-year-old woman. The temporal arteries were not palpable; however, both carotid arteries were pulsating. The E.S.R. was raised to 34 mm. in the first hour. The anterior two-thirds of the tongue were removed under general anaesthetic. Biopsy of the right temporal artery was carried out and showed changes compatible with temporal arteritis.

In the patient described above, the pains in the temples and face would appear to be manifestations of intermittent claudication of the temporalis and masseter muscles. The onset of the ischaemia suggests an acute vascular occlusion. The site of obstruction could not be ascertained precisely; however, because of the tenderness over the right common carotid artery it is suggested that the right external carotid artery or its branches were occluded. We did not feel justified in submitting the patient to carotid arteriogram.

Though precision is not possible, the likely sequence of events would appear to be that the right external carotid artery was incompletely occluded near its origin before the onset of symptoms. As the occlusion progressed pain developed in the muscles of mastication when they were in use. The acute episode would appear to have been caused by complete occlusion of the lingual and facial arteries on one or both sides, due to a spreading thrombus. As the erythrocyte sedimentation rate was raised before the development of infarction, it is impossible to exclude a localized arteritis. However, as there was no clinical or biochemical evidence of giant cell arteritis and the temporal arteries were pulsatile, we believe the occlusion was atherosclerotic in origin. This is supported by the fact that the patient has remained well for a follow-up period of two years. Despite the loss of the anterior portion of her tongue her speech is clear, though she has difficulty in the enunciation of some labial sounds. She is able to wear a denture and to swallow soft foods, but she continues to experience difficulty in chewing firm foods. Overall, she is in good health.

We would like to record our thanks to Dr. Janet Bowen, who carried out the stellate ganglion blocks, and to the nursing staff of St. Luke's Hospital. We wish to thank Mr. Reg Money, of Sydney Hospital, for his assistance with the clinical photography.

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REFERENCES

- Bergan, J. J. (1959). *Quart. Bull. Northw. Univ. med. Sch.*, 33, 38.
Brearley, B. F., and MacDonald, J. G. (1961). *Brit. med. J.*, 1, 1151.
Howard, S., and Cremin, M. D. (1959). *Lancet*, 2, 410.
Schönherr, K., and Rumrich, A. (1953). *Z. Laryng. Rhinol.*, 32, 11.
Wise, D. (1959). *Lancet*, 2, 674.