case, so that total parotidectomy can be proceeded with should the tumour be found to arise from the parotid gland. Lipomas may be superficial and arise in the fat between the parotid gland and the skin, but can also lie deep in the substance of the parotid gland. Sebaceous cysts may be mistakenly diagnosed when a cystic tumour of the parotid gland is present. Pre-auricular adenitis can cause difficulty, but the length of history is usually much less than in true parotid tumours.

The 47 cases (18 male and 29 female) were made up as follows: 3 chronic infection, 2 calculus, 1 tuberculosis, 1 oncocytoma, 2 adenolymphoma, 1 lipofibromatosis, 1 lipoma, 29 mixed parotid tumour, 3 carcinoma, 2 recurrent tumour, 1 congenital fistula, and 1 cyst, of branchial origin.

The carcinoma cases included here were those found to be so on pathological examination, but, except under unusual circumstances, preserving the facial nerve is not advised when the clinical appearance indicates malignancy.

The recurrent tumours occurred in cases in which a local removal of a mixed parotid tumour had been done elsewhere, and in both the recurrence showed microscopical evidence of malignancy.

Two other cases that were obviously malignant, and were not included in this series, gave a history of a local removal of a mixed parotid tumour many years previously. The recurrence rate after local excision or simple enucleation of mixed tumours has been variously stated by different authors to vary between 10 and 30%. Irradiation has no place in the treatment of these tumours, as it never causes the disappearance of the tumour, only a reduction in size. While the natural history of mixed tumours shows that they grow slowly over many years, they are liable to sudden increases in size and malignant change. In one case, not included in this series, a girl of 16 had a mixed parotid tumour treated by local excision, and when it recurred shortly afterwards radiotherapy was given. Within a few months it had ulcerated through the skin, and in spite of wide excision, including a block dissection of the neck, death occurred nine months later. Apart from the danger of recurrence, local excision is too often followed by partial or complete facial palsy.

Recurrence

The first case in this series was done in 1943 and the last in February, 1955, and there has been no recurrence in any of the primary cases, although two of the patients have since died of other diseases. The two recurrent cases previously mentioned, both adenocarcinoma after local excision of a mixed tumour, were treated by total excision of the gland, and the facial nerve was preserved. Two small tumours 4 mm. in diameter have been removed from one of these cases since, but there has been no further trouble during the last fourteen months. There have been no cases of permanent facial paralysis. Lower-lip palsy is present in a few cases, but was due to deliberate division of the lower branch of the cervico-facial nerve. This small series thus agrees with those of Redon (1953), of Paris, who, using a similar technique, reported 165 cases of mixed parotid tumour with no recurrence and no facial paralysis, and Patey (1954), in a recent review, also concurs.

Frei Syndrome

The scar—if made below the jaw, in the skin crease, and curved round the lobe of the ear to split the tragus—is unobtrusive. There is, however, one sequela which may come on from one month to two years after the operation, and that is the Frei syndrome. This consists of flushing and sometimes sweating on the same side of the face as the operation, and occurs during or after a meal. It occurred in 11 out of 45 patients followed up in this series, and varied in intensity from a minor degree of flushing and dampness to a considerable discharge of sweat. About the same incidence of this syndrome is given in other reports of this operation, and no really satisfactory explanation of its cause has been given. It does not disappear spontaneously and is not easily treated. It occurs chiefly in the malar area, which

is supplied by the sensory branch of the auriculo-temporal nerve. The auriculo-temporal nerve is also the motor nerve to the parotid gland, and it is thought that when the tongue is stimulated by hot or strongly flavoured substances the impulses pass via the glossopharyngeal nerve to the otic ganglion and thence to the auriculo-temporal nerve. The divided motor branches of this nerve then anastomose with the distal branches of the sympathetic system which supply the skin and which were severed when the skin flap was raised. Redon always ligates the external carotid artery, but has a 25% incidence of Frei syndrome. Although in this series it has rarely been necessary to tie this artery the incidence of Frei syndrome is about the same.

In one case in 1951 the nerve was identified and carefully preserved; this patient developed a Frei syndrome. Since 1951 an attempt has been made to identify the auriculo-temporal nerve, and in 10 cases in which it was thought to have been clearly identified and sectioned, as far proximally as possible, only one has developed a complete Frei syndrome and one has a flush only.

Conclusion

The results of this review allow the questions posed at the beginning of this paper to be answered as follows:

1. The recurrence rate for mixed parotid tumours can be reduced to nil by total parotidectomy, with preservation of the facial nerve.
2. The facial nerve can be preserved and will recover its functions in every case.
3. The cause of the Frei syndrome is not known, but deliberate identification and resection of the auriculo-temporal nerve may materially reduce the 25% incidence of this complication.

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Medical Memoranda

Perforation of Intestine by Unsuspectedly Swallowed Foreign Body

It must be quite unusual to find, during laparotomy, intestinal perforation caused by a foreign body the patient did not know had been swallowed. The following three cases occurred in the Royal Alexandra Infirmary, Paisley, during 1954.

CASE REPORTS

Case 1.—A woman aged 43 had rheumatoid arthritis, and was in the orthopaedic ward just recovering from an osteotomy of the neck of the femur, when she developed severe pain in the left side of the abdomen with nausea and slight pyrexia. On examination she had marked tenderness and rigidity in the left iliac fossa, suggesting local peritonism or peritonitis. Without rectal and vaginal examination, which could not be made owing to the orthopaedic condition, a twisted ovarian cyst seemed a possible diagnosis, and laparotomy was indicated. At operation there was a mild peritonitis with slight turbid exudate, and a splinter of wood 1½ in. (3.8 cm.) long was found perforating the middle of the small intestine. It was removed and the puncture holes—then almost invisible—were closed. Convalescence was uneventful. The patient did not remember swallowing the splinter.

Case 2.—A woman aged 47 complained of having had pain in the right iliac fossa for 24 hours, with nausea. Her temperature was 99.2° F. (37.3° C.). There was moderate tenderness and slight rigidity in the right iliac fossa, and the condition appeared to be acute appendicitis, but at operation the appendix was normal, and a splinter of wood 1½ in. (3.8 cm.) long was found perforating the bowel at

its mesentery, which was rather indurated. The foreign body was 6 in. (15 cm.) from the ileo-caecal junction. There was no evidence of diffuse peritonitis. The splinter was removed and the patient made an uneventful recovery. She had no recollection of swallowing the splinter.

Case 3.—A man aged 34, an epileptic, had had appendicectomy performed elsewhere in 1952. Since then he had constant, sometimes severe, pain in his scar, and by 1954 he was showing signs of an incisional hernia, for which he was admitted. At operation the scar tissue was peculiarly oedematous and appeared almost cartilaginous, and deep in the wound was found a spicule of bone $2\frac{1}{2}$ in. (6.3 cm.) long penetrating from adherent small intestine into the abdominal wall. Owing to the danger of infection, which subsequently developed, the hernia was not repaired at this time, but was dealt with successfully later. He had no recollection of swallowing this bone. The interesting point arises, whether or not the bone was the cause of the original complaint (as in Case 2 above). One cannot be conclusive, but the history of constant pain since the original operation is suggestive.

COMMENT

It is not suggested that this condition should be kept in mind in the differential diagnosis of every "acute abdomen," but it could be remembered with profit during laparotomy, when the pre-operative diagnosis proves wrong and nothing else can be found. If the foreign body be overlooked the abdomen may seem quite normal, as there may be no evidence at that time of diffuse peritonitis.

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Stokes-Adams Attacks in Bundle-branch Block

"People with heart disease do not faint." Exceptions to this valuable general rule are few. Severe aortic stenosis may be a cause of syncope of exertion, if the stenosis is severe and the heart muscle not too good. Auricular flutter may cause syncope from cerebral anaemia during short periods of 1:1 block, when the heart rate is raised to a high figure and the cardiac output becomes uneconomic. Myocardial infarction, where shock is severe, is a cause of unconsciousness. Since Stokes's original description in 1854, Stokes-Adams attacks have been a well-recognized cause of sudden, brief, and total unconsciousness. Stokes-Adams attacks are presumably due to involvement of the idioventricular pacemaker, at the upper part of the bundle of His, in the pathological lesion which is causing the heart-block. This association is well recognized in complete or partial auriculo-ventricular dissociation. There is no logical reason why such a pathological lesion should not be so placed in the bundle of His that it causes permanent bundle-branch block, transient heart-block, and infiltration of the auriculo-ventricular node to an extent sufficient to produce Stokes-Adams attacks during such transient heart-block. This possibility, so far as I am aware, was first envisaged and described in a communication I made to the British Cardiac Society in 1948, but which was not published. The following paragraphs summarize this communication.

Case 1.—This patient was a man of 70 who, for seven months previously, at intervals of a month or less, had frequent attacks of syncope. These were abrupt in onset, lasted three to five seconds, and were not followed by headache, drowsiness, or any other sequelae. He was just as well after the attacks as before. The heart showed moderate enlargement on cardioscopy. There was a harsh systolic murmur at the apex base. The heart rate was 56 and was regular. The blood pressure was 116/72. The cardiogram showed a P-R interval of 0.2 second and a prolonged QRS complex, with evidence of right bundle-branch block. He was given ephedrine, $\frac{1}{2}$ gr. (32 mg.) thrice daily, and from that day, a period of twenty-one months, has remained free from attacks.

Case 2.—A woman of 61 had a two-years history of from four to eight weeks' interval between attacks, the frequency of which had increased for a month or two previous to examination. The attacks were abrupt and were not fol-

lowed by sequelae, nor were they preceded by any warning. They lasted ten seconds, for the husband was in the habit of counting ten seconds, by which time he knew that his wife would recover. She had been thoroughly investigated for epilepsy. There was an aortic systolic murmur. The blood pressure was 150/90. The cardiogram showed an increased P-R interval of 0.23 second and bundle-branch block. Cardioscopy showed moderate enlargement. Further tracings taken to exclude a previous infarct were done, and in two of these a transient true heart-block, with dropped ventricular beats, was shown.

Both of these patients had a history of attacks of unconsciousness of abrupt onset, lasting for a few seconds only, and not followed by any headache, disorientation, drowsiness, or other sequelae suggesting epilepsy. In both cases there was evidence of a bundle-branch lesion. In each case the administration of ephedrine eliminated the attacks, which had previously been frequent. A third similar case is now reported.

CASE 3

A man aged 55, with no significant past or family history, had for three years previous to examination been conscious of a sudden feeling lasting for a few seconds, which would "well up" and cause him to feel dizzy and lost. In a number of them he had in fact abruptly and completely lost consciousness. He had stopped driving his car and riding. He had not suffered from dyspnoea, coronary symptoms, or other cardiovascular complaints. His work was journalistic and sedentary.

On examination he looked well. The heart rate was 60, the rhythm was regular, the blood pressure was 180/98. The heart sounds were clear and normal. There were no basal rales. The liver was not enlarged. There was no oedema of the legs or feet. The electrocardiogram was that of classical complete left bundle-branch block (see Fig.). Radiologically the heart was normal in size and shape, the transverse diameter by orthodiagraph being 13.3 cm.

Case 3. Electrocardiogram showing complete left bundle-branch block. Leads I, II, III, V_1 , V_2 , V_3 .

The diagnosis of Stokes-Adams attacks was made in an individual free from auriculo-ventricular dissociation, but with a left bundle-branch block. Ephedrine, $\frac{1}{2}$ gr. (32 mg.), three times a day after meals, was advised, the dose if necessary to be raised to 1 gr. (65 mg.) three times a day after meals. Five months later his general physician wrote as follows: "Ever since the ephedrine tablets have been taken regularly the attacks have stopped completely. Prior to this, of course, he had thirty to forty attacks during the previous two years. I thought you would like to have this interesting news of the patient."

COMMENT

The differential diagnosis of such cases, in view of the abruptness of the attacks and the complete unconsciousness, must lie between epilepsy, symptomatic or idiopathic, and unusually abrupt vasovagal syncope. The presence of a bundle-branch lesion should always rouse suspicion that the attack may be due to Stokes-Adams syndrome. In all three cases I have mentioned a therapeutic dose of ephedrine has both confirmed the diagnosis and freed the patient from an unpleasant symptom.

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