frontal bone. Compression of the right common carotid artery caused the bruit to disappear, and with it the sensation of noise in the head. There was no sensory change in the trigeminal field. The patient's appearance was as in Figs. 1 and 2.

FIG. 1.

FIG. 2.

At no time was pulsating exophthalmos present; the only fundus abnormality was dilated retinal veins. Fortunately in this case compression of the internal carotid artery did not provoke faintness or giddiness, so, according to Matas's test, operation for ligation of the internal carotid could be undertaken without undue concern.

Operation.—This was performed by Prof. Lambert Rogers on March 29, 1938. At this stage the patient was practically blind in both eyes. Under local anaesthesia the confluence of the three carotid arteries was exposed on the right side of the neck. The internal carotid was noticed to be larger than normal, approximating in size to the common carotid. The internal and external carotid arteries were ligated with silk; the buzzing ceased, and no untoward symptoms were noted. The wound was closed.

Progress.—Six hours later the patient was conscious and able to use both upper limbs. The buzzing in the head had ceased. On the following day she became drowsy, slow in speech, and incontinent of urine; the pupils reacted sluggishly, but the chemosis was less and the exophthalmos had diminished. Left facial weakness was now noted, and left monoplegia. The right plantar reflex was flexor and the left extensor in type. Later in the day the left leg was paralysed. She was now hemiplegic and stuporous. Recovery then gradually took place, and despite some residual hemiplegia she expressed herself well pleased with the result of the operation, since she could now see and the buzzing in her head had ceased. It had previously been most distressing, and had kept her awake at night. Five weeks after the ligation her appearance was as shown in Fig. 3.

FIG. 3.

FIG. 4.

In May, 1938, her vision was: right 6/6, left 6/6; the only abnormalities were a very slight exophthalmos in the right eye and dilated conjunctival veins. I still see her at intervals. There is no change in her eye condition and little change in her hemiplegic condition; she does her housework and goes for long walks. Fig. 4 is a recent photograph.

Probable Pathological Condition

The pathological condition present in this case can only be surmised from the signs and symptoms which were present in the 31 cases mentioned in the literature on this subject in which necropsies were performed. Charcot established the fact that in no part of its course is the internal carotid so thin-walled as in its passage through the cavernous sinus. This portion of it has little support; it is surrounded by the cavernous venous channel, and is consequently liable to aneurysmal development. In this case I will presume that the aneurysm was on the point of bursting; the patient sneezed violently, and the aneurysm burst into the cavernous sinus, the resulting carotid fistula accounting for the immediate pulsating noise in the head. The cavernous sinus dilated because of the consequent arterial pressure within the venous sinus. There was damming up of the venous blood return from the right eye, with consequent exophthalmos. The enlarged cavernous sinus pushed forward the internal carotid artery and bent the optic nerve, with consequent loss of vision. The total ophthalmoplegia and ptosis were due to the kinking of the third, fourth, and sixth nerves in their passage through the dura, and to the direct pressure of the dilated sinus. The vicious back-pressure in the dilated sinus caused dilatation in the intercavernous sinus; then the left cavernous sinus dilated, and as a consequence there followed exophthalmos, ophthalmoplegia, ptosis, chemosis, and loss of vision in the left eye.

Immediately after the external and internal carotid arteries on the right side were ligated the head noise ceased, vision improved, and the other signs gradually disappeared, leaving no doubt that this was a case of right carotid cavernous arteriovenous fistula.

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Medical Memoranda

Thyroxine as a Practical Treatment of Myxoedema

It is well known that thyroxine is as effective as thyroid extract in the treatment of myxoedema. Nevertheless it is seldom used in practice, for it is more expensive than thyroid extract and is poorly absorbed when given by mouth. As thyroid extract could not be tolerated in the following case, injections of thyroxine were given. Our purpose is to describe a possible method of using thyroxine in such cases. Although we do not advocate its employment as a routine treatment of myxoedema, we think it worth recording that it is a practicable alternative.

CASE REPORT

The patient, a married woman aged 56, was first admitted to University College Hospital in August, 1938, having had diarrhoea and vomiting for four months. On admission she was found to be an obvious case of myxoedema, with a dry skin, thin hair, husky voice, and slow cerebration. Her basal metabolic rate was -53%, and while in hospital her pulse rate varied between 58 and 82. On three occasions thyroid extract was given by mouth, in doses varying from 0.5 grain twice daily to 1 grain three times daily, but each time the treatment had to be stopped owing to persistent vomiting.

She was readmitted to hospital in March, 1941, having had an attack of oedema affecting the upper part of her face, including the eyelids, after taking three tablets of thyroid extract. She also complained of pains in the arms, thighs, and calves. Her condition was about the same as it had been when she was first seen, but she had put on a stone in weight. The basal metabolic rate was -49%.

Intravenous injections of thyroxine were started after the patient had been in hospital two weeks, during which time her pulse rate varied between 56 and 64. At the first injection 1 mg. of thyroxine was given. As this had no ill effect the dose was increased to 3 mg. the following day and to 5 mg. a week later. There was no reaction following this larger dose. Three days later the basal metabolic rate was found to be -13% . The basal metabolic rate was estimated at weekly intervals, and three weeks after the last injection it had fallen to -30% . 7.5 mg. of thyroxine was then given, and a week later the basal metabolic rate was -1% . It fell steadily during the subsequent three weeks and again reached -30% . The next injection was increased to 10 mg. of thyroxine, but the following day the patient complained of pains in all her limbs, palpitations, and diarrhoea. These symptoms lasted from two to three days. It was considered that 10 mg. was too large a dose, and she has since been given 7.5 mg. at four-weekly intervals. During the first four weeks of treatment her weight fell from 9 stone to 8 stone, at which it has remained fairly steady. Her pulse rate was running between 70 and 80 when she was discharged from hospital in July. Since then she has been attending as an out-patient every four weeks for an injection. When last seen her condition was satisfactory. She is active at home and is much more lively than she was, her skin is of normal texture, and she has a quantity of new hair. She feels well, and seems unaffected by the fluctuations in her basal metabolic rate.

COMMENTARY

Why this patient was intolerant of oral thyroid extract but not of injections of thyroxine is not clear. However, our purpose is less to describe the case than the method of treatment. It will be seen that the technique employed was to find out the highest single dose of thyroxine that could be given without ill effect, with the object of spacing the injections so far as was possible. This was found to be 7.5 mg. If the daily utilization in a myxoedematous person is taken to be a little less than 0.3 mg. (Salter, 1940), this dose should last four weeks; weekly determinations of the basal metabolic rate showed that this was in fact the case. The treatment was entirely satisfactory from the patient's point of view, and can be recommended as a suitable method of using thyroxine when thyroid extract cannot be taken by mouth.

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Amoebic Liver Abscess in Northern Ireland

Amoebic liver abscess in Europeans is uncommon nowadays, even in the Tropics. Before the last war it was the commonest cause of death among British troops serving in India. Since the recognition by Leonard Rogers of the pre-abscess stage of amoebic hepatitis and the introduction by him of the emetine treatment of amoebiasis, it has lost its terrors and has become comparatively rare. The case of a soldier serving in Northern Ireland seems worth recording, especially on account of the long interval of perfect health since serving abroad and the complete absence of any history of amoebic infection.

CASE REPORT

The patient, a Regular soldier aged 29, had a history of uninterrupted good health before entering the Army and during his Army service until the middle of February, 1941. He went abroad in December, 1935, served one month in Cairo and eight months under canvas at Mersa Matruh. He returned to Cairo for one month and came back to England. This was the only time he had ever been abroad. His health had always been excellent, and he played football, swam, and rode a motor-cycle regularly. He had never suffered from dysentery of any kind, nor had there ever been any clinical or bacteriological evidence of amoebic infection. He drank a moderate quantity of beer, but rarely took spirits. During the latter half of February he was laid up in his lodgings, in a village in Northern Ireland, with an attack of influenza. After a week's leave he rejoined his unit but never recovered his strength or his appetite, and he gradually lost weight.

He was admitted to a military hospital on April 13, with pain in the upper abdomen, pyrexia, and slight enlargement of the liver. He rapidly lost weight, the pyrexia persisted, and the liver continued to enlarge. On May 25 he was transferred to my hospital. He was then gravely ill, very emaciated, and had a dry furred tongue; his complexion was dark brown. The

Antero-posterior and lateral views, showing right dome of diaphragm very high. A large cavity, with scalloped walls and fluid level, is demonstrated in the right lobe of the liver. There is a hydropneumothorax with a fluid level on the right side, and the upper lobe of the right lung is partially collapsed.

temperature was 100° , pulse 110, and respirations 28. The liver was enlarged to near the umbilicus and was soft and tender. Liver dullness extended upwards to the nipple level. Breath sounds at the right base were feeble, but there were no adventitious sounds. Over the intercostal spaces neither tenderness nor oedema was present, but there was definite tenderness