

looked, whilst the chemist is being deprived of what should be his chief means of livelihood.

There is a close connection between the Medical and Pharmaceutical professions which should make for co-operation and mutual helpfulness. Instead there exists, far too often, misunderstanding and even antagonism, between the members of the two professions. It should, therefore, be our aim to try to foster a better spirit and this can best be done by an appreciation of the other man's value, his rights, and of his point of view.

THREE CASES OF PARALYSIS OF THE FACIAL NERVE.

R. J. FLEMING.

During August 1936, three cases of facial paralysis were admitted to wards 7—8, N.E.H. The Facial Nerve was involved at different levels in the three cases. In view of the relative frequency of facial paralysis and the importance of estimating the level of the nerve lesion we feel justified in quoting these cases and illustrating them with an explanatory diagram.

Peripheral Lesion.

Case 1.—Peter, aged 33 years, a Non-European labourer was admitted to hospital in August, 1936, complaining of a weakness of the right side of his face.

Three months before, he found that he could not lift the right angle of his mouth nor close his right eye.

The weakness gradually increased, and a month later he noticed a small painful swelling just below the lobe of his right ear. After a month the pain disappeared but the paralysis of the face remained.

On admission the right side of the face was paralysed.

- (a) The right cheek was flaccid and ballooned out when he tried to whistle.
- (b) He could not close his right eye and, on attempting the movement, the eyeball rotated upwards.
- (c) He could not wrinkle the right side of his forehead which was smooth and devoid of lines.
- (d) Taste and salivary excretion were normal.

A hard, round, regular, immovable mass, the size of a walnut, was found situated below the lobe of the right ear.

Diagnosis:—A tumour of the Parotid, involving the peripheral portion of the seventh nerve after its emergence from the stylo-mastoid foramen.

Bilateral Peripheral Facial Paralysis.

Case 2.—Sixpence Saktsu, a Shangaan labourer, aged 56, was admitted to hospital on 21st August, 1936, in an unconscious state. It was learned that he had fallen 15 feet on to his head. He was bleeding slightly from the nose, but profusely from the right ear, dressing after dressing being saturated with blood.

Pupils, small and equal. Pulse 60. Breathing through the mouth caused the right cheek to be blown out at each expiration. The right eye remained open.

He remained unconscious for six hours when he demonstrated a peripheral lesion of the right facial nerve.

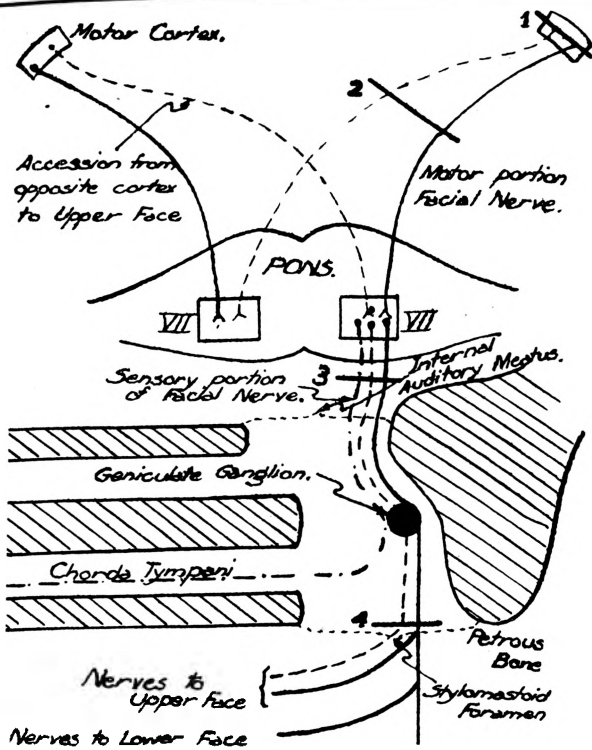
He was deaf on the right side. Seven days later, he developed a paralysis of the left side. He now had a bilateral peripheral facial nerve paralysis as shown by:—

- (a) He had a mask-like expression. The furrows of his 56 years had vanished in a night.
- (b) He could not whistle or blow out his cheeks because he could not bring his lips together.
- (c) He could close neither eye and on attempting to do so the eyeballs rotated upwards.
- (d) He could not wrinkle either side of his forehead.
- (e) Food collected between his cheeks and gums.
- (f) There was loss of taste sensation of the anterior 2/3 of the right side of his tongue.

Three weeks later the paralysis of the left side of the face cleared up, but the right side remained the same.

This demonstrated that a facial paralysis, coming on some time after the injury to the base of the skull has a better prognosis than an immediate paralysis. In the former the paralysis is due to pressure on the nerve by a blood clot or oedema, and in the latter the nerve is torn.

Case 3.—Esau Sane, a native, male, aged 30 years, was admitted to hospital 25th August, 1936, two days after he was assaulted and beaten on the head with sticks.



The Connections of the VII. Nerve from A Synopsis of Surgical Anatomy. Lee MacGregor (modified).

He had found his way to his room and the next morning he could not use his left arm or move the left side of his face.

He stated that he had bled from the right ear.

On admission it was found that he had a left facial paralysis but he could

- (1) Close both his eyes.
- (2) Wrinkle both sides of his forehead.
- (3) Taste was normal.

He had also a complete flaccid paralysis of his left arm, but sensation was unimpaired. The rest of his body was normal. His C.S.F. pressure was 150 cm. of water and the fluid bloodstained.

In the course of the examination he had a Jacksonian fit. His left and paralysed arm first became rigid and was raised. The rigidity rapidly spread to the rest of the body and was followed by generalised clonic contractions lasting one minute. These gradually subsided, leaving the patient exhausted.

X-Ray showed a fissure fracture of the temporal bone.

In emotional states, e.g., laughing, the paralysis of the face was less evident—in fact

it was difficult to realise that one side of the face was paralysed.

This was in contradistinction to Peter who had a peripheral nerve lesion and whose paralysis was accentuated on laughing.

A cortical lesion was diagnosed and confirmed at operation when a small laceration of the motor cortex was seen accompanied by subdural and extradural clots. 24 hours after the operation he could move all the muscles of his left arm except the extensors of the fingers and wrist. The paralysis of the face persisted.

Summary.

Reference to the diagram of the facial nerve will give the explanation of the clinical findings in lesions at different levels of the facial nerve.

We know that the level of the lesions in these cases are as follows:—

(a) Peter.—The nerve is affected after its exit from the stylo-mastoid foramen.

(b) Sixpence.—The right nerve was damaged in its course through the base of the skull on the right side and the left nerve at the base of the brain by a blood clot.

(c) Esau.—A cortical lesion.

(a) Peter.—We see from the diagram that lesion affects the whole of the peripheral nerve including the branch supplying the upper face, but the chorda tympani is not affected and we would expect taste to be normal as indeed it was found to be.

(b) Sixpence.—The lesion through the petrous portion of the temporal bone affects the whole of the motor supply to the face as well as the chorda tympani and, in this case, we found a complete paralysis of the upper and lower portions of the face as well as interference in taste of the anterior 2/3 of the tongue.

(c) Esau.—A cortical lesion does not paralyse but only weakens the upper face muscles, i.e., the frontalis and the orbicularis oculi as these muscles are supplied by both sides of the brain. Only a lesion below the level of the pons will affect the innervation of these muscles.

The chorda tympani fibres accompany the facial nerve only from the pons and a cortical lesion will not affect them.

Conclusion.

Cortical lesions of the seventh nerve cause a lower face paralysis, the upper face muscles being but partially affected and the sense of taste not at all.

Emotional movements of the face are less affected than voluntary movements.

Lesions of the seventh nerve below the pons cause a peripheral paralysis of the whole of that side of the face and if the lesion is above the chorda tympani branch, taste will be unaffected.

A CASE OF DIABETES MELLITUS IN ASSOCIATION WITH HYPERTHYROIDISM

E. M. TOMLINSON.

Mrs. V., aged 48, housewife. Admitted to Ward 23 on September 5th, 1936.

Previous Health—Has been ill for 13 years (see below).

Habits—Diet, eats anything. Sleep, fair. Weight, 120 lbs. on admission; 151 lbs in 1935.

Family History—Mother has asthma.

Major Complaints—

1. Pain on passing water.
2. Lump in neck.
3. Choking feeling.
4. Pain in stomach.

History of present condition—

Up to 13 years ago, 1923, the patient was quite well. At this time she had a child and, after his birth, she was troubled with a burning pain on passing water. She also had a sick feeling in her abdomen before and after her periods, but it had no relation to meals. She was told by a doctor that it was due to "womb trouble." She was curreted but there was no change in her symptoms.

Four years later, 1927, she started having pain in her left side; it lasted five minutes and then disappeared; it comes on only if she works hard, it has no relation to meals and has not been associated with vomiting.

The patient first noticed the swelling in her neck 27 years ago, at the age of 21, following the birth of her first child. She has had seven children and after childbirth the swelling has increased in size.

Eight years ago, 1928, she began to have a choking feeling in her throat and was unable to lie down. At this time she had X-ray treatment to her neck for two months and, although it did not decrease in size, the choking feeling went away. She was then put on iodine for about six months, on and off.

On May, 1935, she was told she had sugar in her urine and was on insulin 30 units daily until January, 1936. Iodine treatment was given again for six months, last year.

There was no change in her condition until July 1936, when she fainted in town, losing consciousness, and was told that her heart was enlarged due to the sugar.

She was in bed one month and, on getting up, noticed her feet were swollen after her first day up. At the present time, her feet swell up if she walks a lot. She has had palpitation since July, and gives a history of failing exercise tolerance, and increased breathlessness on exertion. She sleeps with four pillows on account of the swelling of her neck.

The patient prefers winter to summer. Her hair has been falling out for years; her skin has become dry.

During the last few years she has noticed that she is irritable and easily upset by trivialities.

Her voice has been husky since July, 1936.

She gives a history of boils, pruritis vulvae and whitlows for 13 years.

Condition on examination—

The patient is an emaciated, elderly woman.

Neck—

Bilateral swelling of thyroid, nodular, with two calcified nodules, moves on swallowing; not attached to superficial or deep structures; pulsation present.

Cardiovascular system—

Heart enlarged, ratio $5\frac{1}{4}$ to $9\frac{1}{4}$ by X-ray; 2 normal sounds, rhythm regular. B.P. 148/90. Pulse about 96.

Eyes—

No exophthalmos, no lid lag, wrinkling of forehead and convergence present.

Special investigations—

7/9/1936—Blood urea, 31 mg. per 100cc.

7/9/1936—Blood sugar curve, 0.13%, 0.15%, 0.2%, 0.18%, 0.13%. Sugar present in specimen of urine.

9/9/1936—B.M.R., plus 46%. Pulse 114.

8/9/1936—X-ray chest, peribronchial fibrosis throughout both lung fields; heart enlarged, ratio $5\frac{1}{4}$ to $9\frac{1}{4}$.

Diagnosis—

Diabetes and hyperthyroidism.

Discussion—

In cases of hyperthyroidism there are often signs of a disturbance in carbohydrate metabolism, resulting in glycosuria and sometimes