FACIAL PALSY*

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One of the clinical lectures given by Sir William Gowers (1895) at the National Hospital for Nervous Diseases, Queen Square, London, was on facial palsy, and it included the following diagnostic rule: "Paralysis of all the muscles supplied by the facial nerve on one side only, and without other symptoms, always means disease of the nerve trunk. Practically, moreover, if it occurs without obvious disease or injury near the nerve after it emerges, it means disease of the nerve as it passes through the bone." Now that last word, bone, refers of course to the temporal bone, which is why the aural surgeon has a particular interest in the problems presented by infranuclear facial palsy.

The neatly rounded canal in the temporal bone through which the facial nerve runs for more than 30 mm.—a record distance for any nerve in a bony canal—led to its being called an aqueduct by Fallopius, the anatomist of the great Padua school. It might well be thought that such a course would be a protection against injury or infection, but in fact the nerve is unusually vulnerable in this situation, which is why the facial nerve is more often paralysed than any other motor nerve in the body.

One reason for the frequency of facial palsy is the close relationship of the nerve to the various parts of the auditory apparatus round which it is wrapped during its course through the temporal bone, and whose diseases and injuries it may be called upon to share. But the principal reason is that the facial nerve, its blood and lymphatic vessels, and its sheath fill the Fallopian canal in the temporal bone so that there is no room for expansion. Therefore, if from any cause a segment of the nerve or of its sheath swells up, then it can only do so at the expense of some of the structures which already fill the canal. Because of their fluid content the blood vessels are the most susceptible to pressure of all the soft tissues in the Fallopian canal, so that any swelling of the nerve or of its sheath in the canal is apt to squeeze the blood vessels, causing an ischaemia. This in its turn encourages swelling of the nerve above the zone of ischaemia, which further deprives the nerve of its blood supply, and this may be sufficient to cause a block in the conduction of impulses down the nerve.

Now we know from the work of Weiss (1943) and of Denny-Brown and Brenner (1944) that nerve fibres cannot survive for any length of time if deprived of oxygen, which is, of course, what happens when the blood supply is cut off; and it is this vulnerability of the facial nerve trunk and its attendant vessels to changes in pressure during their long journey in the bony canal in the temporal bone that accounts for the frequency with which the facial nerve is paralysed.

Some idea of the incidence of facial palsy can be gained from the morbidity statistics of Stocks (1949) based on an inquiry into the causes of illness in some 38,000 patients (Table I).

TABLE I.—*Monthly Prevalence Rate (Per 100,000) of Facial Palsy and Disorders of Comparable Frequency*

Thyrotoxicosis	14
Bronchiectasis	13
Facial palsy	12
Psychoses	10
Nerve injuries	9

This supports the view that facial palsy is by no means an uncommon condition; though of course it cannot compete with conditions such as deafness, with a monthly prevalence rate of 2,812, or chronic bronchitis, with 3,110.

We have been able to trace adequate records of 557 cases of isolated nuclear or infranuclear facial palsy from among those which have been referred to us during the past few years through the kindness of the colleagues of one of us (T. C.) at the National Hospital for Nervous Diseases, Queen Square; King's College Hospital; and elsewhere. These are set out in Table II. It will be seen that two-thirds have been included under the heading of Bell's palsy; but before dealing with this large group we would like to say something about the other groups.

TABLE II.—*Causes of Peripheral Facial Palsy*

Bell's palsy	347
Injury	84
Geniculate	39
Infection	27
Neoplasm	22
Spasm	21
Nuclear	11
Birth injury	6

Injury

Injury accounted for 84 cases, or nearly 15% of the total, and in half of these the damage was inflicted during the course of an operation on the mastoid, usually for infection. Fortunately, the incidence of damage to the nerve in the course of mastoid operations is on the wane. One reason for this is that in the newer forms of temporal bone operation such as fenestration, though the surgeon is working much nearer the nerve than formerly, it is in clear view most of the time. During the course of an operation on the ear, damage to the facial nerve is most likely to happen when the nerve is out of sight and when in all probability the surgeon does not appreciate that he is near the nerve. Also, since the advent of the antibiotics the number of operations upon the mastoid for acute infection has dwindled to less than one-tenth of its former total; and such operations used to provide most of the damaged nerves.

Some form of war injury accounted for a quarter of the group, and in more than half of these the nerve was damaged after its exit from the stylomastoid foramen. That more cases of war injury were not seen is because only those cases in which the missile causing the damage to the nerve struck the mastoid tangentially were likely to survive.

A head injury with skull fracture accounted for 16 cases, and seven of these were submitted to operation. In each case a fracture line was found running through the facial canal, the nerve itself being torn across in two cases and badly bruised in five. Facial palsy following a skull fracture is not uncommon, and, as Aldren Turner (1944) has shown, spontaneous recovery is the rule, particularly when the onset of the palsy is delayed. In such cases the nerve will be intact though bruised. When, however, the bony canal is traversed by the fracture line then the nerve may be seriously damaged. In six out of the seven cases submitted to operation there was a dislocation of the incus, a state of affairs

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which could be diagnosed before operation. We would say, therefore, that in a case of skull fracture with facial palsy, dislocation of the incus, as evidenced by conductive deafness and an abnormal appearance of the tympanic membrane, is a definite indication for exploration of the nerve trunk in the Fallopian canal. In other cases the deafness may be due to a fracture into the bony labyrinth, in which case it will be perceptive. Here again there may be severe damage to the nerve trunk which may require exploration to put it right. It has sometimes been thought that deafness and facial palsy accompanying a fractured skull mean that the seventh and eighth nerves have been damaged at the internal auditory meatus; but as the bone at this part is thick and strong it is rarely if ever included in the line of fracture, which prefers the thinner bone of the tegmen tympani and underlying labyrinth and middle ear.

Geniculate

This group includes all the cases of facial palsy in which the lesion is believed to be in the region of the geniculate ganglion as shown by impaired lacrimation as well as loss of taste.

In some there is a herpetic eruption on the pinna and neck, preceded by severe pain and often associated with partial deafness and vertigo, due, it is believed, to involvement of the adjacent eighth nerve ganglia. These various forms of palsy with herpes were described by Ramsay Hunt, whose name is attached to the syndrome of herpes and facial palsy with or without deafness and dizziness. Sometimes, however, there is no herpes and the eighth nerve is normal, and within the last few years we have included several cases in the geniculate group because lacrimation was defective—cases which otherwise would have been regarded as Bell's palsy. Absence of secretion from the lacrimal gland does not necessarily mean a dry eye, as the conjunctiva is partly lubricated by mucous glands not under the control of the secreto-motor fibres passing through the geniculate ganglion. Therefore, unless a specific test of the ability to lacrimate is carried out, deficiency due to involvement of the geniculate ganglion can easily escape notice. We use a modification of Schirmer's (1903) test in which a narrow strip of filter paper is hinged on the lower lid of each eye so that it hangs down, and the patient then inhales ammonia. The resulting lacrimation can be compared on the two sides by the speed and extent to which the strip of filter paper is moistened by tears. We found that when ammonia is inhaled up only one nostril lacrimation is always more profuse from the eye on the same side as the stimulated nostril.

Infection

This group is smaller than might be expected because we have excluded from it a number of cases of fleeting palsy with an acute otitis media, and also cases of primary cholesteatoma, which have been placed under the neoplastic group.

Also not every case of facial palsy associated with otitis media has been recorded, and we have the impression that the real incidence of facial palsy with otitis media is much higher, possibly double that recorded in this series.

The cause of the palsy in infective cases is usually an extension of a chronic infective process from the middle ear by erosion into the Fallopian canal. In some the labyrinth may also be diseased, and in such cases recovery of facial movements does not always follow surgical removal of diseased bone.

Neoplasm

This group includes 12 cases of primary cholesteatoma or intratemporal dermoid, as named by Jefferson and Smalley (1938), two cases of neurofibroma of the facial nerve trunk itself, and one glomus jugularis tumour. In every case the onset of the palsy was gradual, and in one it took two and a half years to develop. The remainder were due to malignant tumours of the parotid gland or in the temporal bone.

Neoplasms involving cranial nerves other than the eighth have not been included in this series, nor have cases of facial palsy associated with acoustic neurofibromata.

Spasm

Clonic facial spasm is a distressing and by no means rare condition. It has been included here because in many cases there is usually some slight paresis as well.

We have submitted 13 of these cases to decompression, but in none was any lesion noticed in the nerve as it lay in the Fallopian canal. Guarded trauma to the nerve by squeezing with fine dissecting forceps is followed by paresis for a few weeks and cessation of the spasm. In a few the spasm has been relieved for more than 18 months, but in many it has returned within that period.

The squeezing of the nerve under direct vision is, we think, safer than blind injection of alcohol and certainly more effective; and it does not carry with it the risk of irreversible palsy. However, it is not by any means the final solution. We are at present deliberately cutting the nerve trunk in the Fallopian canal and then allowing it to regenerate. This, of course, stops the spasm, and normal facial movements reappear within a few months. It is as yet too early to say whether the relief from spasmodic movements is permanent.

Nuclear

This group includes cases with an isolated episode during the course of disseminated sclerosis, and also cases of poliomyelitis. The clue to the latter disease is considerable constitutional disturbance with complete facial palsy, without, however, any loss of taste or lacrimation, as the nerve fibres carrying these impulses separate from the facial nerve trunk proximal to the geniculate ganglion.

At this stage it will be convenient to consider the differential diagnosis between lesions of the facial nerve trunk in the distal part of the Fallopian canal, into which group all cases of Bell's palsy are placed, lesions at the geniculate ganglion, and lesions in the facial nucleus itself (Table III).

TABLE III.—*Peripheral Facial Palsy: Differential Diagnosis*

	Bell's	Geniculate	Nuclear
Taste	Often lost	Lost	Normal
Lacrimation	Normal	,,	,,
Eruption	None	Usual	None
Pain	Often	Severe	,,
Hearing and balance	Normal	Often affected	Usually normal
Constitutional symptoms	None	Usual	Usual

Bell's Palsy

Under this heading are included all cases of peripheral facial palsy for which no cause can be found. Again, to quote Gowers (1893), who, referring to Bell's palsy, wrote: "The features of these cases are so uniform, allowance being made for difference in degree, that we are justified in regarding the pathological condition as the same in all—a neuritis within the Fallopian canal."

It is generally agreed that the exciting cause of Bell's palsy is an ischaemia, and Kettel (1947, 1954) has found that in some cases there are changes in the bone near the canal. He believes that most cases of Bell's palsy are due to ischaemia of the facial nerve near the stylomastoid foramen. Following lack of blood supply the nerve loses its power of conduction and venous stasis arises with ensuing swelling of the nerve, which becomes secondarily compressed within the facial canal. Hilger (1949) suggests arteriolar spasm, and Sullivan and Smith (1950) have demonstrated that such an effect can follow exposure to cold. While in some cases cold may well play a part, it cannot be the only cause; or, indeed, it may not by itself cause palsy but may need some other unknown predisposing factor to make the nerve susceptible to cold, and Hilger believes that there may be a defect in the autonomic nervous system.

Other titles for Bell's palsy include rheumatic palsy, refrigeration palsy, and "paralysis e frigore." Though each of these aliases suggests an aetiology, the cause is, we feel, still in some doubt. Therefore we prefer the name Bell's palsy, which does not commit one to a definite cause, but it does recall the memory of Sir Charles Bell, the surgeon and anatomist, who established among other things the function of the facial nerve which he described as the "respiratory nerve of the face" (Bell, 1821), for he held that facial movements were primarily an accessory to respiration. His interest in the facial nerve soon led to this name being attached to all cases of facial palsy. As time went by, certain definite causes, such as injury and infection, were recognized and named, and it is possible that as our knowledge and experience is extended the large proportion of cases of facial palsy still named after Bell will be reduced as new causes are discovered.

As the condition is not in itself fatal, the opportunity for detailed histological examination rarely presents itself, though there are new accounts by Kettel (1954) and Jongkees (1954) of the histological appearance of the nerve showing degenerated nerve fibres, swollen myelin sheath, but no sign of inflammation.

There is a lot to learn from examining the nerve under the binocular dissecting microscope as it lies in the Fallopian canal. Ballance and Duel (1932), who did so much to arouse interest in the facial nerve, described a swelling of the nerve in the lower part of the Fallopian canal in cases of Bell's palsy. Cawthorne (1946, 1952), using the binocular dissecting microscope giving 10 diameters of magnification, noted an abnormal appearance of the nerve just above the stylomastoid foramen, where it was often constricted, with swelling of the nerve trunk above the constriction and haemorrhagic streaks and patches of discoloration in the nerve. The opportunity for inspecting these cases under the dissecting microscope has been extended, and we have at times found swelling and areas of discoloration in the nerve much higher up in the vertical portion. In two in which the geniculate ganglion was thought to be affected the nerve trunk adjacent to the ganglion was found to be swollen and discoloured. In nearly all the cases of Bell's palsy submitted to decompression there has been an obvious lesion localized to one small segment of the nerve trunk and almost always in the descending portion.

However, to return to the clinical features of Bell's palsy, there was no significant difference between the sexes (males 169, females 178), though the females are slightly in the lead. If exposure to cold was a regular aetiological fact one might have expected a preponderance of males. The age incidence is shown in Table IV. Nearly 70% were found between the ages of 21 and 50.

TABLE IV.—*Age Incidence in Bell's Palsy*

Age at Onset	No. of Cases	Age at Onset	No. of Cases
0–10 years	3	41–50 years	82
11–20 ,,	30	51–60 ,,	51
21–30 ,,	71	61–70 ,,	28
31–40 ,,	77	71–80 ,,	5

There were 184 right-sided cases and 161 left-sided. Only two bilateral cases were seen, though in 33 patients there had been more than one attack. One patient who had had no fewer than five attacks told us that her brother had suffered three attacks, and we were able to establish that both her brother and her sister had extremely cellular mastoids. This extensive cellular system in the mastoid was noted in all recurrent cases. Possibly the nerve trunk in such patients is more susceptible to thermal changes.

Prognosis in Bell's Palsy

A question that patients with Bell's palsy never fail to ask is, How long will it be before the facial movements are fully restored? It is said that 80–85% of cases will recover completely and unaided. This may well be so, but this high proportion of complete recoveries is due in no small measure to the fact that many are incomplete from the start. Table V shows the relationship between the extent of the palsy and eventual recovery in a series of cases which was observed (without surgical treatment) over a period of at least six months. In this group, when the palsy was complete the chance of full recovery was 42%, whereas when it was incomplete the chance of recovery was 85%. In the complete group 6% showed no sign of recovery after six months.

TABLE V

Complete Bell's palsy		111
Full recovery	47	
Partial ,,	57	
No ,,	7	
Incomplete Bell's palsy		67
Full recovery	57	
Partial ,,	10	

The other important factor in assessing the prognosis is the electrical activity of the nerve and of the muscles it supplies. This can be examined by testing the ability of the nerve trunk or of the fine nerve endings in the muscle to transmit effective electrical stimuli of short duration. The contraction of the muscles can be observed clinically or by electromyography.

A nerve which is cut off either anatomically or physiologically will gradually degenerate and in doing so will lose its power to transmit an effective electrical stimulus. This gradual loss of electrical activity may take two or even three weeks, so that with the present tests available it is not possible to tell for certain within three weeks what is happening to the nerve.

At the end of that time, however, the electrical activity of the nerve is a good index of the severity of the lesion. This is well shown in Table VI, in which the term "faradic response" is used, though in many the tests were recorded as intensity duration curves.

TABLE VI.—*Response to Electrical Stimulation in Bell's Palsy*

Faradic response normal		74
Full recovery	70	
Partial ,,	4	
Faradic response reduced		62
Full recovery	31	
Partial ,,	31	
Faradic response absent		42
Full recovery	3	
Partial ,,	32	
No ,,	7	

Thus we find that when there is a normal response to electrical stimulation the expectation of full recovery is 94%. If, on the other hand, the nerve will not respond to electrical stimulation at all, then the chance of full recovery drops to as low as 7%. It is upon the result of these and other electrical reactions that the decision to decompress the nerve in a case of complete Bell's palsy is made. If there is no response to electrical stimulation then decompression of the nerve in the descending part of the Fallopian canal is indicated. Ideally the decompression should be carried out as soon as possible after the onset of paralysis, but so far we do not have any means of telling early which cases are unlikely to recover spontaneously and so we must wait at least three weeks for the electrical tests.

Electrical reactions are, of course, carried out on all cases of facial palsy, and upon these reactions and upon the cause of the paralysis depends the decision to explore the facial-nerve trunk at the supposed site of the lesion and, depending upon what is found, to relieve pressure, to bring about an end-to-end anastomosis, or, if a gap which cannot be bridged exists, to insert an autogenous nerve graft.

Surgical Procedures

If total paralysis from whatever cause with loss of electrical conductivity is present after a month, full recovery of movement is most unlikely and surgical exposure of the nerve trunk in the Fallopian canal should be considered. Though surgery cannot always restore complete function, it can often ensure restoration of the normal symmetry of the face at rest, and the ability to close the eye and move the brow and mouth, even though there may be some difference

in movement between the two sides. When the nerve has been badly damaged recovery of movement can be expected only after surgical restoration of the continuity of the nerve. In Bell's palsy some degree of spontaneous recovery is the rule in most cases, though in many recovery is incomplete and there may be considerable spasm. When, however, the nerve appears to be electrically dead decompression often encourages a better and quicker recovery of function. Sometimes decompression is followed within a few days by some return of function. This cannot be because of new nerve fibres growing down, and can only be accounted for by the restoration of activity as the result of decompression to some of the nerve fibres which did not formerly give any sign of life. This reversibility of function brought about by relief of pressure on the nerve is the principal reason for advising decompression in Bell's palsy (Cawthorne, 1951).

The results of these procedures are given in Table VII, in which they are classified as "good," "fair," or "poor," depending upon the recovery of facial movement. A good

TABLE VII.—*Results of Facial Nerve Operations*

	Good	Fair	Poor
Decompression for Bell's palsy ..	86	21	3
Exploration for injury or infection ..	31	17	6
Nerve graft	19	8	5

result means a normal appearance of the face at rest, with ability to close the eye and to move the nose and the lips. A fair result means either a normal appearance at rest or slight spasm on the affected side, with, however, fair movements of eye, nose, and mouth. A poor result means either no movement or only slight return of movement, or pronounced spasm.

Comment

In this short survey of facial palsy it will have been noted that as regards treatment surgical exposure of the nerve trunk at the site of the lesion is the only alternative to leaving things to nature. Support to the paralysed muscles, electrical stimulation of the paralysed muscles, and massage of the paralysed muscles contribute nothing at all towards the recovery of conductivity in the nerve, and so they should be regarded as supportive measures that may maintain tone in the muscles and are undoubtedly good for morale. What other benefits they confer is not certain.

In a case of facial palsy which is slow to recover and for which surgery is either not indicated or desired, an intra-oral splint will reduce the deformity from a drooping mouth. Vigorous manipulation of the face with grease-covered fingers three or four times a day is sufficient exercise for paralysed facial muscles. As soon as there is some recovery, active movements in front of a glass should be practised at least twice a day.

If all treatment, including local surgery, fails to bring about any return of function at all, the choice will then be between facio-hypoglossal anastomosis and a fascial sling procedure; the respective merits of these procedures were discussed by Sir Charles Ballance (1934) and Sir Harold Gillies (1934). We have no personal experience of either of these forms of treatment, though we have seen good results from each. We still feel, however, that in a case of facial palsy where the nerve is electrically dead an attempt should be made whenever it is surgically possible to expose the nerve at the site of the lesion. In this way it is possible to see what is wrong, and this is the first stage in providing the right remedy.

Summary

The frequency of facial palsy is attributed to the facial nerve's long and tortuous course through a small bony canal in the temporal bone.

Anything which causes a segment of the nerve or of its covering to swell in the bony canal is apt, by squeezing the blood out of the vessels, to result in ischaemia. This is thought to be the cause in Bell's palsy, which in this series of 557 cases of facial palsy accounted for 62%. Injury was responsible for 15% and a lesion at the geniculate ganglion for 7%.

By testing the ability to taste and to lacrimate in a patient with facial palsy it is often possible to differentiate between a lesion near the stylomastoid foramen, at the geniculate ganglion, and in the nucleus.

The results of exposing the facial nerve trunk at the site of the lesion in 196 cases are given; and it is suggested that in a case of facial palsy when the nerve is electrically dead an attempt should be made, whenever it is surgically practicable, to explore the nerve at the site of the lesion.

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INFLUENZA VACCINATION IN A RESIDENTIAL BOYS' SCHOOL

REPORT TO THE MEDICAL RESEARCH COUNCIL COMMITTEE ON CLINICAL TRIALS OF INFLUENZA VACCINE

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Cases of clinical influenza began to occur in Wellington College, Berks, soon after the school returned from the Christmas holidays on January 21, 1956. The outbreak reached its peak in February and was later shown to be due to influenza virus A. Of 664 boys present at the beginning of the term, 200 had been inoculated on December 9 or 11, 1955, with a saline vaccine prepared from the A/Eng/19/55 strain—one of three vaccines used during the same winter in a Medical Research Council field trial, the results of which are to be reported elsewhere. The A/Eng/19/55 strain, isolated in Carmarthen in April, 1955, is one of a group referred to by Isaacs (1956) as Eire '55 viruses. These viruses differ antigenically from those of the Scandinavian group. In addition, 100 of the 200 inoculated in 1955 had been inoculated in December, 1954, with a saline vaccine which contained a Scandinavian A strain (A/Eng/1/54),