BELL'S PALSY: SOME PROBLEMS OF PROGNOSIS AND TREATMENT

BY

G. A. DALTON, F.R.C.S., F.R.C.S.Ed.
Formerly E.N.T. Registrar, United Birmingham Hospitals

This study is based on a survey of the patients suffering from facial paralysis examined by me in the E.N.T. department of the United Birmingham Hospitals during a two-year period 1954 to 1956.

The aims of the investigation were to seek criteria which will give guidance on the probability of spontaneous recovery in idiopathic facial paralysis and to assess the value of present methods of treatment.

Material and Methods

Of the 107 patients (54 male, 53 female) examined, 56 were affected on the right, 49 on the left, and 2 bilaterally. Their ages ranged from 5 to 75 years, the highest incidence being in the 30–40 age-group. All the palsies were of lower-motor-neurone type, and so in a sense could be described as cases of Bell's palsy; for in 1821 Bell distinguished between "paralysis which proceeds from the brain, and that affection of the muscles of the face when from a less alarming cause they have lost the controlling influence of the respiratory (i.e., facial) nerve." However, the term Bell's palsy is nowadays reserved for those cases of peripheral facial palsy in which there is no obvious cause such as injury, infection, or new growth (Cawthorne, 1951).

Preliminary investigations established pathological causes in 10 patients. They were: (*a*) chronic mastoid infection (three patients); (*b*) intratemporal epidermoid of the type described by Jefferson and Smalley (1938) (two patients); (*c*) fracture involving the temporal bone (two patients); (*d*) sarcoidosis (one patient with bilateral paralysis); (*e*) acute middle-ear infection (one patient); (*f*) brain-stem glioma, which presented originally as a nuclear lesion (one patient). These are listed for completeness and are not discussed further.

Bell's Palsy and Ramsay Hunt's Syndrome

The remaining 97 patients are considered as a group. One of the 97 returned with a palsy on the opposite side nine months after her first attack, so 98 cases are available for consideration

It might be expected that they would comprise, on the one hand, the idiopathic affection called Bell's palsy and, on the other, the facial paralysis associated with herpes zoster affecting the head or neck, and often with auditory-nerve symptoms, described by Ramsay Hunt (1907).

Collier (1951, 1954) pointed out that Bell's palsy is probably a condition of multiple aetiology, some cases being due to exposure to cold, while others occur in small epidemics, sometimes associated with epidemics of herpes zoster and of anterior poliomyelitis.

In the present series eight patients attributed the condition to exposure to cold, two to recent childbirth, and two to dental extractions. Two patients told of another member of the household who had a transient facial paralysis; in one of these instances the children of the house were recovering from chicken-pox at the time.

Aitken and Brain (1933) showed by a complement-fixation test the presence of a zoster antibody in the serum of each of nine cases of Ramsay Hunt's syndrome; they then proceeded to demonstrate the antibody in the serum of 4 out of 22 cases of Bell's palsy. These four were indistinguishable clinically from the 18 in which the reaction was negative.

I have therefore regarded those cases of Ramsay Hunt's syndrome in the present series as simply more florid cases of Bell's palsy, bearing in mind that the latter term embraces a variety of conditions, some of which may be due to a zoster infection.

Investigations

A careful history was taken in each case and the completeness or otherwise of the paralysis assessed visually. Otological and neurological examinations were carried out; in many cases radiography of the mastoids, audiometry, and caloric tests of labyrinthine function were performed.

An attempt was made in each case to test the function of the three branches of the nerve which arise within the temporal bone. They are the chorda tympani, the nerve to the stapedius, and the greater superficial petrosal nerve.

The *chorda tympani*, the most peripheral branch, arises 4–6 mm. above the stylomastoid foramen. It conveys secretomotor fibres to the submandibular and sublingual glands, and taste fibres from the anterior two-thirds of the ipsilateral half of the tongue. The cell bodies of these afferent fibres lie in the geniculate ganglion of the facial nerve in the medial wall of the attic of the middle ear. No attempt was made to investigate the secretomotor function, and the gustatory function was assessed by electrical testing, using a galvanic current. An indifferent electrode having been attached to one arm, the circuit is completed by a metal electrode applied to the tongue. The current strength it slowly increased until the patient indicates that he is aware of the current, and the threshold recorded in milliamperes. In this way the threshold is recorded for various parts of the tongue. It is found that in normal individuals this threshold is rarely higher than 1.5 mA and usually below 1 mA, while in patients in whom the chorda tympani is known to have been divided in radical mastoid operations (but in whom the "common sensation" due to the mandibular nerve is intact), the threshold is much higher, usually in the order of 4 mA. Nearly all patients with a known or suspected chorda lesion have commented on the difference between the metallic taste on the sound side of the tongue and the sensation of an electric shock on the affected side.

This test is a variation of that described by Mackenzie (1955), who applied both electrodes to the tongue, and similar to that of Krarup (1958). It appears to be a valid method, for the same result could be obtained repeatedly in the same patient, and in those with a taste-defect the area of hyposensitivity corresponded accurately with the anatomical distribution of the chorda tympani.

It is interesting that while some patients noticed an absence or alteration of taste on the affected side at the onset of the paralysis, the majority in whom a chorda lesion (from any cause) was demonstrated were unaware of any loss of taste.

The *nerve to the stapedius* arises above the chorda tympani in the vertical portion of the facial nerve. The stapedius exerts a protective damping effect upon sound vibrations reaching the inner ear and is activated reflexly

by sounds of fairly high intensity (Hallpike, 1935). Patients were questioned about their reactions to loud sounds, and some gave a clear-cut history that such sounds as children's voices and clashing crockery were almost intolerable during the early stages of the paralysis.

The *greater superficial petrosal nerve* arises at the level of the geniculate ganglion. It supplies secreto-motor fibres to the lacrimal gland and conveys taste from the soft palate via fibres which, like those of the chorda tympani, have their cell bodies in the geniculate ganglion. (1) The rate of secretion of the lacrimal gland was studied by Schirmer's (1903) test, in which a strip of filter paper is hooked over the lower lid to act as a wick, and the rate of flow along it compared with that along a similar strip applied to the opposite eye. The test proved rather difficult to apply, owing to the fact that in facial palsy the lower punctum tends to sag away from the globe, causing the eye to "water." In fact, in only two cases was unequivocal hyposecretion observed on the same side as the facial paralysis. (2) The taste on the soft palate has been investigated as on the tongue.

Strength–duration curves were plotted in most cases in which the paralysis persisted for four weeks or longer, and electromyography was carried out in three patients. These tests are referred to later.

Prognosis

Associated Symptoms

It is widely held that the severity of associated symptoms, especially pain, at the onset worsen the prognosis for complete recovery of facial function (Tumarkin, 1936; Sullivan and McAskile, 1952; Cawthorne, 1952; Kettel, 1959). Therefore in the present series a clinical classification has been adopted according to the presence of symptoms, apart from those due to a lesion of the facial nerve itself (for example, loss of taste). Four groups have been distinguished. Group A: facial paralysis, herpetic vesicles, eighth nerve symptoms, and pain—that is, Ramsay Hunt's syndrome. Group B: facial paralysis, eighth nerve symptoms, and pain. Group C: facial paralysis and pain. Group D: facial paralysis only.

Group A.—Seven patients presented a complete facial paralysis, eighth-nerve symptoms, and herpetic vesicles, affecting the distribution of one or more cranial or cervical nerves, and associated with severe pain. Ramsay Hunt postulated the existence of a sensory area of distribution of the facial nerve, this area being the external auditory meatus and the external surface of the concha. He regarded this nerve supply as being superimposed on the generally accepted nerve supply to the region, by the auriculo-temporal, auricular branch of the vagus, and greater auricular nerves. Herpetic eruptions were seen in the area he described in five cases, while in the other two the tongue and palate were affected. All seven suffered from aural vertigo, three from perceptive deafness, and one from a recurrent laryngeal-nerve palsy. Two patients made a complete recovery—one in five weeks and one in seven weeks from the onset; four had made a partial recovery within 12 months; and one showed no recovery at all in that time.

Group B.—Five cases have been placed under this heading because they had auditory-nerve symptoms and severe pain, in addition to a complete facial paralysis. They would seem to represent a clinical halfway stage between Bell's palsy and Ramsay Hunt's syndrome. Four had vertigo, and two perceptive deafness with tinnitus. One patient was admitted for lumbar puncture because of slight nuchal rigidity. The C.S.F. contained 131 cells/c.mm., of which 98% were monocytes. Two patients had recovered complete facial function at 4 and 16 weeks from the onset respectively, while the other three had a partial paralysis 12 months after the onset.

Group C.—The patients whose only other complaint was pain at the onset of the paralysis numbered 44, of whom 36 had a complete and 8 an incomplete paralysis. The description varied from a dull ache to excruciating pain, and the duration of the pain from a few hours to two weeks. Some patients had multiple sites of pain and in several the distribution of a cutaneous nerve was defined accurately. The frequency of the sites mentioned were: external auditory meatus, 16; mastoid process, 13; neck, 11; mandible, 5; occiput, 4; maxilla, 2; auriculo-temporal nerve, 1; ophthalmic nerve, 1. In every case the pain was limited to the same side of the head and neck as the facial paralysis. Five patients did not return for re-examination. One-third of those who were followed up (13 out of 39) achieved partial recovery within a year and 26 made a complete recovery.

Group D.—42 patients presented with facial paralysis and no other symptoms or signs (except those due to a lesion of a branch of the facial nerve); 23 had a complete palsy, while 19 retained some function of the nerve throughout. Five patients also did not reattend after the first examination. Less than one-sixth of those who were followed up achieved only partial recovery within a year, while 31 out of 37 made a complete recovery.

The recovery rates in the four groups are shown in Table I. While the numbers in groups A and B are small, the overall pattern of Table I confirms that the probability of complete recovery is lessened by the presence of pain at the onset, and is much diminished by the presence of auditory-nerve symptoms and herpes. It should be noted, however, that of the 12 cases in groups A and B four made a complete recovery, three of them within seven weeks of the onset.

TABLE I.—*Associated Symptoms and Recovery Rate*

Group	No. of Patients	Paralysis Complete	Recovery			
			Complete	Partial	None	Not Known
A	7	7	2	4	1	0
B	5	5	2	3	0	0
C	44	36 (82%)	26 (59%)	13 (30%)	0	5 (11%)
D	42	23 (55%)	31 (74%)	6 (14%)	0	5 (12%)

Branch Lesions

One of the aims of this investigation was to discover whether the presence or absence of lesions of the intra-temporal branches of the facial nerve would offer a guide to prognosis. In Table II the recovery of the 88 patients who continued to attend is analysed according to the branch lesions discovered at the first examination. It is clear that in this series the ultimate result bore no relation to the presence or absence of taste-loss or intolerance of loud sounds. The fact that both patients with a "dry eye" failed to make a complete recovery may, however, be significant.

Incomplete Paralysis

The impression that when the initial paralysis is incomplete complete recovery can be expected was confirmed in this study, for 22 patients whose paralysis

TABLE II.—*Relation of Intratemporal Branch Lesions to Ultimate Recovery of Facial Nerve*

Branch	Function	No.	Recovery		
			Complete	Partial	None
Chorda tympani	Lost	44	30	13	1
	Retained	44	31	13	—
Nerve to stapedius	Intolerance of loud sounds	8	8	—	—
	No intolerance	80	53	26	1
Greater superficial petrosal:					
Taste on palate	Lost	9	7	2	—
	Retained	79	54	24	1
Lacrimation	Diminished	2	—	2	—
	Not diminished	86	61	24	1

was never complete continued to attend, and all made a complete recovery.

Duration of Paralysis

It is well known that the longer the interval between the onset of paralysis and beginning of recovery the less likely is complete recovery to occur. Of 61 patients who achieved complete recovery, 59 had visible voluntary movements within 6 weeks, one in 8 weeks, and one in 12 weeks; while of 26 patients who achieved only partial recovery, one showed some improvement in 3½ weeks, one in 4 weeks, two in 7 weeks, and 21 at intervals of from 9 weeks to 6 months from the onset. Cawthorne (1951) found that if recovery was going to be complete it usually started within a month of the onset.

Recurrent Cases

In this series, five patients gave a history of more than one attack of facial paralysis. Four had experienced a previous attack on the opposite side and one on the same side. One patient, a woman of 47, was observed during both attacks. In August, 1955, she had a complete right-sided facial paralysis with severe mastoid pain. There were no branch lesions. Voluntary movements did not return for three months, though strength–duration curves and electromyography suggested that denervation was incomplete. Recovery was slow and incomplete, with marked synkinesia. In May, 1956, she reattended with a complete facial paralysis on the opposite side. There was no pain, but taste was absent on the same side of the tongue. Recovery began on this side in three days and was complete in eight weeks. The evidence of this case thus supports my view that the presence of pain is of more prognostic significance than is a lesion of a branch of the nerve, such as loss of taste.

Pathology

The facial nerve and its connective-tissue sheath traverse a rigid canal, which is 30 mm. long, within the temporal bone. Many authors, including Ballance and Duel (1932), Morris (1938), Cawthorne (1946), and Kettel (1947), who have operated on the facial canal in cases of Bell's palsy, have described how, in many cases, the nerve is under tension and bulges when the sheath is slit after the canal has been opened, especially in the lower part of the vertical portion of the canal, where the sheath is thickest.

It is believed (Blunt, 1956; Kettel, 1947, 1959) that this oedema of the nerve is due to a dysregulation of the circulation within the facial canal. They postulate that ischaemia of the nerve due to a primary arteriolar vasoconstriction near the stylomastoid foramen leads to oedema and secondary ischaemia. Blunt concedes that an initial swelling of the nerve due to some cause other than primary ischaemia might set up a vicious circle of compression, ischaemia, oedema, and further compression, but found no convincing evidence of such an initial neuritis.

Wyburn-Mason (1954) has produced evidence that some cases of Bell's palsy are due to a neuritis or irritative lesion of the second and third cervical nerves, and he suggests that in these cases the greater auricular nerve involves the facial nerve by way of the branches of communication between the two nerves in the region of the parotid gland.

Denny-Brown, Adams, and Fitzgerald (1944) reported necropsy findings on a case of Ramsay Hunt's syndrome. They found typical necrotizing ganglionitis of the second dorsal root ganglion, and, while the facial nerve was infiltrated with dense patches of lymphocytes, the geniculate ganglion was virtually normal.

Similar findings reported by Findlay (1950) and by Kettel *et al.*, 1959, led Kettel (1959) to agree with the conclusion of Denny-Brown *et al.* that the evidence for geniculate ganglionitis in Ramsay Hunt's syndrome is invalid.

Functional Block or Degeneration?

James and Russell (1951), in a series of 58 cases, drew attention to the fact that those who achieved complete recovery showed the first signs of improvement in two to four weeks, while in those in whom recovery was incomplete the return of voluntary movement was delayed for two to four months from the onset of the paralysis. The recoveries in the present series conform to the pattern of those of James and Russell, and it is over the treatment of the patients who start to recover later (and, as has been shown, incompletely) that considerable diversity of opinion exists.

In the larger group of patients who recover early it is believed that the nerve below the lesion is in a state of "physiological block" or neuropraxia—that is, it is capable of conducting impulses—and normal function is possible as soon as the lesion causing the block subsides. Neuropraxia may persist for several days or weeks before recovery or degeneration occurs.

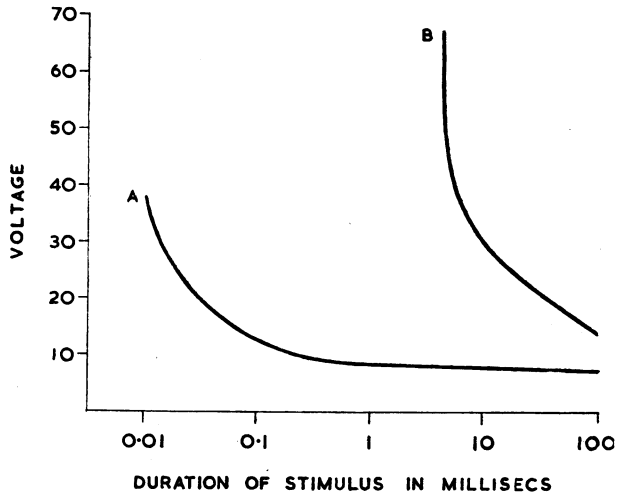
In the smaller group in whom delayed recovery occurs the nerve fibrils below the lesion are believed to have undergone degeneration. Recovery is then possible only when regenerating axons traverse the site of the lesion and follow the peripheral neurilemmal tubes to the muscle fibres. If the tubes are shrunken and distorted recovery will be imperfect; if axons enter the "wrong" tubes, synkinesia—associated movements of different parts of the face—will result; while if the lesion is proximal to the origin of the greater superficial petrosal nerve, lacrimation on eating may give rise to the annoying symptom of "crocodile tears."

The state of the nerve below the lesion may be assessed by the strength–duration curves, the faradic test, and electromyography. In the present series strength–duration curves were plotted in most cases in which clinical recovery had occurred in one month, and were repeated at monthly intervals. Electromyography was carried out on three patients only, and the faradic test as such was not used at all.

Electrodiagnosis

Strength–Duration Curves.—The time for which a given voltage must be applied over the "motor points"

of individual facial muscles to produce a contraction is determined, and the results are plotted for various voltages as in the Chart. If there is functional block but the nerve has not undergone degeneration a normal curve (A) is obtained. If degeneration has occurred the curve shows a shift to the right because denervated muscle which is being stimulated directly is less sensitive than muscle which is innervated.



Strength-duration curves. A=Normally innervated muscle. B=Denervated muscle.

Faradic Test.—It is evident from the Chart that electrical stimuli shorter than a certain critical duration will not activate denervated muscle, however strong the current may be ; this is the basis of the faradic test, for in a faradic current the duration of the impulses is too short to excite denervated muscle but long enough to stimulate functioning nerve fibrils.

Electromyography.—Here an individual muscle is needled and the electrical disturbance on attempted contraction is recorded on a cathode-ray oscillograph. Spike potentials denote intact functioning nerve fibres even when no obvious movement is present. Fibrillation potentials indicate denervated muscle fibres which are capable of function if reinnervated.

These three methods of electrodiagnosis are not infallible. The strength–duration curve represents the curve for the most excitable fibre present, and may not take account of other less active or degenerate fibres. The electromyograph may show the presence of active motor units, but from the patients' point of view a sufficient number of motor units to produce an acceptable cosmetic result is the most important consideration.

Collier (1954) stressed that too much should not be expected of these electrical aids to diagnosis, but advocated their repeated use as a guide to progress.

Methods of Treatment

Treatment in the present series was by standard methods. Analgesics and bed-rest were advised when necessitated by pain and vertigo, and local treatment was directed initially to ensuring that as little harm as possible came to the paralysed muscles while the function of the nerve was in abeyance.

A rubber-covered wire splint, hooked into the angle of the mouth and looped over the pinna like a spectacle frame, was used to prevent overstretching of the muscles by the drag of the paralysed face. In prolonged cases an intraoral splint was fitted by dental colleagues to the upper teeth or denture for the same purpose.

Radiant heat was prescribed if pain was present. As soon as pain was absent galvanic stimulation was given daily to maintain the contractility of the muscles. Later the interval was lengthened to twice weekly.

When voluntary movements could be discerned active exercises were prescribed, care being taken that simultaneous movement of the sound side of the face was limited to a minimum to avoid overstretching of the muscles of the weak side.

All observers are agreed that 75 to 85% of cases make a full recovery under such a regime, which in effect consists of waiting for spontaneous recovery of the nerve. Korkis (1959) has shown that the percentage of early complete recoveries may be substantially increased by treating all patients in the early stages by cervical sympathetic blockage.

Authoritative opinion is divided on the value of surgical treatment in the 20% or so of patients who have not begun to recover within a few weeks of the onset, and the advocates of decompression are not entirely agreed on the indications for intervention.

To Decompress or Not ?

As swelling of the nerve and compression by the rigid canal are a feature of Bell's palsy, surgical decompression seems at first sight to be a logical procedure, but Collier rightly insists that we should be clear about the object of the operation. Is it to prevent degeneration or to promote regeneration ?

If it is to prevent degeneration the operation should be carried out in the early stages—when we know that 80% would have recovered completely and most of the remainder partially in any case. Even supposing that all patients with a complete paralysis were submitted to operation early, on the grounds that there is no means of selecting the unfortunate 20% who will not spontaneously recover completely, there is no evidence that more patients would get complete recovery. The available evidence on early decompression is in fact discouraging. Kettel (1947, 1959) described how he decompressed the nerve in the early stage of paralysis on five occasions. Operation was carried out after 12 hours, 7 days, 8 days, 8 days, and 12 days of complete paralysis in the five cases respectively, and only partial recovery was obtained in each. Kettel stated that in two cases the results were among the poorest he had had from decompression. It is difficult to resist the conclusion that, even in his very experienced hands, decompression was harmful in the early stages of paralysis. He has given as his indications for decompression, paralysis which is still complete after two months, recurrent paralysis, paralysis with severe pain, and cessation of spontaneous improvement short of complete recovery. He disregards the faradic test as an aid to prognosis.

Cawthorne (1951) has advocated decompression when complete paralysis has been present for a month and the faradic reaction is negative. He believes that while such cases are unlikely to obtain complete recovery, with or without operation, speedier and more complete recovery occurs after operation, and that troublesome spasms of the facial muscles are less likely to occur.

Sullivan (1952) has advised decompression if no recovery has occurred in six weeks, or earlier if complete

facial paralysis is accompanied by pain and loss of the faradic response.

Decompression in the Present Series

Decompression was carried out in four patients, each of whom had a complete paralysis. Complete recovery occurred in one, partial recovery in two, and no recovery in one.

Case 28.—A man of 42 suffered a complete paralysis with no pain or other symptoms. Electrical tests were not carried out. There was no improvement in eight weeks and decompression was performed. The nerve was found to be swollen. Five days later voluntary movements were present and recovery was complete within four months of the onset.

Case 86.—A woman of 28 with a right complete facial paralysis and mastoid pain was treated conservatively for two months without improvement. The strength–duration curves were to the right in one month and unchanged in two months. Electromyography showed fibrillation but no motor unit potentials. At operation the upper third of the vertical portion of the nerve was swollen. Clinical improvement followed three weeks later (11 weeks from the onset) but never became complete. Five months after the onset electromyography showed some motor unit potentials and no fibrillation potentials.

Case 99.—A 14-year-old girl suffered a complete right facial paralysis with mastoid pain. The strength–duration curves showed a shift to the right in four weeks and at eight weeks a slight improvement, but as there was no clinical improvement decompression was carried out. The nerve was swollen. At 12 weeks from onset there was a sudden return of the strength–duration curves to normal, preceding by two weeks clinical improvement of the face. When she was last seen seven months from the onset, recovery was incomplete but continuing.

Case 92.—A woman of 28 suffered a complete right facial palsy associated with herpes and vertigo. Though told that it was a forlorn hope, she was anxious for everything possible to be done, and decompression was undertaken at 16 weeks, when the nerve was found to be atrophic. No recovery was found in the ensuing 12 months.

By contrast, in the following two cases decompression was advised but not carried out. Nevertheless, the ultimate results would probably not have been better had the operation been performed.

Case 54.—A woman of 26 suffered a complete right facial paralysis with otalgia and was found to have lost her taste on the right side of the tongue and soft palate. At eight weeks the strength–duration curves were to the right and there was still complete paralysis; admission was therefore arranged for decompression. When she was admitted two weeks later there was slight voluntary movement in the orbicularis oculi, and the operation was cancelled. Steady improvement occurred, both clinically and in the strength–duration curves, and 12 months later her face was normal except for a slight weakness of the lower lip.

Case 88.—A man of 34 suffered a complete right facial paralysis with otalgia and vertigo. As there was no improvement and the strength–duration curves showed a shift to the right, decompression was advised at eight weeks. For domestic reasons he was not admitted at once, and was seen at 13 weeks, when there was voluntary movement in all parts of the face. A year later the strength–duration curves were normal and his facial movements nearly so.

While no valid generalization could be made about the value of decompression on the evidence of three cases operated on at eight weeks from the onset, and two cases in which the operation seemed indicated but was not performed, it seems likely that complete recovery would not have been achieved in Case 28 had the patient been treated conservatively, and that in Cases 86 and 99 a comparable result would probably have been achieved without operation. Those who advocate decompression agree that in the cases they select for operation incomplete recovery is to be expected whatever method of treatment is adopted, but maintain that operation produces more complete recovery. Many surgeons relate personal experiences of cases where recovery follows so closely after operation that they are convinced of a cause-and-effect relationship, but in view of the high rate of spontaneous recovery shown in this and other series at about the time when operation is usually advised, my view is that the question of the value of decompression must remain an open one.

Finally, the following case is of interest as it raises the possibility of a further avenue to be explored.

Case 21.—A woman of 67 suffered a complete right facial paralysis associated with vertigo; herpes on, below, and behind the pinna; and paresis of the right vocal cord. Owing to the appearance of fluid in the tympanic cavity, conductive deafness, and radiological changes in the mastoid, a cortical mastoidectomy was performed three and a half weeks from the onset. The bone was found to be vascular and soft, and the cells to contain blood-stained exudate. The facial nerve was not exposed. Within two days facial movements were apparent and she proceeded to complete recovery within seven weeks.

Tumarkin (1936) described a very similar case where he performed an extensive cortical mastoidectomy in a patient with Ramsay Hunt's syndrome and obtained immediate improvement in the paralysis and relief of pain.

Kettel has quoted a case of Flodgren's, where a cortical mastoidectomy was performed in a case which he regarded as Bell's palsy. The paresis was noted to improve immediately blood-stained fluid was aspirated from the mastoid cavity.

Conclusion

A large field of inquiry still exists to determine the most satisfactory methods of treating Bell's palsy. Owing to the high rate of spontaneous recovery, valid conclusions can be drawn only from a large-scale clinical investigation with adequate controls as suggested by James and Russell (1951) and by Kettel (1959). Such an investigation should aim at evaluation of the following: (1) Cervical sympathetic block in the earliest stage of the condition, as advocated by Korkis (1959). (2) "Vascular decompression" by extensive cortical mastoidectomy (Tumarkin, 1936, 1951) in severe cases where otoscopic or radiological evidence of fluid in the middle-ear cleft is forthcoming. (3) In cases of delayed recovery, decompression of the nerve as soon as electromyography indicates (by fibrillation potentials) that degeneration is beginning (Kettel, 1959).

Summary

There is evidence that some cases of Bell's palsy are due to zoster infection. Therefore it was felt that, in a series of cases of Bell's palsy, manifest cases of Ramsay Hunt's syndrome (herpes zoster oticus) should be included.

The prognosis for recovery of facial function is worsened by pain and associated nerve lesions at the onset, but the extent to which the branches of the facial nerve itself are clinically involved has no prognostic significance except possibly in the case of lacrimal hyposecretion.

Incomplete and short-lived paralysis carry a good prognosis for complete recovery.

Current views on the pathological processes involved are presented and the value of electrodiagnostic tests is discussed.

No clear evidence emerges from this series to prove the value of facial-nerve decompression, and there is a strong case for a large-scale clinical investigation to evaluate this and other methods of treatment.

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REFERENCES

Aitken, R. S., and Brain, R. T. (1933). *Lancet*, **1**, 19.
Ballance, C., and Duel, A. B. (1932). *Arch. Otolaryng. (Chicago)*, **15**, 1.
Bell, C. (1821). *Phil. Trans.*, **111**, 398.
Blunt, M. J. (1956). *J. Laryng.*, **70**, 701.
Cawthorne, T. (1946). *Laryngoscope (St. Louis)*, **56**, 653.
—— (1951). *Proc. roy. Soc. Med.*, **44**, 565.
—— (1952). *Lancet*, **2**, 39.
Collier, J. (1951). *Proc. roy. Soc. Med.*, **44**, 569.
—— (1954). In *Modern Trends in Diseases of the Ear, Nose, and Throat*, edited by M. Ellis, p. 202. Butterworth, London.
Denny-Brown, D., Adams, R. D., and Fitzgerald, P. J. (1944). *Arch. Neurol. Psychiat. (Chicago)*, **51**, 216.
Findlay, J. P. (1950). *Facial Paralysis*. Angus and Robertson, Sydney.
—— Guldberg-Moller, J., Olsen, S., and Kettel, K. (1959). *A.M.A. Arch. Otolaryng.*, **69**, 220.
Hallpike, C. S. (1935). *Proc. roy. Soc. Med.*, **28**, 226.
Hunt, J. R. (1907). *J. nerv. ment. Dis.*, **34**, 73.
James, J. A., and Russell, W. R. (1951). *Lancet*, **2**, 519.
Jefferson, G., and Smalley, A. A. (1938). *J. Laryng.*, **53**, 417.
Kettel, K. (1947). *Arch. Otolaryng. (Chicago)*, **46**, 427.
—— (1959). *Peripheral Facial Palsy*. Blackwell, Oxford.
Korkis, F. B. (1959). *A.M.A. Arch. Otolaryng.*, **70**, 562.
Krarup, B. (1958). *Acta oto-laryng. (Stockh.)*, Suppl. 140, p. 201.
Mackenzie, I. C. K. (1955). *Lancet*, **1**, 377.
Morris, W. M. (1938). Ibid., **1**, 429.
Schirmer, O. (1903). *Albrecht v. Graefes Arch. Ophthal.*, **56**, 197.
Sullivan, J. A. (1952). *Laryngoscope (St. Louis)*, **62**, 449.
—— and McAskile, K. (1952). In *Diseases of the Ear, Nose, and Throat*, edited by W. G. Scott-Brown, **2**, 221. Butterworth, London.
Tumarkin, I. A. (1936). *Proc. roy. Soc. Med.*, **29**, 1685.
—— (1951). Ibid., **44**, 572.
Wyburn-Mason, R. (1954). *Brit. med. J.*, **2**, 679.

A NEW TREATMENT OF ANOREXIA NERVOSA

BY

PETER J. DALLY,* M.B., M.R.C.P.

AND

WILLIAM SARGANT, M.B., F.R.C.P.

From the Department of Psychological Medicine, St. Thomas's Hospital, London

Ever since Gull (1874) first described anorexia nervosa it has continued to present difficulties to general physicians and psychiatrists alike. A great deal has been written about the condition, many detailed psychiatric and endocrinological studies have been made, but little advance in treatment has occurred. Early on, Gull recognized that the patient's refusal to eat was due to "a morbid mental state" and "the inclinations of the patient must be in no way consulted . . . patients should be fed at regular intervals and surrounded by persons who would have moral control over them; relatives and friends being generally the worst attendants." He thought the prognosis was on the whole a favourable one, but doctors have since found these patients very unwilling to eat despite all the "moral" and other kinds of persuasion given. In recent years frank pessimism has sometimes been expressed about the eventual outcome of treatment. A recent study from the Maudsley Hospital showed that 15% of patients in the group followed up had died from the illness or its complications.

Hormone preparations, including thyroid, insulin, and cortisone, have all been tried with little effect. Electric shock has frequently been given, but the results are far from encouraging, and more harm than good may be done unless there is marked endogenous depression. Because of the unsuccessful results of treatment in so many patients, one of us (Sargant, 1951) treated selected chronic cases by modified leucotomy, and suggested that this was preferable to the much graver risks of allowing states of inanition to continue indefinitely. Since then the fashion in treatment has returned almost to where it started. In Gull's time, tube feeding was sometimes used as a last resort, but most workers since then have agreed that it is most undesirable from a psychological point of view. Yet within the last two years papers have again been published advocating a routine use of tube feeding as the most practical and best alternative to modified leucotomy (Williams, 1958; Wall, 1959).

In a letter on the general undesirability of tube feeding in this disease, we mentioned the use of a new method of treatment in which modified insulin treatment was combined with large doses of chlorpromazine (Dally, Oppenheim, and Sargant, 1958). We have found this method to be far more effective and simple than any other we have tried. For many years we used modified insulin alone in the treatment of anorexia nervosa, but with very little success. Then N. Moore, in a personal communication to one of us (W. S.), said that, while investigating the use of reserpine in schizophrenia at St. Patrick's Hospital, Dublin, he had also noted its value in the treatment of certain cases of anorexia nervosa. The use of reserpine, however, produced states of severe depression in some of our patients, and this led us to try chlorpromazine instead. Then we started to combine chlorpromazine with the modified insulin treatment. This paper now reports the first 20 cases of anorexia nervosa treated by this combined method over the past three years, and compares results with those obtained in 24 similar cases, all referred for a psychiatric opinion, and treated in St. Thomas's Hospital by other methods over the past 20 years. Table I compares these two groups of patients.

TABLE I.—*A Comparison of Patients of Groups A (Chlorpromazine and Insulin) and B (Other Methods)*

	Group A	Group B
Average age at onset of illness	18·8± 5·5 years	18·0± 5·2 years
,, weight lost before treatment	37·1±15·1 lb.	33·5±10·3 lb.
,, length of illness before treatment at S.T.H.	49±71 months	22±18 months

Selection of Patients

Anorexia nervosa is a syndrome which needs to be clearly defined. We regard the essential criteria for its diagnosis to be: (1) refusal to eat, whether or not accompanied by anorexia at the start; (2) severe loss of weight; (3) absence of evidence of schizophrenia, severe depression, or organic disease; and (4) amenorrhoea of at least three months' duration in female patients. Eventually most cases of anorexia nervosa will also develop

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