Surgery of the Facial Nerve*

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It is only a short time ago that no treatment was possible for a paralysis of the facial nerve. A severed nerve was doomed never to function again and in the other cases of damage one had to be satisfied with such pseudo-treatments as galvanization of the facial musculature, salicylic acid, massage. For quite some time statistical analysis has proved (Taverner, 1954; Verjaal, 1959) that these are of no value except for keeping the patient quiet and the doctor satisfied. In cases of permanent disfigurement the neuro-surgeon was called in to make a connection between the peripheral part of the facial and the central part of another cranial motor nerve (e.g. XI or XII), or the plastic surgeon was asked to restore some symmetry at rest.

Since the work of Bunnell (1927), Martin (1931) and Ballance and Duel (1932) much has changed as regards the possibilities to restore the language of facial expression. Hundreds, even thousands of operations have been performed (Cawthorne, 1946; Kettel, 1959; Sullivan, 1950 etc.) inside the temporal bone and the percentage of complete recovery or at least important improvement proved to be high even after complete severance of the nerve.

Outside the temporal bone during surgery on the parotid gland and after traumas very good results have been accomplished (Conley, 1955; Maxwell, 1954; Miehlke, 1960, etc.), and also the inner-ear canal and the pontine angle have been opened to surgery (Dott, 1958; House, 1963; Miehlke, 1960, etc.). The main advantage of the reconstruction of the nerve

* Paper read at the Section of Otology, Royal Society of Medicine, at Oxford, 3 August 1967.
itself over a connection with other cranial nerves is the recovery of emotional movements. Neither the plastic surgeon, nor the neurosurgeon can give the patient more than symmetry at rest and at best some voluntary contractions which can be trained. How important this difference is will be clear to anyone who has ever compared the results of facial-nerve reconstruction with the other techniques (Fig. 1).

Fig. 1.
Complete facial paralysis treated
(a) by facial nerve grafting
(b) by plastic surgery (fascia-strips)
(c) by connection of central accessory and peripheral facial nerve.
(A) at rest; (B) voluntary movements of right side; (C) smiling (note complete lack of emotional movements on the right side). By kind permission of Mr. Kettel.
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I do not think that it is necessary to go into these points any further. But, though the possibilities to obtain good results by surgery in cases of facial paralysis are theoretically recognized by all those who have given any thought to the problem, the practical effect of this knowledge is far from optimal. It still happens, though rarely, that neurosurgeons and plastic surgeons try their skill before the only rational restoration of the nerve has been tried or even discussed (Portmann in Kettel, 1959), but this will die out, I am sure, with an older generation, doctors being for an important part a very conservative minded lot, who seem to be reluctant to change anything they learned during their training. No, more important is a mentality that does not change. Today like in my youth, students are told that Bell's palsy cases nearly always recover. In this small sentence two statements are untrue and lead to a lot of unnecessary life-long suffering. Quite a lot of facial paralysis cases are not Bell's palsy and about 25 per cent. of Bell's palsy cases do not recover completely. I know that Dr. Taverner does not agree with me on the indication to operate in cases of real Bell's palsy, but I am sure he agrees with me that facial palsy is an emergency. It is absolutely necessary to know the cause of a facial paresis or paralysis in order to choose the right treatment, be it A.C.T.H. during the first days of a real idiopathic case, as Dr. Taverner gives with statistical evidence of success, be it immediate surgery if it proves to be a chronic otitis.

It still happens in my country, and I heard from friends that it sometimes happens in other countries as well, that patients with a facial paralysis were treated for weeks and even months with heat, vasodilating drugs, massage, histamine infusions, cortisone, and scores of other drugs, but that no ear surgeon had seen them to find the Shrapnell perforation and the cholesteatoma. I know that hearing in these cases was often normal or nearly normal and discharge was absent or minimal. However, the only chance to get good recovery in cases of facial paralysis caused by a chronic otitis is immediate operation and inspection of the Fallopian canal with eradication of any diseased bone or cholesteatoma around the nerve. If the operation takes place before the sun sets over the paralysis, the results are not bad. After 24 hours the prognosis deteriorates with great speed.

There are more cases of facial paralysis that need immediate operation and during the Round Table Discussion in Copenhagen, May 1964, it was evident that those who are familiar with facial paralysis agree that complete immediate post-operative facial paralysis should always be operated upon without delay. Very often the nerve is completely severed or badly bruised and at re-operation immediately after the damage has been inflicted it is relatively easy to repair it. Later granulations, scar tissue, and ingrowth of connective tissue into the nerve make surgery more difficult and the chance of recovery smaller (Fig. 2). Waiting for
miracles is stupid and unjustifiable. The same is more or less true for post-traumatic facial paralysis due to fracture of the skull. Here, however, some difficulties are present. It is not always easy to be sure whether the paralysis was immediate and complete. A bruised face, loss of consciousness, seriousness of the general condition may all help to camouflage the data about the paralysis. In cases where any uncertainty about the prognosis exists electrodiagnosis may be a great help. As long as the electrical tests do not show denervation, operation is not indicated, but if all excitability is lost, it is. Dubious cases are those with some denervation. In my opinion serial examinations can indicate whether denervation is increasing or decreasing and in this way help us make our decision whether to operate or not.

There is not much discussion either as to the cases of facial paralysis that should never be operated upon, e.g. if the surgeon cannot reach the site of the lesion or if he cannot change the condition of the nerve if reached at all.

Polyneuropathy may show itself in the facial nerve alone, herpes zoster, syphilis may affect the seventh nerve, but the treatment is non-surgical.

The great point of discussion is not whether facial-nerve surgery is important. Everybody agrees that in many cases this surgery can be of great importance for some patients with paralysis. That this surgery must be performed at the right moment i.e. as soon as possible is no point of discussion either. The fact that many patients are sent too late is not the result of any rational reasoning but of a certain unfounded idea of the type: It could be Bell’s palsy, it often is, why not in this case, let us wait. Or: I feel that this post-operative paralysis cannot be due to any mistake I made. It cannot happen to me. I did not look at the facial function after the operation, but it must have been intact, let us wait. This, however,
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is culpable indolence, perhaps disastrous for the individual patient, but not an important point for the discussion.

This important point of discussion is to be found in a group of facial paralyses that spontaneously recover in the great majority of cases, but not always. The most important of them is Bell’s palsy, but late paralyses after operation or skull trauma, partial palsies due to the same causes, acute otitis media causing facial paralysis, give more or less the same problems. Even in these cases it is unanimously accepted that the prognosis can be evaluated by electrical tests, e.g. electromyographic measurement of voluntary muscular activity, fibrillations, the measurement of the excitability threshold of the various branches of the nerve, nerve-conduction tests and so forth. It is important never to forget the clinical picture. The electrical tests must be evaluated as part of the entire group of data and also as part of serial examinations indicating either increase or decrease of symptoms. No unanimity, however, does exist on the efficacy of surgical treatment, decompression, in a selected group of these paralyses.

Let me try to bring the main points of the discussion as objectively as I can.

Those who operate do not think that they can perform miracles, but they think they can give the patients a chance of better and quicker

Fig. 3.
Half schematic drawing of the facial nerve in a case of Bell’s paralysis.
recovery if the nerve is decompressed as soon as they find indications of progressive denervation. In the first place they base this conviction on the observation of the facial nerve during decompression. This shows a typical picture of swelling inside the facial-nerve sheath (Fig. 3). As soon as the sheath is opened the swelling starts to bulge out and after a short time the size of the incised sheath would be completely insufficient to go round the nerve again. The outside of the sheath is covered by many swollen vessels, often double or three times the calibre and the number one finds in normal nerves. The swelling is only rarely found outside the vertical part of the Fallopian canal. It mostly stops at the entrance of the branch of the medial meningeal artery, which enters the canal at the site of the peripheral knee. The fresher the case the more important the swelling. Histologically the swelling has been shown to be oedema, without any signs of inflammation, but with degeneration of the nervous elements. This sight is very suggestive of a compression of the nerve inside its sheath, and of a deliverance of the nerve by the decompression.

The second argument is that the results of decompression in early cases (within about 10 days) are better than the results of decompression performed after some 4 to 6 weeks and these again are better than results in operations after about 3 months. Let me stress that in my own material, operations were only performed in cases that had a complete paralysis showing signs of increasing denervation. In early cases we see a large percentage of patients who show some return of function within some days or even hours after operation. This is in complete contrast to the patients we followed during the period in which we hesitated to operate within 8 weeks. One of the reasons we had to try and find a way to demonstrate a bad prognosis at an earlier moment was that the condition hardly ever changed between 2 and 8 weeks after the beginning of a complete paralysis. Another point is that the sometimes severe pain disappears immediately after operation. A third point of importance to those who operate is the difficulty of the diagnosis of Bell’s palsy. It is a diagnosis per exclusionem. A palsy of sudden onset of lower-motor-neuron type with no evidence of any other neurological or ear diseases, or infections (syphilis, virus), that might have caused it. When you know them well they all behave exactly like each other. As Taverner puts it: “They are all alike—peas in a pod. You become suspicious when you are confronted with a Bell’s palsy which does not follow the pattern.” Nevertheless it still is not an exception to find “Bell’s palsies”, according to the views of connoisseurs of Bell’s palsy, which prove to be something else. In my series of nearly 250 decompressions on patients who were diagnosed as Bell’s palsy, four had tumours of the middle ear, without any trace of middle-ear disease, or radiological deviations, three others had masked osteitis in the mastoid. I am sure that a better man would have found them, but since I have not learned the tricks to avoid all the
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mistakes, I like to have a look inside the ear in cases of selected so-called idiopathic or Bell’s paralysis. I do not mean to include the so-called Bell’s palsies we found to be chronic otitis before the operation. This number is much higher.

I have to confess at the same time that in about 7 per cent. of the operations for Bell’s palsy we do not find the swelling described above. Here we must also have made a mistake and performed an operation either without the right diagnosis or too late, I am afraid. One argument I only bring for the sake of completeness, I do not dare to attach too much value to it. Taverner tells us about a not unimportant percentage (25 per cent.) of Bell’s paralysis cases who showed complete denervation, “they have such bad faces that they are miserable for the rest of their lives”.

In my series of about 250 cases in which an important number, operated upon during the last 6–7 years, showed complete denervation, only one is really dissatisfied, a doctor who complains about tinnitus since the operation. His face gives him no complaints. I do not dare to value this argument too highly because I have no answer to the remark of one of my opponents who stated: for psycho-somatic successes you do not need operations.

As regards the arguments of those who do not proceed to an operation even if they do find cases with bad prognoses, I think the most important is that no proof has been given that the operation really improves the prognosis in cases of denervated Bell’s paralysis. If this means that all our arguments do not give statistical evidence, I agree, and should like to give more significance to the probability of my views by subjecting the problem to statistical tests, without forgetting, however, that statistics do not prove either. But since I am convinced by my own arguments, given in this paper, I could never accept that my own wife or children were put in the negative group in case of a search for statistical evidence only because a random distribution technique had decided it this way.

As I feel sure that I should treat my patients according to the same standards as my relatives I cannot start to make a series of statistically indicated operation versus conservative treatments. I tried to make a comparison with a conservative friend to compare his “conservative” with my “operative” series and had a doctor from my department down to his clinic.

But alas, he has found a treatment which, he believes, gives the solution to the problem of Bell’s idiopathic facial paralysis i.e. A.C.T.H. during the first 7 to 10 days. I am very sorry that I hardly ever see the patients within a week and they have up to now never had anything else but heat, faradization or cortisone.

Therefore I am still at the same point. Perhaps we can try to use A.C.T.H. versus decompressions for those patients who are sent after 1 week, but the number is small and we shall be obliged to pool our data,
their evaluation, the indication for operation, and the results. As Buchthal put it in Copenhagen: “To prove that surgery really helps, you need a sufficient number of patients. It would be easier to gather sufficiently large groups if different departments could agree on a scheme describing the clinical and laboratory findings before and after operation.” It must be possible to do such a thing. Who takes over where I failed?

Velasco from Chili gives a little foretaste in the comparison of two groups of about 10 patients, one operated upon and one conservatively treated. Though the groups were small the results obtained seem to be very suggestive e.g. in the operated group many patients showed complete recovery, in the non-operated group nobody did.

The other objection of the conservative group is directed against the compression hypothesis, the rationale of the operation. The high endoneural pressure has never been measured, only supposed. Here at least I can show my good will in a photograph of the instrument we designed to measure the endo-neural pressure. It gave us some results which are giving us hope that we shall be able to prove our point, but the instrument is still too complicated and only works when it is nearly vertical, this means that we shall have to look for instruments that can really be used during an operation on a living patient and not only on models and cadavers (Fig. 4).

There are for that matter valuable new data to be found. McGovern and Hansel induced temporary bilateral facial paralysis in dogs by tying the nerve trunk near the stylomastoid foramen with catgut. The histologic findings were similar to the reports of nerves from cases of Bell’s palsy. Decompression was performed on one side, the other side was used as a control. The decompressed nerves tended to recover 2–4 weeks earlier than the controls. The same was true when the bilateral paralysis was produced by complete trans-section and resuture. The recovery time was longer but the same tendency of quicker return of function on the decompressed side was present and striking in some dogs. The authors think that the absence of secondary compression is the cause of favourable comparison.

Jain and Sharma in rabbits observed that infusion of saline under pressure into the facial nerve within the Fallopian canal produced complete facial paralysis which lasted for a period directly proportional to the duration of the compression.

Rosenburg and Alford examined the value of decompression in cases of controlled injury of the facial nerve in dogs. It proved to be beneficial when performed within 3 weeks of the injury.

McGovern, Thompson and Link repeated these experiments with similar results and concluded: “We believe that the mechanical factor alone is the reason why the facial nerve is paralysed more often than any other nerve in the body.”

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FIG. 4.
Measurement of the pressure inside the facial nerve sheath. Instruments in use during an operation.

Let us hope that we can come to a unanimous opinion about the indications and the values of operations for the various pathological conditions I discussed.

There are still two other points I want to discuss shortly. One is the moment. In general it can be said in the words of the pioneers in this field: "the moment to operate is now, no delay is justifiable." I think this is still fundamentally true. As soon as you have decided that an operation is unavoidable, do it immediately. But sometimes you cannot be sure. In patients who show a complete paralysis with all the signs of complete denervation more than 3 months after the start, a decompression will not give a measurable improvement according to our results. The same is true for post-traumatic paralyses. Only grafting can help. The results of grafting after 1 year are not worse than those after 3 months and never 100 per cent. Wait and see how the condition develops. If the result is worse than you can achieve by grafting you can still operate. The second point is the surgeon and I would like to quote Sir Terence Cawthorne, who when he was asked to state which datum would most strongly influence his decision whether to proceed to an operation or not, answered: "The name of the surgeon." He discussed the VIIIth nerve, but I am sure he will allow me to use his dictum for the VIIth also. And I should like to finish this little survey with three case histories.
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Those who do not believe in the effect of decompression always point to the fact that new attacks of Bell’s paralysis are seen in patients who underwent a decompression previously. I have seen three and decided to perform another operation on those three. In the first case the surgeon had never found the facial nerve, in the second patient only the site of the stylomastoid foramen was reached and the foramen widened. In the third case a very nice decompression had taken place, but the decompressed nerve proved to be the chorda tympani.

Nowadays, I must confess, I distrust the statement that recurrence of Bell’s palsy is possible after a decompression operation, if it is well performed.

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