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THE OPERATIVE TREATMENT OF FACIAL PALSY *

BY

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(With Special Plate)

LECTURE I

I am especially pleased to accept the invitation to address you since it offers me the opportunity, here in his own circle of confreres, to pay tribute to the genius of my dear friend and colleague Sir Charles Ballance in this work. But for him I should never have undertaken it. Whatever of interest I may be able to present to you results from the inspiration of having worked so long with a man of such unequalled skill, such indomitable energy, and such amazing industry.

Historical

Nearly six years ago Sir Charles and I began working together on animal experimentation at my country place in the Berkshire Hills, seventy miles from New York. A laboratory quite adequate for our purpose was built and maintained, at first through the generosity of a few personal friends, and later by subsidies from four foundations.† Our first effort was to verify certain conclusions which Sir Charles had arrived at in a series of experiments on rhesus monkeys in England. After the performance of experimental operations on monkeys, in which the peripheral end of the divided facial nerve was united to the central end of another divided nerve of the neck, no test had been made to determine whether the divided ends of the facial nerve had reunited. If they had done so, the result of the experimental sutures would have been vitiated. Hence we repeated these experiments, and in each of them the central segment of the facial nerve was removed from the aqueduct. Some months later the brain was removed and the nerves in the posterior fossa stimulated with the faradic current. The facial nerve at the internal auditory meatus on the side of operation was stimulated, but no response in the muscles of the face occurred. If the facial nerve had been sutured to another nerve—for example, the glossopharyngeal—stimulation of the glossopharyngeal nerve in the posterior fossa resulted in contraction of the muscles of the face. We then sought some method by which the associated movements, accompanying cure of facial palsy by suture of the facial nerve to various motor nerves in the neck, might be eliminated.

Direct Line Repair

The idea occurred to us to attempt a direct repair of the facial nerve by introducing a piece of freshly excised graft from another nerve into the gap made by the removal of a piece of the facial nerve in the aqueduct. Accordingly, on August 27th, 1930—on a mandrill baboon—the facial nerve was divided in two places in the aqueduct 5 mm. apart, and reversed. On September 19th, in a hamadryad baboon, a segment of the facial nerve 6 mm. in length was removed and replaced by 6 mm. of fresh graft removed from an intercostal nerve. Both subjects recovered the use of their facial muscles. They were the first animals on which a direct line repair had been attempted. Subsequently we did a large series of experiments with grafts.

At the end of a year we had demonstrated that facial movements could be restored by introduction into the gap of a divided facial nerve, from which sections had been removed, of freshly excised autoplastic grafts of any desired length, either from motor or sensory nerves, either reversed or unreversed. These restored nerves conveyed both voluntary and emotional impulses. The muscles moved synchronously on both sides of the face, and there were no grotesque movements such as occurred after recovery following suture of the facial nerve with other motor nerves in the neck.

No one had inspected the facial nerve at the seat of injury in the various nerve-anastomosis operations in human cases, and consequently there were no statistics to guide us as to the extent of the gap in the divided nerve. We had supposed that it might be 5 mm. but not more than 10 mm. Subsequent work showed us that this was a gross underestimate. We felt that a gap of about this amount could be repaired by a graft taken from the external respiratory nerve of Bell (long thoracic nerve), without placing too much traction on the ends of this nerve when it was reconstituted. However, when gaps of 40 mm., and in one case 200 mm., had been bridged it was obvious that another nerve had to be used in which restoration of continuity was not essential. For this reason a sensory nerve was eventually selected, after experiments with intercostal nerves had been made. The anterior femoral cutaneous nerve provided lengths more than sufficient for any of our cases, and was our final choice.

Clinical Cases

The first human case in which a fresh graft was used, taken from the exterior respiratory nerve of Bell, was in an infant 18 months of age.

There had been an acute infection by the *Streptococcus haemolyticus* of the middle ear and mastoid air cells, with a post-auricular subperiosteal abscess. The young operator had proceeded as if he were dealing with an adult mastoid. As a result we found the facial nerve absent from a level just below the horizontal semicircular canal to the posterior border of the parotid gland. The exploration was made twenty-four days after the injury. It was possible to identify the facial nerve by faradic stimulation of the stump protruding

* Two special lectures delivered at the Royal College of Surgeons of England on October 10th and 11th, 1934.

† The Carnegie Corporation: The Milbank Foundation: The Lillian Babbitt Hyde Foundation: The New York Foundation.

from the parotid gland, otherwise a much more extensive dissection would have been necessary. (Faradic response in the distal segment of a divided facial nerve remains for from forty-eight to seventy-two hours. I have verified this in over fifty monkeys, and several times in man. Hence the importance of early investigation of cases of traumatic origin.) Twenty-seven millimetres of graft were employed in this case. The proximal end was laid up against the proximal end of the divided facial nerve ; the distal end was sutured to the distal stump by one strand of " 000000 " silk. The patient eventually recovered the use of her face (see Figs. 1 and 2 on Special Plate).

The second case presents a striking contrast of conditions.

The patient, a female aged 34, suffered from a chronic purulent otitis, for which a radical operation had been done. Immediate facial palsy had ensued. She was first seen by me eleven months later, when, after removal of 37 mm. of injured facial nerve from the tympanum to a point several millimetres distal to the foramen of the Fallopian aqueduct, the facial nerve was reconstituted by a freshly excised graft from the nerve of Bell. The wound was infected. She eventually recovered, with a straight face in repose, and good, though not perfect, synchronous movements, both voluntary and emotional (see Figs. 3 and 4 on Special Plate).

We learned much from these cases. First, we proved that suppuration was not a bar to successful transplantation of grafts. In the last four years, in a series of sixty-nine operations on facial palsy, a large majority have been done in the presence of infected wounds. Necrosis of a graft on account of infection has occurred only once. In that case a second effort was successful. Secondly, we discovered that injuries involved a larger extent of the facial nerve than we had supposed. Of the sixty-nine operated cases in my series forty have required grafts in the Fallopian canal. These forty cases have had gaps on an average over 20 mm. in length. The shortest was 7 mm., the longest 40 mm. This does not include a series of five cases in which operations on tumours of the parotid gland resulted in gaps in the facial nerve more than 40 mm. in length. In one case 200 mm. in four strands, from the proximal stump at the stylo-mastoid foramen to the divided distal branches in the face, were required. In the course of a year I operated on thirteen cases of facial palsy: eight of them necessitated grafts varying from 8 mm. to 36 mm. in length. Successful transplantation of fresh grafts was effected in all but one, and that succeeded on a second trial. Varying degrees of recovery took place, and I had an opportunity of studying the results.

Mechanism of Repair Process

The one phenomenon that gave me constant cause for speculation was the fact that in every case there was a period of waiting, followed by a response to faradic stimulation, and then spontaneous movements of muscles. In the case of a slight injury to the sheath of the facial nerve, with infection and inflammatory compression, a successful result had been obtained by decompression and slitting of the sheath of the nerve in the aqueduct. Faradic response (followed by spontaneous movement in the muscle) occurred in a period measured by weeks. In the chronic cases, in which a graft had been necessary, the interval between the time of repair and the first evidence of faradic response in the muscles was very much longer —measured by months instead of weeks.

In either case, once a faradic response appeared the subsequent improvement in the muscles was about the same. Now we all know that when a nerve is injured (to a degree that destroys its power to convey faradic stimuli), from the point of injury down to the last end-plate the nerve cells are rendered ineffective. The restoration of function in the distal segment must follow degeneration and removal from the conveying tubes of all the now useless neural fibres, followed by a growing-in, from the proximal segment, of new axons to take their place.

Now this degeneration and emptying process occurs rapidly after any serious injury of the facial nerve. The axons distal to the injury are rapidly disintegrated. The products of this disintegration are removed by the circulation, leaving the conveyors—the empty tubes—ready to receive the axons, which are pushed on through the proximal segment, from the central nuclei. This can all happen in the course of a few weeks, and we consequently see—in acute cases, where the conveyance of neurons has been facilitated by decompression of the nerve—recovery taking place in a relatively short period of time. Why, then, the long delay in the case of grafts? The distal segment has long been empty, Wallerian degeneration being complete. Speculating over this it seemed probable that the obstruction was offered in the graft, which as soon as it was transplanted became part of the distal segment. The fresh graft was full of non-degenerated axons ; they must be degenerated and removed before it can be utilized as a conveyor. This is a slow process with the meagre circulatory apparatus with which the graft is provided in its transplanted bed. It is enough to ask it to live for a long period in its new environment without it having to deal with the complex problem of getting rid of thousands of degenerating axons.

But if this graft were like the rest of the distal segment —that is, if its tubes had been cleared by Wallerian degeneration—it could live in its new environment in exactly the same way, and yet, being clear of these obstructing, useless neural elements, might be ready immediately to act as a conveyor. Why not, then, cut the nerve, which one proposed to utilize eventually for graft material, and allow it to remain *in situ*, undisturbed as to circulation, until Wallerian degeneration had advanced sufficiently, so that when a portion of it was used as a graft the emptied tubules would no longer offer an obstruction to the advancing neurons from the proximal segment of the facial nerve?

The plan was tried on a series of rhesus monkeys. The anterior femoral cutaneous nerve was severed and allowed to remain in its bed for varying periods from eight to thirty-five days—so that Wallerian degeneration could readily take place. The facial nerve was severed at the same time—so that Wallerian degeneration might take place, *pari passu*, in it as well.

When portions of the nerves treated in this manner were used as grafts facial response was restored in one-quarter to one-half the time formerly required by fresh grafts. I obtained faradic response in from sixteen to twenty-six days through 10 mm. of prepared graft—that is, graft already degenerated after the grafting operation.

Having proved its efficacy on monkeys, I began two years ago to employ the method in man. I have now used it in thirty cases. Faradic response has come through much earlier in all cases. In a number of cases this was as early as thirty days after transplantation. It has not been unusual, when I have employed prepared grafts, to observe a state of progress in six or eight weeks, only reached in six or eight months in the earlier cases where fresh grafts had been used. The degeneration in the nerve to be used as a graft requires two to three weeks. The additional time in hospital is well expended, since it saves so many months in the final recovery.

Details of Operative Technique

May I digress at this point to say how important I consider practice on cadavers and monkeys to be for any surgeon who is to attempt this work, no matter how skilled he may be in the usual operations on the temporal bone. On any particular case it seems to me essential

for the best good of the patient that the operator should have in mind definitely what he proposes to do. This should be acquired by previous practice. Trial and success, or trial and failure, may definitely fix the operator's mind as to what he may or may not do. For example, I had worked up a technique on the cadaver, with a motor-driven burr, which I hoped would materially shorten the time of exposure of the nerve in the aqueduct. On my fourth effort, when I fancied I was becoming quite proficient, a catastrophe occurred which made me abandon the motor-driven burr for all time, in favour of a slower but surer method. No one can tell you how you are to do your own ivory carving. You must do it your own way, with your instruments, according to a plan you have learnt ; you should not practise on your case in hand, but carry out your previously determined method. You may on your second, or fourth, or fifth case change your plan of operation for your third, fifth, or sixth. Practise the change of idea on the cadaver before attempting it on the living case. You may change your mind after trying it, as I have done on two or three occasions, for the good of the patient.

Instruments.—A word regarding the details of operation and after-care. As I have said before, each operator must practise with his own instruments, in his own way, the method of removal of the bony wall of the aqueduct. I personally have discarded the use of a motor-driven burr, or a chisel, or a gouge. While these may be faster, I feel that I am by this method sacrificing speed to accuracy. I prefer small rongeur forceps, hand gouges, and straight curettes with non-flexible shanks. All of these should be very sharp. I also devised a number of hand burrs and files, which I have now discarded. Practice on the cadaver with favourite models has helped me more than newly devised instruments.

Exposure.—I always uncover the nerve by removal of the outer wall of the aqueduct, working from the stylo-mastoid foramen upward. In every instance the outer wall of the canal up to the site of injury and for a few millimetres beyond should be removed. I find that the exposure of the nerve is greatly facilitated by removal of several millimetres of the floor and posterior bony wall of the external auditory meatus. This is particularly useful as one approaches the narrow and deep portion of the aqueduct lying between the horizontal semicircular canal and the oval window. This truly represents the Scylla and Charybdis of the operation. Fortunately, in many of the cases it is unnecessary to remove this part of the aqueduct. It is never necessary to do so in decompression for Bell's palsy. Injuries to the nerve above the level of the horizontal semicircular canal occur rarely, except in radical mastoid excavations. When it is necessary to uncover this portion of the nerve the only safe approach is from before backward, through the posterior bony canal wall and hypotympanum. Cracking through the horizontal semicircular canal above and dislocation of the stapes below are the two great dangers. The removal of the bony covering from this point up to the geniculate ganglion is very easy, as it has only the thickness of stiff tissue paper.

Neurectomy and Insertion of Graft.—The nerve having been uncovered from 5 mm. above the site of injury to the stylo-mastoid foramen, the sheath should be slit open with the sharpest of Graefe knives over all this area. The sheath must not be torn apart ; it must be gently incised. When the tube is laid open the bundle of nerve fibres appears. Inspection in every instance will show the site of injury where scar tissue may be expected to form during healing, or where scar tissue is already present. The distal segment—that is, the nerve beyond the injured or scarred zone—should be cut well beyond the lowest point of scarring, and the injured segment of nerve removed. The neuroma of the proximal segment should be cut off cleanly and squarely. This must be done gently to avoid crushing of the axons. Crushing instead of cutting with the keenest of blades either of the proximal or distal ends of the facial nerve or of the graft will immeasurably delay the time of recovery. The length of the gap does not matter. The distance can then be measured, the graft taken from the " prepared " anterior femoral cutaneous nerve, and laid in. The graft should be long enough so that it may be tucked in rather than stretched between the freshly excised ends of the facial nerve.

Haemorrhage.—This formerly gave me much concern, and I have spent hours in stopping it with hot saline solution before inveigling the graft into position. The use of adrenaline, peroxide, and other haemostatics is most inadvisable. What I have now learnt is that if the ooze of blood from the bone and soft tissues is temporarily stopped, so that the ends of the graft can be opposed to the sectioned facial stumps, they almost immediately become glued together sufficiently to prevent the entrance of blood. The subsequent ooze about the graft provides a fibrinous bed which fixes it in position. There is no doubt that a blood clot between the divided ends will delay the advance of the axons very considerably.

Embedding.—The slit sheath is gently laid up against the graft, and the incision covered with a strip of dentist's gold leaf. The wound is left wide open, and packed loosely with short wicks of sterile gauze, wrung out of sterile normal salt solution. The gold simply prevents the gauze from sticking to the graft and disturbing it in subsequent dressings. The dressing superficial to the gold leaf should be changed daily, until the whole graft is embedded in healthy granulations. The gold may or may not be removed in two or three weeks. It has often been left in, and has proved quite innocuous. At the end of a month, if the wound is quite healthy, it may be closed by a plastic operation. I have found it a great advantage to postpone the plastic operation for a month. The anaesthesia for this operation makes it possible at such a time to try a faradic current of sufficient strength to demonstrate a response, which will be comforting to both surgeon and patient. Beginning faradic response is the invariable harbinger of returning voluntary and emotional response in the muscles.

The Graft.—For the nerve graft any motor or sensory nerve will do. I have found the anterior femoral cutaneous nerve most satisfactory. It can always be found even through a deep layer of fat. A long transverse incision is made four or five inches below the fold of the groin, down to the fascia lata over the sartorius muscle. The internal saphenous vein comes into view: from a half-inch to one and a half inches external to this, two branches of the anterior femoral cutaneous nerve pierce the fascia lata over the sartorius and run down the thigh. Either branch will furnish any desired length of graft. I have used as much as 200 mm. from one branch. The selected branch is rendered easy of later identification by a suture of heavy black silk, the ends of which are laid in the subcutaneous tissues perpendicular to the skin wound. The nerve is cut and further identified for subsequent dissection by a narrow strip of dental gold wound around the cut end of the distal segment. It is essential that the nerve should not be disturbed in its bed, otherwise Wallerian degeneration may not be complete. I often reject the first 10 or 15 mm. below the cut end for this reason. At the second operation, when the degenerated

nerve is removed for graft material, it is well to take two or three times the measured distance of the gap. You may make a mistake with the first piece or you may decide to employ two or three strands. The nerve once cut across is very difficult to find again. Having all you may want may save you much time in an operation which at best is very long.

The graft should be handled with the utmost gentleness by fine mouse-toothed forceps grasping only the sheath. Squeezing with blunt forceps or other instruments crushes the delicate empty tubes.

Homoplastic Grafts

Owing to an influx of cases there were at the Manhattan Eye, Ear, and Throat Hospital at the same time twelve cases together in my clinic. With the thought of economizing time I determined to see if homoplastic grafts might not be successfully employed. Dr. Eggston, the hospital pathologist, advised that grafts from persons having the same blood group only be attempted, on the principle that homoplastic skin grafts taken from individuals of the same blood group are more successful.

Accordingly, all patients were "grouped." One patient in whom the nerve had undergone preliminary incision and degeneration was found to be of the same group as two others. Grafts from the nerve of this individual were used for all three. The grafts were successful in all of the three cases. Figs. 5 and 6 show recovery following the use of a graft taken from an elderly woman and implanted into a man of the same blood grouping. I have since successfully employed homoplastic grafts in three other cases.

The necessity for the use of homoplastic grafts in repair of the facial nerve may be a rare occurrence. The demonstration of the fact that long grafts caused to degenerate *in situ* rapidly restored function to muscles may be of some importance in peripheral neural surgery, particularly if there comes another cataclysm like the recent Great War.

Conclusion

Tello, in Cajal's work, *Degeneration and Regeneration in the Nervous System,* has shown by physiological experiment the value of degenerated nerve grafts. I hope I have demonstrated their practical application to peripheral nerve surgery. I wish finally to urge upon you the great advantage of immediate investigation of the site of accidental injury in a case of facial palsy. In many instances the removal of a spicule of bone, the lifting of a fractured plate of bone, the decompression and cleansing of ten or more millimetres of nerve with a slitting of the sheath to relieve inflammatory pressure, will ensure an almost perfect recovery, where neglect would be followed by only partial recovery, with grotesque disfiguration for life (see Figs. 7 and 8 on Special Plate).

Moreover, when such an investigation reveals the fact that there is an actual section of the nerve or extensive damage, one can immediately do the preliminary incision of the femoral cutaneous nerve, and, two or three weeks later, transplant a graft from this to replace the gap. In such a case, for forty-eight to seventy-two hours after the initial injury one will have the advantage of being able to pick up the distal segment and verify it by faradic stimulation, and will know something of the problem to be faced in making the transplant later on. No matter what the length of graft necessary in such cases, one may rest assured of recovery if the graft is transplanted successfully, and to do this is a matter of technique (see Figs. 9 and 10 on Special Plate).

The question always arises: "When are we justified in operating on cases of long standing?" I would say: "Operate on any case in which there is galvanic response in the muscles sufficient to show that the muscles have not undergone too much fibrous atrophy. *The nerve can always be repaired.* If there is sufficient muscle fibre left the case will be greatly improved. I am sure the quality of the result will always depend on the condition of the muscle: the time element enters largely into this.

LECTURE II—BELL'S PALSY

In my opinion Bell's palsy presents a condition analogous to the palsy of a facial nerve following injury during operation or following local necrosis and infection. In Bell's palsy toxic products are carried by the blood stream to the sheath of a nerve, confined in a narrow bony tube: a swelling ensues locally. Pressure on the axons produces a palsy of the facial muscles. It may produce pain, herpes oticus, or loss of taste, depending on the position of the inflammatory swelling. Now in the purulent cases from local infection, as well as in the injury cases, where the sheath is directly injured or compressed, the resulting palsy of the face is not due to the presence of pus *per se,* but rather to the inflammatory swelling and consequent mechanical pressure on the confined axons.

If the compression is severe enough, and lasts sufficiently long, the axons will be choked out of existence, and Wallerian degeneration will take place in the nerve distal to that point. If the compression is slight the axons may undergo only a paresis (just as one's arm or leg may "go to sleep" from a cramped position). In this case, although there may be a temporary loss of function, recovery will take place without any degeneration of the nerve cells or fibres. The electrical reaction of degeneration will not occur: faradic response will not be lost. Now if the opportunity were presented of carefully following every case of restoration of function in toxic palsy of the face, I am convinced that these varying degrees of compression would invariably be manifest in the electrical reactions. The idea that every case of Bell's palsy passes through the typical electrical reactions of Wallerian degeneration, either rapidly or slowly, is, I believe, erroneous. On the contrary, I feel certain there are many cases in which Wallerian degeneration does not occur. The axons in the nerve fibres become functionally sluggish or inactive, just as they might from a local anaesthetic, and recover without being degenerated, removed, and replaced. Just what percentage of all cases this type represents has never been determined. A more careful observation of all cases, at their inception, is most essential. It seems evident that early complete loss of faradic response is rare.

In a general way, without accurate statistics, one might infer from reports that of all cases perhaps 80 or 85 per cent. fully recover. I venture the conjecture that a majority of these milder cases never undergo Wallerian degeneration.

The remaining 15 or 20 per cent., I fancy, undergo a violent toxic infection with a severe inflammatory reaction (a compression within the Fallopian aqueduct), which renders the axons useless: in a day or two faradic response disappears. In such cases the regeneration is never complete. The facial movements may be entirely lost for all time, or may make some degree of recovery. The amount of recovery always bears a definite relation to the severity of the initial invasion. The relation of cause and effect in Bell's palsy is quite analogous to that in the accidental cases.

Incision of the Nerve Sheath

I attempted in the first lecture to point out that early intervention, no matter how violent the infection, is likely to lead to a nearly perfect recovery. Why not, then, apply this principle to the *violent* cases of Bell's palsy? Theoretically, leaving them alone is sure to be

followed by an incomplete recovery, with a grotesque appearance for life. Early operation might ensure almost complete return of function even in the cases of most violent invasion. It requires only some accurate means of knowing which cases will recover, and which will not recover, without surgical intervention. I believe that more careful study of the electrical reactions in all cases will eventually enable us to say, at a much earlier stage, that in such-and-such a case, left alone, the recovery will never be complete, whereas if operated on it might probably make a complete recovery. The earlier this determination can be made the better the chance of perfect movements of the face following intervention.

While I was discussing this with Sir Charles Ballance during the earlier years in which we were carrying out the animal experimentations, he repeatedly said: "The cases of Bell's palsy which make a partial recovery and then go on without any further improvement for weeks or months ought to have the nerve in the Fallopian aqueduct uncovered early and be decompressed by incision of the sheath, to relieve the pressure, *at that stage*. Such cases (unless they are appropriately treated) in all probability will go through life with a grotesque deformity." In principle I thoroughly agreed with this opinion. I realized, however, that there is great difficulty in convincing those who first come in contact with these cases that this is sound judgement. The trouble is that those who first see and treat cases of Bell's palsy are very loath to subject them to such a radical procedure as that of uncovering the nerve and incising the sheath, in the hope that the recovery will be sufficiently good to warrant it. The thought of subjecting a nerve to a trauma similar to that which is actually the cause of so many palsies of the face, at a stage when partial recovery has already taken place, is too appalling. Hoping that I might correct what I thought to be an erroneous belief, I tried a series of experiments on rhesus monkeys, in which I uncovered the nerve and incised the sheath.

Ten monkeys on which I incised the sheath of the nerve over an area of 10 to 15 mm. were examined daily afterwards over a long period. Nine of the ten suffered no facial paralysis, no Wallerian degeneration, no loss of faradic response. One of them gradually lost faradic response so that there was evidence of complete Wallerian degeneration at the end of nine days. Seventeen days later, however, faradic response began again, and in six weeks the face had apparently recovered. This case, undoubtedly, had suffered an injury to the axons, or had undergone a slight infection. However, he recovered quite as rapidly as any complete case of Bell's palsy. The other nine seem to prove that the actual trauma caused by incising the sheath, when carefully done, does not cause facial palsy.

I then induced facial palsy on a series of monkeys by exposing and freezing the nerve with ethyl chloride. One case was left without incising the sheath. On all the other cases the nerve sheath was incised over the frozen area, and a few millimetres distal and proximal to that area. The cases on which the nerve sheath was incised recovered facial movements in one-half the time required for recovery in the other cases.

The same experiment was made on a series of monkeys in whom facial palsy had been induced by the injection of 90 per cent. alcohol into the nerve sheath. The cases in which the nerve sheath was incised over the area of toxic involvement recovered twice as rapidly as those in which the sheath was not incised.

Clinical Results

Fortified by this experience I was sufficiently encouraged to incise the sheath of the nerves of several cases of Bell's palsy in which the recovery was very incomplete, and had remained unchanged for many years. In every instance, despite this long period of inactivity, the relief of the pressure by incising the sheath of the nerve has resulted in a very marked improvement. May I give some examples from recent experience?

Case 1.—Miss M., a graduate nurse, appeared at my surgery. She had retired the previous night feeling quite well. She awakened with a complete right facial palsy. Under the pretext of electrical treatments she was seen daily. She never lost her faradic response in the facial muscles, although there were no spontaneous movements for more than a fortnight. At the end of a month she was apparently entirely well. Here is a case with no electrical reaction of Wallerian degeneration of the nerve, despite the fact that there was complete loss of facial movements for a time. This is a perfect example of the type where recovery is complete. I fancy that a very large percentage of Bell's palsy cases are of this type.

Case 2.—Miss D., two years ago, suffered an attack of Bell's palsy, recovering completely in four weeks in practically the same manner as the case I have just recorded. While I did not see her, I fancy she was like the first—a mild attack without Wallerian degeneration. She now appears with a complete palsy on the same side, which has lasted six weeks without any apparent improvement. The electrical reactions of Wallerian degeneration are present. On the ninth week from the onset, conditions remaining the same, at her insistence that something be done, I uncovered the nerve, by the removal of the external wall of the Fallopian canal from the stylo-mastoid foramen to the level of the horizontal semicircular canal, and gently incised the sheath over this area with the sharpest of Graefe knives. In one week faradic response had returned in all facial muscles. The second picture, two months later, shows an apparently complete recovery. Had she gone on for months before operation I believe she would have made only a partial recovery with marked disfiguration for life, as happens in most of these severe cases (see Figs. 11 and 12 on Special Plate).

Case 3.—Mrs. C. Bell's palsy two years ago. After several months some motion began in the face. She now has a return of function roughly estimated at 50 per cent., accompanied by a spasmodic tic in the lower part of the face. The aqueduct was opened from the stylo-mastoid foramen to the level of the horizontal semicircular canal. The sheath was gently incised over this area. It was thickened with scar tissue. A small strand of nerve fibres traversed the area. They were not disturbed. A strand of degenerated anterior femoral cutaneous nerve was laid on this, extending from the proximal segment above to the distal segment peripheral to this area. In one month she declared that she felt much more power in her face, and the mouth was straighter in repose.

Case 4.—Miss S. Right Bell's palsy ten years ago, followed in a few months by partial recovery. Left Bell's palsy six months ago, followed by partial recovery. Recurrent attack on right side, causing complete palsy of the already partially paralysed side. At operation a double decompression by removal of the outer wall of the aqueduct from the stylo-mastoid foramen to the level of the horizontal semicircular canal was carried out. The nerve sheaths were incised over this area. Recovery of equal synchronous movements of both sides of the face, voluntary and emotional control, took place. The result is not perfect by any means, yet the girl has been rescued from social ostracism. This case was figured in my paper in the *Archives of Otolaryngology* (1932, xvi, 773).

I have also two cases of decompression of more than twenty years' standing, both of which, despite the long period, show a distinct improvement of facial movements.

Conclusion

May I say, in conclusion, that the principles of operative treatment of facial palsy, by direct repair of the injured nerve, have been well verified by clinical experience. I believe that, although operative treatment of the severe cases of Bell's palsy is not yet a fixed principle, with continued study of indications for early intervention it will eventually become so. If this should happen, many of the 15 or 20 per cent. of severe cases which are now doomed to go through life with a grotesque disfigurement will be cured by early operation.

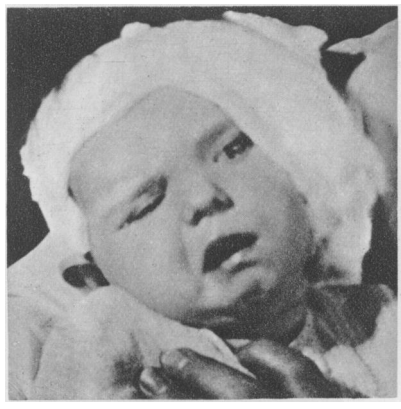

FIG. 1

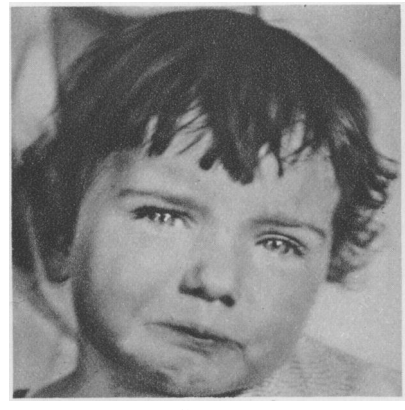

FIG. 2

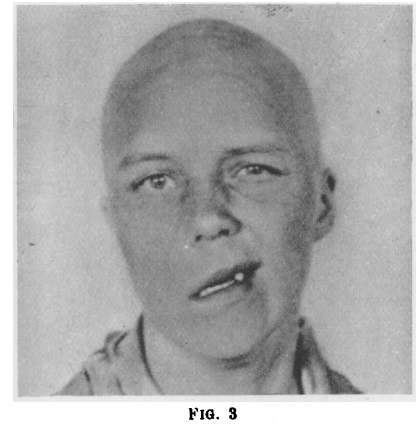

FIG. 3

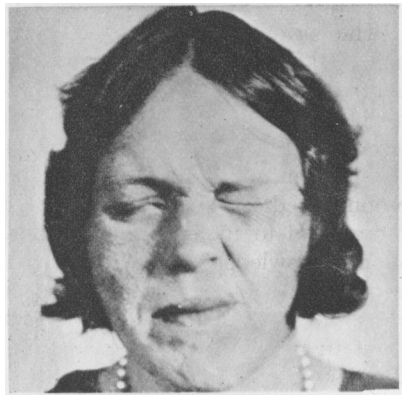

FIG. 4

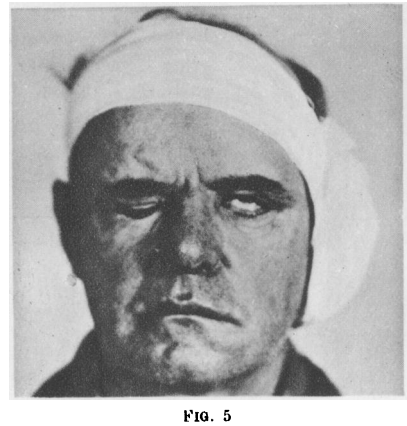

FIG. 5

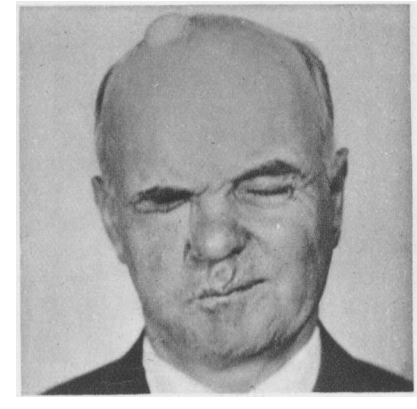

FIG. 6

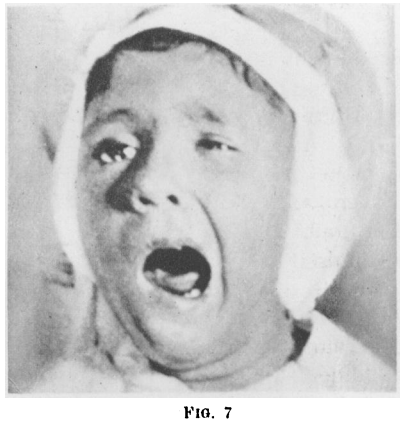

FIG. 7

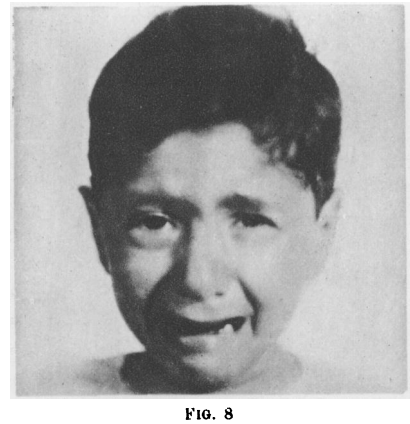

FIG. 8

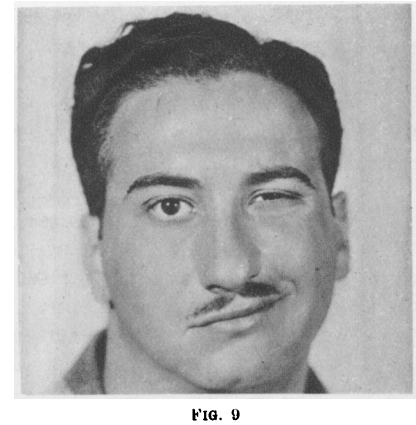

FIG. 9

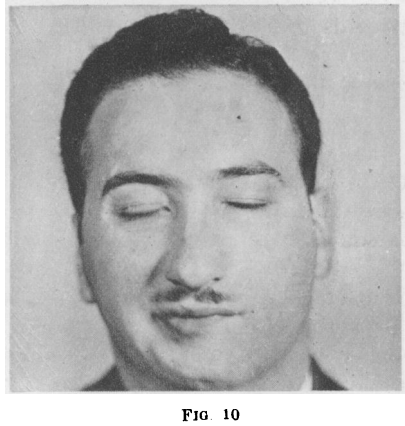

FIG. 10

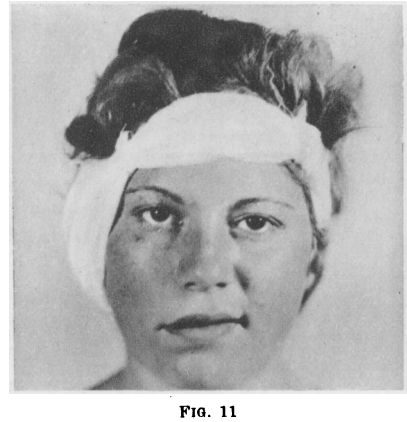

FIG. 11

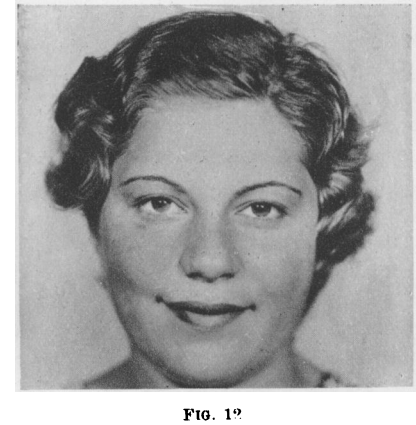

FIG. 12