ON THE PROGNOSIS OF PERIPHERAL FACIAL PARALYSIS OF ENDOTEMPORAL ORIGIN

E. P. J. LAUMANS

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TER VERKRIJGING VAN DE GRAAD VAN DOCTOR IN DE GENEESKUNDE
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PROF. DR. L. B. W. JONGKEES

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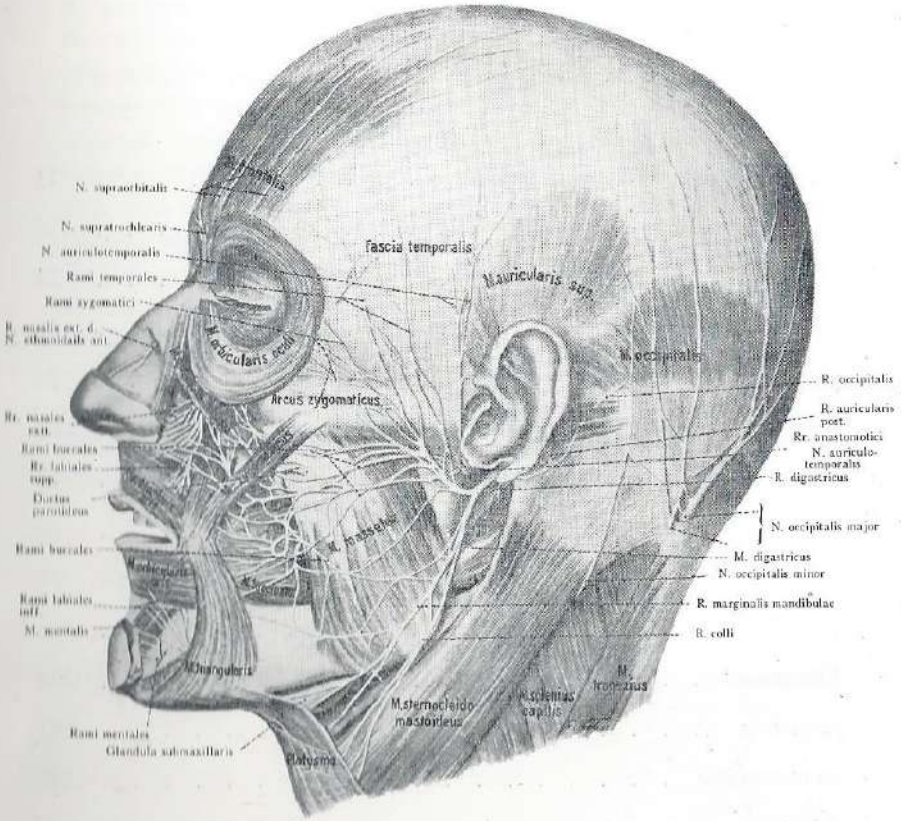

FACIAL NERVE AND MUSCLES OF THE FACE
(after RAUBER-KOPSCH)

INTRODUCTION

AND PROBLEM DEFINITION

Paralysis of the facial nerve is a serious condition. It is a source of considerable discomfort to the patient at eating and drinking; it impedes speech and causes stasis of lacrimal fluid in the eye, which blurs vision and forces the patient to dry the eye continually. Apart from these and other inconveniences, however, it deprives the patient of voluntary and involuntary facial movements, causing the loss of the facial expression which is the unconscious reflection of emotions and moods. More than the organic inconveniences, this loss of the possibility of affective expression causes the patient with facial paralysis great psychological and social difficulties. He is not always capable of coping with these problems, and this may lead to conflict situations. We are acquainted with a female patient who developed a severe depression as a result of traumatic facial paralysis; this depression failed to improve even when surgical decompression led to excellent restoration of nerve function.

An analogous case, but one with a more tragic outcome, is that of a young bricklayer described by MINKOWSKI in 1891; this man committed suicide eight weeks after the onset of spontaneous facial paralysis. At the postmortem, MINKOWSKI made a careful histological examination of the facial nerve and the temporal bone. The report on this examination constitutes the oldest known description of the histological features of the facial nerve in idiopathic (genuine) paralysis.

The abovementioned cases, fortunately, are exceptions, but every patient who knows that his appearance is disfigured by a paralysis, is in a condition of psychological alarm. He is shocked by the uncomprehended event and ashamed in the presence of other people; he lives in anxious uncertainty and doubts his chances of recovery. Of course he attaches importance to the practitioner's reassurances, but an ultimate relief of tension does not occur until he himself has noticed the first signs of improvement. A rapid

onset of recovery, therefore, is of paramount importance, not only with a view to the patient's mental disposition but also in terms of the vital chances of the affected nerve. Slightly paraphrasing a statement by COHEN (1960), therefore, we would be inclined to say: "Facial paralysis, a medical emergency!".
This implies that adequate therapeutic measures must be taken immediately; frequent follow-ups should be made to evaluate the effect of the treatment instituted, and this treatment should be altered when insufficient or no improvement has occurred within a short time. The fact that many therapists assume an expectant attitude or continue conservative therapy, once started, for several weeks or months although no improvement occurs, is perhaps explained by the view held by some, that the course of a facial paralysis can hardly be influenced, if at all, or by the experience that this condition generally has a good prognosis and shows a spontaneous remission in many cases. For example, the chance of spontaneous remission in Bell's palsy is estimated by many authors at 75-85% (CARTER, 1952; GARCIN, 1954; CAWTHORNE, 1956; BOONE, 1959; KETTEL, 1959). Other investigators have a less favourable expectation, e.g. PARK & WATKINS (1949) 66%; VERJAAL (1955) 67%; MATTHEWS (1959) 53%; TAVERNER (1955) not over 45%. The same holds true for facial paralysis following cranial injury, in which the chance of spontaneous recovery is estimated by some at as high as 80-90% (GROVE, 1939; TURNER, 1944; ROWBOTHAM, 1945; GURDJIAN, 1956; KAHN, 1956). This optimism, however, is not entirely shared by some other investigators, e.g. ROBSON & DAWES (1960).

As to the cure percentages presented, it should be pointed out that these are largely dependent on the criteria employed by the authors. Unfortunately, these criteria are not always mentioned so that the exact meaning of the term "cure" often remains obscure. Obviously this qualification does not have the same meaning for every author, as demonstrated by a comparison of percentages given for Bell's palsy. TAVERNER, who applies very strict criteria, was unable to exceed 45%. Apart from this, the abovementioned figures apply to the groups *in general*, independent of the severity of paralysis in individual cases. It has been found, however, that the prognosis in cases of complete paralysis is considerably less favourable than that in partial paralysis. For Bell's palsy, CAWTHORNE & HAYNES (1956) found that only 47 of 111 patients with

complete paralysis (42%) made a full recovery. In the case of paralysis of traumatic origin, moreover, it is of fundamental importance whether the paralysis occurred immediately after the injury or after a certain free interval.

While the chances of spontaneous recovery are relatively good in the abovementioned groups, they are definite unfavourable (in fact they are non-existent) in paralysis resulting from chronic otitis media, a severe surgical trauma or a tumour. It is not rational to take an expectant attitude or to institute conservative therapy in such cases. However, even the most favourable statistics show that also in the group of Bell's palsy and cranial injuries 10-15% of cases show no complete recovery. The patients only partly recover, often only after a prolonged period; some patients do not recover at all. A percentage of 10-15 is not small; in our opinion, it is certainly too large to justify an exclusively conservative attitude. With regard to the diagnosis "Bell's palsy" — a diagnosis by elimination — experience has shown, moreover, that there is always a possibility that the condition will ultimately prove to be, not Bell's palsy but a paralysis resulting from latent otitis (VAN OPPENRAAY, 1959; FEMENIC & SUBOTIC, 1960) or from an incipient tumour (KETTEL, 1959).
JONGKEES (1957) summarized his point of view in this connection as follows: "Conservatism has its limits, because otherwise disfigurement threatens patients who could have been saved easily by an operative procedure", and RODRIQUEZ & SKOLNIK (1954) stated: "Conservative management of peripheral seventh cranial nerve paralysis must be based on careful diagnosis and scientific assessment of the status of the facial nerve". Consequently we should like to emphasize that all patients suffering from facial paralysis should be considered serious cases from the beginning and be treated without delay.

According to the treatment of choice, cases of endotemporal facial paralysis can be classified as follows.

I. Cases exclusively suitable for *conservative* treatment, in which group we include:

Facial paralysis due to	AURAL HERPES ZOSTER (RAMSAY HUNT's syndrome)
" " "	LUETIC NEURITIS

II. Cases to be treated exclusively and as early as possible in a *radical* manner (surgery, radiotherapy), in which group we include:

Facial paralysis due to		CHRONIC OTITIS MEDIA
"	"	" INTRATEMPORAL TUMOURS
"	"	in direct connection with OPERATIONS ON THE TEMPORAL BONE
"	"	" FRACTURES OF THE TEMPORAL BONE (if the lesion is accessible)
"	"	" EXTERNAL VIOLENCE TO THE MIDDLE EAR OR MASTOID BONE

III. Cases for which there is no fixed plan of therapy, the treatment of choice being determined in each individual case in accordance with its severity and the therapist's views; in this group we include:

		BELL'S PALSY
Facial paralysis due to		ACUTE OTITIS MEDIA
"	"	" EXTERNAL OTITIS
"	"	of delayed onset after OPERATIONS ON THE TEMPORAL BONE
"	"	" FRACTURES OF THE TEMPORAL BONE (if the lesion is accessible)
"	"	" EXTERNAL VIOLENCE TO THE MIDDLE EAR OR MASTOID BONE

With regard to the treatment of facial paralysis from causes as mentioned in group I and II, there is hardly any difference of opinion in current international literature; unfortunately, such a difference persists in practice; many of the group II patients are referred to the surgeon only after too long a period of conservative treatment or expectation.
The situation is entirely different for the cases collected in group III. Here, the advocates of exclusively conservative treatment and those of early surgical intervention are joined by those therapists (the majority) who start with conservative measures but switch to surgery when insufficient or no improvement is seen. The various authors differ considerably as to the reasons for which the original therapeutic approach is changed and the time at which surgery is resorted to. There is no uniformity of action in this respect; every author bases himself on personal understanding and experience. This absence of a fixed rule of approach has its cause in the fact that, for lack of reliable tests, it seems impossible with the present body of knowledge to distinguish — in the vital early stages of facial paralysis — such cases as are unlikely to show complete recovery. Treatment, therefore, is of necessity based exclusively on clinical experience. In Bell's palsy, for example, the general opinion is that patients who show no unmistakable improvement of facial function within 6-8 weeks of the onset of paralysis, have only a small chance of full recovery. Many workers carry out decompression of the facial nerve in the Fallopian canal by that time. However, JAMES & RUSSELL (1951) state that: "Decompression of the facial nerve in the facial canal six to eight weeks after onset seems an irrational procedure, for, if decompression really helps it should be done within two weeks of the onset, when there is first evidence of nerve degeneration"; and PARK & WATKINS (1949) said: "If decompression were done within a few hours after the onset of facial weakness, it would be theoretically possible that in no case would a complete reaction of degeneration develop".
These statements sound very plausible and are in accordance with the views held by most authors; as early as 1936, however, TUMARKIN complained: "We are tantalised by the feeling that if only we could have operated the first few days of their illness, we could certainly have saved many patients. But supposing we do see those cases in the first few days of their paralysis, how dare we advise operation when we know that any given patient has a four to one chance of spontaneous recovery".

This was the problem 25 years ago, and it is still the great problem today. It would seem that, if some method could be found of selecting cases with a serious prognosis in the very early stages of facial paralysis, the decision as to the therapeutic approach to be adopted would be greatly facilitated (WILLIAMS, 1959). The detection of such a method has been the object of our investigation; the results obtained have been collected in this thesis and are submitted to detailed consideration. We have confined ourselves to peripheral facial paralysis of endotemporal origin, as included in the abovementioned group III.

DEGENERATION AND REGENERATION

Every cross-striated muscle fibre is connected, via its own motor end-plate, with one of the terminal branches of one motor nerve fibre; this motor nerve fibre is the axis cylinder (axon) of a motor cell of the central nervous system (anterior horn cell of the spinal cord or motor cell of a motor nucleus of one of the cerebral nerves). One motor cell, its axis cylinder and all muscle fibres connected with it, together form what SHERRINGTON (1925) called a "motor unit". (fig. 1)

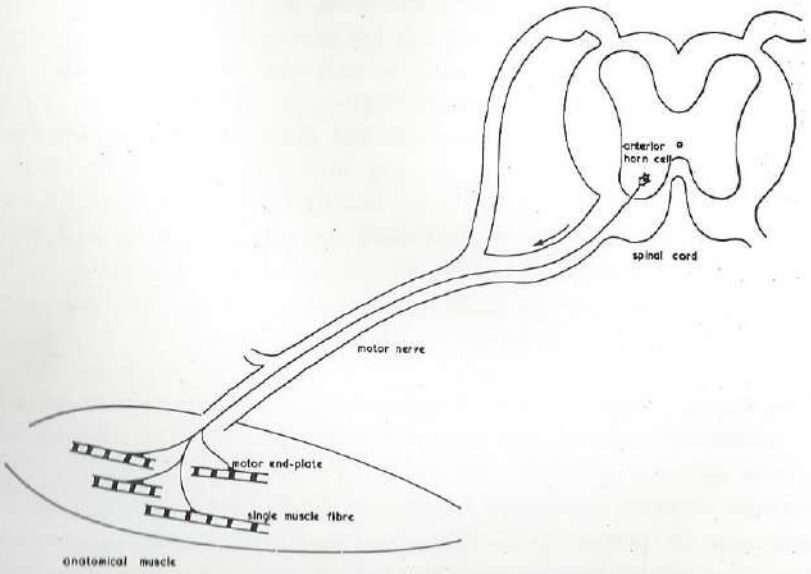

Fig. 1. Schematic drawing of a motor unit.
(freely after E. Fisher, in S. Licht: Electrodiagnosis and Electromyography, New Haven 1956)

Under normal conditions a muscle fibre can only contract when it receives the appropriate stimulus via its motor nerve; if the conduction of such a stimulus is lacking as a result of a lesion of the motor nerve, no muscle contraction is possible. According to the severity of the situation, SEDDON (1943) introduced the following classification of peripheral nerve lesions, based on histological findings.

NEURAPRAXIA: interference with function, unaccompanied by a degeneration of nerve distal to injury, but probably associated in the more severe cases with interruption of continuity of the myelin sheath at the level of the injury. This is the mildest degree of damage to a nerve.

AXONOTMESIS: division of the axon with peripheral degeneration of both axon and myelin sheath, but without appreciable damage to the neurilemmal and endoneurial sheaths. Because the Schwann sheath remains intact, the regenerating axis cylinder has a guide; consequently it less readily enters an incorrect pathway, and this greatly benefits the quality of functional restoration.

NEUROTMESIS: complete disruption of the essential elements of the nerve, though not necessarily accompanied by gross section of continuity. Complete degeneration of the axis cylinder distal to the site of injury consequently occurs. Since the continuity of the Schwann sheath has also been interrupted, the regenerating axis cylinder lacks a guide. The chances of good functional restoration in neurotmesis are therefore less favourable than those in axonotmesis.

This classification was originally introduced by SEDDON for nerve paralyses of traumatic origin, but it can also be applied to paralyses of different aetiology. The schema is of attractive simplicity and has been accepted by many investigators, e.g. BAUWENS, BIEMOND, BOWDEN and, particularly in paralyses of the facial nerve, by BOONE, KETTEL and MIEHLKE. Naturally, various transitions between the abovementioned three conditions occur, and mixed lesions are encountered in many cases.

Restoration of the physiological function of nerve conduction is closely correlated with the severity of the nerve affection. In neurapraxia there is only loss of conductivity across the lesion, and recovery is usually rapid

and complete. Terms frequently used in this connection are "reversible nerve block" or "physiological nerve block". In axonotmesis and neurotmesis, the axis cylinder and the myelin sheath — which are organic parts of the neuron and therefore trophically dependent on the cell body (MAXIMOW & BLOOM) — have undergone complete degeneration. Functional restoration in these cases is possible only by regeneration of the degenerated peripheral portion of the nerve fibre. This regeneration consists of outgrowth of the central axon stump; each axon divides into a number of sprouts, which continue to grow to the periphery and finally contact the muscle fibres. The Schwann cells, which are trophically independent of the neuron, play an important role in this regeneration. Proliferation of the Schwann cells is seen both in axonotmesis and in neurotmesis; they scavenge the fragments of the disrupted axis cylinder and in axonotmesis — where the Schwann sheath itself is undamaged — the cells grow out and constitute a guide for the newly formed axon sprouts. If the nerve lesion was so severe that the Schwann sheath and the perineurium are also injured (neurotmesis), regeneration is much more difficult. Again there is outgrowth of sprouts from the central stump, but these sprouts have no guide because the Schwann sheath is also damaged; in neurotmesis the proliferation of Schwann cells is much more uncontrolled than in the case of axonotmesis; particularly if the gap between the central and the peripheral stump is wide, there is wild proliferation of Schwann cells which, with the connective tissue reaction which occurs in the perineurium, impedes the outgrowth of the young sprouts. As a result, these roam about and, for a large part, fail to contact the paralysed muscle fibres. This explains why neurotmesis has a much poorer chance of good restoration of the nerve function than axonotmesis.

If degeneration is incomplete, the nerve fibres which have remained unimpaired become active aids in reinnervation of the denervated muscle fibres. In addition to the outgrowth of sprouts from the central stump of the degenerated nerve fibres (terminal regeneration; terminal sprouting) outgrowth of nerve rami occurs from the residual nerve fibres (collateral regeneration; collateral sprouting). This collateral sprouting is mainly seen in the region of the terminal branches of the intact nerve fibres in the muscle, but also at a more proximal level along the length of the nerve fibres themselves. These phenomena were observed in animal experiments and described by HINES, WEHRMACHER & THOMSON (1945), VAN HARREVELD

(1945) and WEISS & EDDS (1946). Their observations have been confirmed by several other investigators. The fact that collateral regeneration can also occur in man has been demonstrated by WOHLFAHRT (1955, 1958). Detailed histological data on these phenomena were published in a recent study by COERS & WOOLF (1959).

Data on the rate of growth of the regenerating axon tips are widely varied. At present the only method of determining this rate is by dividing the length (in mm.) of the distal segment of the nerve by the number of days elapsed since the injury; only a rough approximate can be obtained in this way. There are numerous factors which influence the rate of regeneration, e.g. the distance between the lesion and the cell body, the length of the distal segment, the blood supply, etc.. There is also a difference between experimental and clinical values, and between nerve lesions with intact continuity and those in which a nerve suture or nerve graft was carried out.

There is an initial delay (scar delay) before the apparent beginning of outgrowth of the axon tips, and probably also some delay between the arrival of the axon tips and the formation of functional connections with the end-organs. Scar delay in animal experiments was found to average 7-10 days (GUTMANN, 1942; JASPER, 1945; GOLSETH & FIZZELL, 1947; BOWDEN & SHOLL, 1954).

The first sign of reinnervation can be found either in the return of motor unit action potentials in the electromyogram or in the first detectable visual muscle movement. Return of motor unit action potentials precedes return of visible muscle movement by an average of 8 weeks.

TABLE I. AVERAGE RATES OF FIBRE GROWTH IN ANIMAL EXPERIMENTS.

	return of action potentials	*visible muscle movement*
JASPER (1946)	2.5 mm./day	1.5 mm./day
GOLSETH & FIZZELL (1947)	2.6 mm./day	2.0 mm./day
YOUNG (1949)	3.8 mm./day	
BOWDEN & SHOLL (1954)	3.9 mm./day	

TABLE II. AVERAGE RATES OF FIBRE GROWTH IN MAN.

	return of action potentials	*visible muscle movement*
JASPER & BALLEM (1949)		
ulnar, radial, median nerves	2.5 mm./day	
peroneal nerve	1.6 mm./day	
BOWDEN (1954)		
(22 cases of radial nerve lesion)		
axonotmesis	2.2 mm./day	1.8 mm./day
nerve suture	1.2 mm./day	0.9 mm./day
others	1.4 mm./day	1.0 mm./day

According to these data, the rate of regeneration in man is about 1-2 mm./day.

Restoration of nerve function after degeneration is possible only by regeneration. Restoration by regeneration is never complete; there are always *sequelae*, the most important among which are incomplete return of voluntary and affective movement, synkinesis and contracture. These sequelae are particularly unmistakable in the face, in which abnormal movements and a difference in facial expression between the normal and the affected side immediately draw attention. Numerous publications have been devoted to these phenomena.

SYNKINESIS

Synkinesis (associated movement) is the phenomenon of a voluntary or reflex contraction of one muscle being accompanied by synchronous involuntary contraction of one or several other muscles, e.g. lifting of the angle of the mouth or dimpling of the chin on closing the eyes or raising the eyebrows; narrowing of the palpebral fissure at showing the teeth.

The finest example of synkinesis as a result of reflex contraction is twitching of the muscles around the mouth at blinking.
Synkinesis may show varying degrees of intensity; in some cases it is hardly perceptible, and in other cases it is so pronounced that normal movements of facial expression and speaking, eating and drinking are accompanied with very disturbing facial twitching. It may even be that a movement such as frowning cannot be executed as voluntary movement but only as synkinesis (e.g. by baring the teeth). The same holds true for reflex synkinesis; in attempting to provoke the nasopalpebral reflex in the convalescent phase of peripheral facial paralysis, we repeatedly observed that percussion between the eyes was not followed by the normal contraction of the orbicularis oculi muscle but by contraction of a muscle elsewhere in the face, e.g. twitching of the angle of the mouth. Many investigators have pointed out that the degree of synkinesis is independent of the nature of the nerve lesion or its localization; only the severity of the lesion is of importance in this respect.

OPPENHEIM (1911) reports that patients recovered from traumatic nerve lesions, invariably showed abnormal movements of the facial musculature. BALLANCE (1934) noticed the same in his experiments in monkeys submitted to grafting of the facial nerve after section. DUEL (1934) performed this operation in patients with facial paralysis and found that all showed the associated movements so apparent in the experiments in monkeys. HOWE, TOWER & DUEL in 1937 again carried out extensive experiments in monkeys in order to find an explanation for these abnormal movements. Again, associated movements were observed in all cases of recovery from facial nerve lesions. MARTIN (1944) states that cases of posttraumatic facial paralysis do not often show associated movements; he observed synkinesis, however, in many cases of Bell's palsy whenever an attempt was made at strong volitional movements. TICKLE (1948) states "Synkinesis or associated movements are always present after a severe injury to the facial nerve". According to JAMES & RUSSELL (1951), associated movements are an inevitable consequence of nerve regeneration. TAVERNER (1955, 1959) examined a large number of patients with Bell's palsy and reached the conclusion that all patients whose paralysis was of such severity as to lead to denervation, showed some degree of synkinesis or other sequelae. In our own patients we saw these associated movements in all cases of severe facial paralysis, regardless of the cause of paralysis.

The *pathogenesis of synkinesis* has long been a subject of much discussion. GOWERS and HITZIG (1872) assumed a nuclear origin. Their views are supported by such authors as REMAK (1898), BITTORF (1931), PETZ (1933), WARTENBERG (1946) and WILDHAGEN (1952). Although the views held by these investigators differ in detail, they are based on the same fundamental theory — that severe facial nerve lesions are associated not only with degeneration of the peripheral nerve segment but also with retrograde changes in the ganglion cells of the facial nerve nucleus. As a result, these cells become hyperexcitable and consequently they respond to a wide variety of stimuli, e.g. to radiation (REMARK, BITTORF) of motor stimuli arriving from higher centres in the adjacent ganglion cells (which have or have not remained intact). WARTENBERG refers to a "release phenomenon", by which he means that, in the cells affected, loss of control on the part of the higher centres has occurred. In this way, differentiated innervation of separate muscles or muscle groups is impossible; simultaneous innervation of various muscles governed by the facial nerve nucleus occurs. In the same way, it is believed, a reaction to provocation of the nasopalpebral reflex takes the form of synchronous contraction of several muscle groups. WILDHAGEN reports that he has electromyographically confirmed this so-called "mass innervation". By means of needle electrodes, he simultaneously derived action potentials from the frontal muscle, the orbicularis oculi and the zygomaticus major; whenever an attempt was made at isolated contraction of one of these muscles, action potentials were also recorded in the two other muscles, and these action potentials were of the same appearance and intensity in the three derivations.

LAMY (1905) and LIPSCHITZ (1906) are of a different opinion. They contend that, if the retrogradely injured ganglion cells indeed recover only incompletely, a decrease in excitability (analogous to the decreased electrical excitability in the regenerating nerve) is to be expected rather than increased excitability. According to LAMY and LIPSCHITZ, associated movements can be explained as follows. Sprouts which grow out at regeneration of the central axon stump, do not reach the appropriate pathways; they grow downward in every direction and reinnervate distal branches, to supply muscles with which no connection originally existed. The synchronous movement of different parts of the face is explained by the innervation of several muscles by neural fibrillae split from a single axon. This explanation was confirmed in detailed experiments and

anatomical and histological findings obtained by HOWE, TOWER & DUEL. These investigators pointed out that the origin of the adventitious facial movements was situated low in the motor pathway. The presence of branched axons offered the only anatomical and histological explanation of this phenomenon.

The theory of misdirection of regenerating fibres as mechanism underlying synkinesis has been supported also by FOWLER (1939), MARTIN (1944), TICKLE (1948), HILGER (1949), JAMES & RUSSELL (1951), KETTEL (1959) and many others. WEDDELL, FEINSTEIN & PATTLE (1944) demonstrated associated movements by electromyography in a case of suture of the ulnar nerve. BOWDEN (1954) did the same in 47 cases after suture of the ulnar nerve. TAVERNER (1955, 1959) and ESSLEN (1960) did the same in patients with peripheral facial paralysis. By simultaneous electromyographic examination of two or more facial muscles, using concentric needle electrodes, ESSLEN attempted to establish whether the associated movements in 50 patients with peripheral facial paralysis were a result of complex misdirection (mass movements, WILDHAGEN) or of simple misdirection. ESSLEN uses the term simple misdirection with reference to the situation in which only one axon sprout contacts a foreign muscle, whereas in complex misdirection two or more sprouts are misdirected to different muscles. Complex misdirection was found in only one of these 50 patients, while the remaining 49 all showed simple misdirection. In his summary, ESSLEN states: "The theory of misdirection of regenerating axons seems so well-founded by clinical, electrophysiological and histological observation that the (former concurring) hypothesis of irritation of the nuclear region, promoted originally by GOWERS (1879), must be considered obsolete".

In cases of partial denervation, associated movements may arise, not only from misdirection of regenerating nerve fibres (terminal sprouts) but also from additional misdirection of the sprouts which grow out from the unimpaired residual axon trunks (collateral sprouts). This possibility has been especially pointed out by BOONE (1959).

According to MORRIS (1939), associated movements disappear in time as recovery becomes complete; MARTIN (1944) described two children with traumatic facial paralysis; one showed synkinesis for 30 months after recovery, after which it ceased; in the other child the tic-like movements lasted for 2 years after recovery. The majority of authors agree, however,

that associated movements never disappear completely (LIPSCHITZ, HOWE, FOWLER, WILDHAGEN, TAVERNER. Our experience agrees with this; associated movements, once established, showed no diminution over a follow-up period of up to 3 years; there is no conceivable reason why this situation should subsequently change.

Faulty outgrowth of nerve fibres can give rise to other phenomena also. The most widely known is the "*crocodile tear*" phenomenon or paroxysmal unilateral lacrimation when eating. This is believed to result from misdirection of secremotor fibres orginally extending into the chorda tympani; during recovery after involvement of the 7th nerve at or proximal to the geniculate ganglion, these fibres are diverted into the greater superficial petrosal nerve, thus reaching the lacrimal gland. JAMES & RUSSELL found this phenomenon in 6 of 28 patients with denervation of facial muscles; TAVERNER (1959) saw it in 15 of 164 patients and LEONHARD (1960) in 5 of 36 patients of the degeneration group. BOYER & GARDNER (1949) reported that this disability can be relieved by section of the greater superficial petrosal nerve. PESCH & GRINDA (1960) advise section of the chorda tympani in order to eliminate the centripetal paths of the reflex.

CONTRACTURE

Contracture of a voluntary muscle is clinically defined as fixed shortening of the fully relaxed muscle (DENNY-BROWN, 1953; BOWDEN, 1954; MATTHEWS, 1959). Contracture should be clearly distinguished from spasm, i.e. the occurrence of paroxysmal involuntary muscular contractions of varying duration. Both conditions may occur following paralysis, and after facial paralysis they are known as "postparalytic facial contracture" and "postparalytic hemifacial spasm". Unfortunately, these designations are sometimes indiscriminately used, e.g. DENNY-BROWN & PENNYBACKER (1938) use the term postparalytic facial contracture with reference to a "curious type of involuntary contraction and spasm". According to ALAJOUANINE & THUREL (1934), postparalytic hemifacial spasm is always based on a contracture: "on the basis of a permanent contracture, fascicular or fibrillar paroxysmal contractions may occur; the paroxysmal contractions disappear during sleep but the permanent contracture persists, although it may be less pronounced".

Contractures, particularly those in the face, are unmistakable; this is because the facial muscles have no fixed point of insertion but insert directly into the skin or subcutaneous tissue. Facial contractures are manifested by, for example, abnormal depth of the nasolabial fold, deviation of the philtrum to the affected side or narrowing of the palpebral fissure when the face is in repose.
A contracture is not always an undesirable and troublesome residual symptom; particularly in cases showing only poor restoration of muscle function, contracture formation is a welcome phenomenon which contributes to the symmetrical appearance of a previously drooping face. It is a remarkable fact that contracture formation is most pronounced precisely in the presence of severe residual paralysis; contracture formation is as a rule only slight in the case of good functional restoration.

The *nature of contracture* formation is still uncertain. Opinions, like those on the phenomenon of synkinesis, differ. HITZIG, OPPENHEIM, MYERSON, GRINSTEIN, BITTORF and WILDHAGEN point out that contractures are always seen in concomitance with associated movements and they assume the same nuclear origin for both phenomena, viz: hyperexcitability of the facial nucleus. LIPSCHITZ agrees that associated movements are usually seen in concomitance with contractures; the reverse, however, is not always true in his opinion, and he consequently believes that the two phenomena cannot be based on the same mechanism. Our experience is in accordance with HITZIG's; of 115 patients with peripheral facial paralysis who recovered incompletely all 115 showed associated movements, whereas contractures were seen in 94. SPILLER (1919) believes that stimuli are constantly being transmitted from the injured nerve part to the muscles which consequently cannot relax; as a result they gradually shorten, and contracture arises. MARINESCO, KREINDLER & JORDANESCO (1931) support this view because they succeeded, in two cases, in complete control of postparalytic facial contracture by procaine block of the pre-auricular facial branches. LIPSCHITZ explains the contracture formation on the basis of atrophy and degeneration of the denervated muscle fibres, giving rise to fibrosis and shrinking. SAHLI, FUCHS, TOWER (1939), TEN CATE (1945), DENNY-BROWN (1953) and BOWDEN (1954) share this opinion; BOTMAN (1954) speaks of "hypertonicity" of the muscles. FEINSTEIN (1946) ascribes the contracture to the presence of areas of "fibrous metaplasia"; at electromyographic examination of contracture patients he found many fields of "electrical

silence". RICHARDSON (1951) on the other hand, mentions grouped discharges at electromyographic examination as the earliest sign of facial contracture; TAVERNER (1955) also states that contracture must be regarded as a "sustained contraction of facial musculature; contracture was found to be associated with the continuous low-frequency firing of single motor units with no recognizable pattern".

In many of our patients contractured muscles were electromyographically examined by means of insertion of concentric needle electrodes into the muscles under examination. In no case did we find the abovementioned discharges if the patient succeeded in attaining complete relaxation; the clinical features of contracture remained unchanged in repose. We are inclined to believe that contracture is indeed based on fibrosis of the muscle; the muscles concerned are hard to firm on palpation, and the same sensation is experienced when the needle electrodes for electromyography are inserted. The patient subjectively experiences the presence of a contracture as a sensation of stiffness.

If, however, fibrosis and connective tissue shrinking resulting from atrophy and degeneration of denervated muscle fibres are accepted as the cause of contracture formation, then it is difficult to understand why no contracture formation is seen in cases without any reinnervation, in which paralysis remains complete. There is maximal atrophy in these cases, the muscles remain flabby and no contracture occurs. With regard to facial paralysis, this has been pointed out by KETTEL. We have seen the same in our patients; complete paralysis persisted in 9 of our series of 220 cases (5 tumours, 2 surgial traumas, 1 head injury, 1 uncertain cause); in none of these cases was contracture formation seen.

The literature frequently states that imperfectly adjusted *galvano-therapy* (too protracted, too high voltage) promotes contracture formation. This is a delicate point; the criteria of correct application of electrotherapy are not invariable. FEINSTEIN (1946) says that fibrosis of the muscles is seen precisely when electrotherapy has been insufficient. LIPSCHITZ, JAMES & RUSSELL and MOSFORTH & TAVERNER (1958) found no correlation between electrotherapy and contracture formation. Our own experience in this respect is that contracture formation also occurs when no electrotherapy has been used, and that the abovementioned 9 patients with residual complete paralysis developed no contracture despite protracted galvanotherapy. The true mechanism of contracture formation, so far as we know, is still obscure.

Incomplete return of voluntary movement, synkinesis, contracture and other sequelae of regeneration cannot be cured by any known medical or technical method; they can only be minimized. Patients can be trained to suppress their facial movements and reduce emotional expression. If considerable facial deformity exists, much benefit can be derived from correction by plastic surgery.

All this trouble might be prevented if it were possible to prevent the occurrence of degenerative changes in the nerve fibres.

EXAMINATION OF THE PATIENTS

In the first chapter the various forms of endotemporal facial paralysis were classified according to the mode of treatment — conservative or radical (surgical, radiological).

A number of patients remained — group III — whose common denominator was precisely the lack of a uniform therapeutic plan. As has been pointed out, the chief reason for this is the uncertain and variable prognosis in these cases. In this group Bell's palsy occupies by far the most important position. This is not surprising because this type of paralysis also constitutes the largest proportion of all cases of peripheral facial paralysis in general (MERWARTH, 1949: 70%; PARK & WATKINS, 1949: 75%; CAWTHORNE & HAYNES, 1956: 62%). We diagnosed Bell's palsy in 144 of a total of 220 patients with facial paralysis examined in the course of three years, i.e. 52% (see Table III).

TABLE III. CAUSES OF PERIPHERAL FACIAL PARALYSIS IN 220 PATIENTS

Bell's palsy	114 (52%)
Surgical trauma	27
Head injury	22
Acute otitis media	13
Chronic otitis media	15
External otitis	1
Aural herpes zoster	10
Tumours	6
Infectious mononucleosis	2
Congenital	3
Uncertain	7
	220

Selecting from this series those patients who, according to the classification given in the first chapter, are included in group III, we obtain a total of 141 (see Table IV).

TABLE IV. CAUSES OF PERIPHERAL FACIAL PARALYSIS IN 141 GROUP III PATIENTS

Bell's palsy	114 (80%)
Surgical trauma	7
Head injury	6
Acute otitis media	13
External otitis	1
	141

This table demonstrates that the patients with Bell's palsy constitute about 80% of the group. A possible similarity in pathogenesis between Bell's palsy and the other types of paralysis in group III would be of great importance in determining the correct mode of treatment for the group as a whole.

In order to be able to discuss the problem, we will have to define the term "*Bell's palsy*", as we use it. Bell's palsy is a diagnosis by exclusion — a tentative diagnosis —, which means that we use the term Bell's palsy only in the absence of any other demonstrable or plausible cause. Cases of facial paralysis associated with aural herpes zoster are included in statistics on Bell's palsy by some investigators, e.g. TAVERNER (1955), MATTHEWS (1959), DALTON (1960) and GREGG (1961), but in our opinion these cases do not belong to this group.

Yet Bell's palsy is not a general term referring to a number of poorly differentiated paralytic pictures; it is a type of facial paralysis with its own anatomical and histological substratum. Clinical diagnosis is impeded by the lack of a characteristic symptom complex; the only strikingly constant feature is the acute onset of the paralysis in a completely healthy subject. In the absence of other anomalies it is precisely this anamnestic datum that indicates the probability of Bell's palsy. The pathology and aetiology of this condition are not yet entirely known; ischaemia of the facial nerve as a result of local vasomotor dysregulations (vasospasm) is regarded as the most likely cause by many investigators (WORMS &

CHAMPS, 1931; AUDIBERT, MATTEI & PAGANELLI, 1936; HILGER, 1949; SOKCIC, 1954; JONGKEES, 1955; LHOTSKY, 1958; KETTEL, 1959; FURSTNER, FEJES & KRALOVANSKY, 1960; MIEHLKE, 1960; KORKIS 1961).
Today, as in the past, the term Bell's palsy is often replaced by "rheumatic facial paralysis" and "paralysis a frigore" — designations which suggest a certain aetiology. Qualifications such as "idiopathic", "cryptogenic" or "genuine" leave the aetiology undecided. The designation "ischaemic paralysis" is in accordance with modern views on the pathogenesis of the condition. However, so long as the pathogenesis and aetiology are not definite, it is in our opinion advisable to adhere to the name "Bell's palsy" — a designation introduced in appreciation of the work of Sir Charles BELL who, in 1821, demonstrated that the facial nerve is the motor nerve of the face.

Ischaemia of a nerve as a cause of its dysfunction in our opinion also affords a plausible explanation in cases of facial paralysis of delayed onset following head injury and surgical trauma, and in cases of palsy seen in acute otitis media and — though rarely — in cases of external otitis.
Facial paralysis of delayed onset after *head injury* (fractures of the temporal bone) can arise from a circulatory disturbance in the nerve as a result of pressure exerted by loose bone fragments, blood along the fracture lines or dehiscences in the Fallopian canal, swelling of the mucosa of the canal caused by oedema or secondary infection, haemorrhage in the nerve sheath or in the nerve itself, etc. All these factors may lead to the occurrence of what ULRICH (1926), on the basis of histological examination of the temporal bone and the facial nerve in a large number of cases of head injury, described as a "secondary neur*itis* in the widest sense of the word".*) JAMES & RUSSELL (1951) considered "the behaviour of the affected nerve in Bell's palsy remarkably similar to the behaviour of cases of traumatic facial paralysis following fracture of the skull". SOKCIC (1954), in 5 patients in whom he decompressed the facial nerve because of paralysis which occured a few days after a head injury, observed features similar to those seen in Bell's palsy, viz: considerable oedema of the nerve in the region between the stylomastoid foramen and the second genu. He believes that this suggests a circulatory disturbance in the nerve as a cause of post-

*) The term "neur*itis*", although not unusual, is in fact inadequate when used with reference to a condition not entailing inflammation.

traumatic paralysis of delayed onset. VON SCHULTHESS & DUBS (1957) also assume that the cause of late posttraumatic facial paralysis is the same as that of Bell's palsy, namely a circulatory disturbance. They consequently advise conservative treatment on the same lines as used in Bell's palsy during the first few days, in anticipation of spontaneous recovery. GREINER, KLOTZ, GAILLARD & ISCH (1960) likewise maintain that "it is the vascular phenomena that are of essential importance; the pathogenesis of these traumatic facial paralyses can be compared, within limits, with that of paralysis a frigore. Compression by a haematoma is probably less often causative than ischaemic changes, which may entail cylinder axis degeneration on the one hand and, on the other, promote oedematous exsudation — which accentuates both compression and ischaemia and thus creates a veritable vicious circle". An analogical explanation is given by HAGUENAUER (1960) in his thesis. Our own experience is in agreement with this; we have witnessed several operations for late posttraumatic facial paralysis during which a nerve was found which showed considerable swelling particularly in its vertical intra-mastoidal part; at one of these operations the surgeon (Prof. JONGKEES) remarked: "if I did not know any better I would diagnose this as Bell's palsy".
The same cause (ischaemia resulting from compression caused by "something") can play a role in palsies of delayed onset following *surgical intervention*. Any ear-surgeon actually recognizes this, consciously or unconsciously, for the first measure he takes is removal of the bandage or tampons with which he has filled the operation cavity. Pressure exerted by this bandage, or oedema caused by irritation resulting from the bandage, or stagnation of blood, exudate or pus under the bandage, are regarded as the commonest causes of the nerve disturbance. Experimental investigations by BENTLY & SCHLAPP (1943), WEISS (1943) and DENNY-BROWN & BRENNER (1944) have shown that it is not so much the mechanical pressure on the nerve as ischaemia of the nerve caused by this pressure that must be taken into account. Mechanical pressure as such is relatively well and long tolerated by the peripheral nerves.
It is a well established fact that *acute inflammation* causes oedema (and therefore circulatory changes) in the adjacent regions. This, we presume, is the cause of facial nerve paralysis which occurs as a complication of *acute otitis media* and — in rare cases — in *external otitis*.

These considerations and findings have led us to the conclusion that the

forms of facial paralysis which we have included in group III in view of lack of agreement as to the correct mode of treatment, show a similarity in pathogenesis, viz: ischaemia of the facial nerve to such an extent as to cause interference with the normal conduction of nerve impulses. Consequently this group will be discussed as an entity.
The majority of patients show spontaneous recovery within a few weeks. This implies that the nerve lesion was of a mild nature, and that no degeneration of nerve fibres occurred. Instead, there has been only temporary elimination of the conductive function of the nerve. This condition is known as reversible conduction block or neurapraxia (SEDDON, 1943). Some investigators (e.g. BOONE, 1959) reject the phenomenon of neurapraxia. It is difficult to understand, however, how else some of the Bell's palsy patients described by BOONE could have made a *complete* recovery 12-14 days after the onset of paralysis.
A certain number of patients, however, do not make a full recovery. If we are to spot those patients and to do anything for them, we must know the severity of the functional impairment in each individual case of facial paralysis at a time sufficiently early to permit us to take measures counteracting a possibly imminent unfavourable course.

The concrete questions to be answered as early as possible in any individual case of facial paralysis — especially complete paralysis — are:

First question: *Is this paralysis transient (reversible nerve block) or is it a degenerative disorder of conduction, so that recovery is dependent on regeneration of the nerve fibres affected?*

Second question: *If the paralysis is still in the reversible stage at the time of examination, are there any signs (if so, which?) heralding an unfavourable development, i.e. irreversibility?*

We will study the extent to which these questions can be answered on the basis of personal findings and of those reported by others. To do this we shall examine in turn the data resulting from clinical investigation and those that can be obtained with the help of special electrical tests. So as to ensure a sufficiency of data from a homogeneous group of patients, Bell's palsy will be considered in the following sections as a representative for group III as a whole.

CLINICAL INVESTIGATION

THE EXTENT OF THE PARALYSIS

According to FINDLAY (1952), JONGKEES (1955), TAVERNER (1959), DALTON (1960) and others, initial complete paralysis indicates a serious prognosis, whereas in partial paralysis complete recovery is usual. This is confirmed by the findings of CAWTHORNE & HAYNES (1956), who saw a full recovery in 47 of 111 patients (42%) with complete Bell's palsy, as against 57 of 67 patients (85%) with incomplete paralysis.
We intend to compare these figures with our personal observations, but in studying the case histories concerned for the relevant data, a few points came up which should be first mentioned. The patient's own opinion cannot be used in determining the extent of a paralysis. We have repeatedly seen that a facial paralysis which was partial at the first examination, became complete one or two days later, whereas the patient indicated that, in his opinion, it had meanwhile "improved a little"; the patients often seem to adjust themselves rapidly to the new situation, particularly as regards eating and drinking. On the other hand, we have often noticed muscular movements which the patient himself had not yet recognized. It is consequently only the investigator's objective opinion which is valid in determining the extent of the paralysis.
Another important point is the interpretation of the word *initial* (see above); none of the abovementioned investigators defined the use of this term. Only in exceptional cases are patients seen by the specialist within one or two days of the onset of paralysis. This is improving, however, with increasing cooperation between the specialist and the general practitioner, to whom most patients report within 24 hours; our personal experience confirms this improvement. For reasons to be subsequently explained, we have set 2 weeks as the limit within which a paralysis can be regarded as initial.

TABLE V. EXTENT OF PARALYSIS ON EXAMINATION AT WEEKLY INTERVALS AND ULTIMATE RECOVERY IN 35 CASES OF ACUTE FACIAL PARALYSIS (BELL'S PALSY).

T = total paralysis
P = partial paralysis

Nr	name	1st day exam.	1st examination	end 1st week	end 2nd week	end 3rd week	end 4th week	end 5th week	end 6th week	ultimate recovery
1	Bl	4	P	P	P	P	P	P	P	complete
2	JO	3	P	P	P	—	—	cured		complete
3	Ry	6	P	P	P	P	—	cured		complete
4	Sh	6	P	P	P	P	—	—	cured	complete*
5	VB	3	P	P	P	P	P	—	cured	complete
6	BS	4	P	P	P	P	cured			complete
7	Be	4	P	P	P	P	—	cured		complete
8	DW	6	P	P	P	P	—	P	P	complete
9	HK	5	P	P	P	P	—	P	P	complete
10	Hs	5	P	P	P	P	P	P	P	*incomplete*
11	Ra	5	P	P	P	P	—	—	cured	complete
12	SM	4	P	P	P	cured				complete
13	Lb	4	P	P	P	cured				complete
14	Bo	3	P	T	P	P	—	P	—	complete*
15	SJ	2	P	T	P	P	P	—	P	complete*
16	Sh	3	P	T	P	P	—	—	—	complete
17	Sh	3	P	T	P	P	P	—	P	*incomplete*
18	Ho	2	P	T	P	P	P	P	P	*incomplete*
19	Rs	2	P	T	T	P	P	—	P	*incomplete*
20	Go	2	P	T	T	T	T	T	T	*incomplete*
21	Bu	2	P	T	T	T	T	T	T	*incomplete*
22	Rn	2	P	T	T	T	—	—	T	*incomplete*
23	SB	5	T	T	P	P	—	P	—	complete*
24	Pe	5	T	T	P	P	P	—	P	complete
25	Kr	8	P	P	P	P	—	—	P	complete*
26	Sr	8	P	P	P	P	cured			complete
27	Bu	8	P	P	P	P	P	cured		complete
28	MB	8	P	P	P	cured				complete
29	VS	7	P	P	P	cured				complete
30	Hb	8	T	T	P	P	—	P	P	*incomplete*
31	Ml	7	T	T	P	P	P	—	P	complete
32	Br	8	T	T	P	P	—	cured		complete
33	KB	7	T	T	P	P	P	—	P	complete*
34	RK	7	T	T	T	T	—	—	T	*incomplete*
35	Ne	8	T	T	T	T	T	—	T	*incomplete*

* see explanation page 35

In 35 patients with Bell's palsy first seen within a week of the onset, the course of the paralysis was studied and the data obtained were tabulated (Table V). The extent of the paralysis — partial (P) or total (T) — was recorded at the first examination and 1, 2, 3, 4, 5 and 6 weeks after the onset. The condition 1 year after the onset of the paralysis was regarded as the ultimate result.

The designation total paralysis requires no comment; the qualification partial paralysis is less explicit, for it includes all degrees of paralysis from the most severe pareses (patients no. 4, 10, 14, 16, 17, 18, 20, 22) to the very mild forms with only slight loss of function (patients no. 2, 3, 29). A subdivision of patients with partial paralysis is impracticable in view of the limited size of the group as a whole.

For the same reason the interpretation of the ultimate result was kept simple and confined to only three qualifications, viz:

complete — complete cure in the opinion of the patient, those about him and the investigator.

*complete** — complete cure in the opinion of the patient and those with whom he is in contact, but with small imperfections at careful examination by the investigator (e.g. an edge of lashes remaining visible at maximally tight closure of the eye; minimal movement of the angle of the mouth associated with palpebral movement, etc.). These imperfections escape the patient and those about him and will certainly not be pointed out to them by the investigator.

This division into *complete* and *complete** was made exclusively for the sake of exactness; it is of no practical importance because both groups of patients must be regarded as clinically cured.

incomplete — unmistakable signs of incomplete cure or cure with residual symptoms (incomplete return of voluntary movement, synkinesis, contractures).

A study of Table V reveals the following with regard to prognosis:

— the course of a paralysis is still variable the first two weeks after its onset; a total paralysis can recede to become partial and a partial paralysis may become total. The latter, however, was exclusively seen within the first week; paralyses partial at the end of the first week were never seen to become total afterwards.

— at the end of the second week the severity and course of the paralysis are well-established. In our series there was only 1 patient (no. 19) in

whose paralysis a change occurred after this time (the existing total paralysis became a partial paralysis). It is for this reason that we set the limit within which a paralysis can be called "initial" at two weeks.

The paralysis was total in 6 and partial in 29 patients at the end of the second week. Comparison with the situation at the end of the first week showed that, at that time, 17 patients had a total and 18 a partial paralysis. The prognostic significance of these differences is clearly revealed when these data are tabulated together with the ultimate results (table VI).

TABLE VI. EXTENT OF PARALYSIS AT THE END OF THE 1ST AND 2ND WEEK AFTER THE ONSET AND ULTIMATE RECOVERY IN 35 CASES OF BELL'S PALSY

		Nr.	Ultimate recovery complete	incomplete
END 1ST WEEK:	partial paralysis	18	17 (95%)	1
	total paralysis	17	8 (45%)	9 (55%)
END 2ND WEEK:	partial paralysis	29	25 (85%)	4
	total paralysis	6	—	6

(The results are percentually given so as to facilitate comparison with the findings of other investigators. We are aware of the fact that the percentages calculated cannot be accurate because of the smallness of our series.)

The above table demonstrates that the chance of complete recovery for a patient who shows total paralysis at the end of the first week, is about 50% — a chance which falls to zero when the paralysis is still total at the end of the second week. For a patient with partial paralysis at the end of the second week, however, the chance of complete recovery amounts to 85%; if the paralysis was partial from the beginning, then the chance of full recovery is even as much as 95%.

On the basis of these findings we can confirm the previous statement that "in *partial* paralysis complete recovery is usual"; in our series the chance of a cure was 85% at the end of the second week — a figure corresponding with the data presented by CAWTHORNE & HAYNES.

In *complete* paralysis, the prognosis in our series was obviously dependent on the time at which this condition prevails. CAWTHORNE & HAYNES — who do not make this distinction — arrived at a chance of cure of 42%; in our series such a percentage was found only at the end of the first week; in no case did we see complete recovery from a paralysis which was still total at the end of the second week. The statement that „initial complete paralysis means a serious prognosis" was therefore also confirmed.

MUSCLE TONE (TONUS)

In VAN RIJNBERK's "Nederlands Leerboek der Physiologie" (1945), TEN CATE defines tonus as "the condition of involuntary tension characteristic of healthy skeletal muscle at rest, which constitutes the basis of all reflex and voluntary movements executed by the muscles". It is a state due to reflex activity and transmitted to the muscle through its motor nerve. If the motor nerve is severed, the tonus is abolished and the muscle is inert.
According to MARTIN and KETTEL, the presence or absence of muscle tone is the most important sign available in facial paralysis. TICKLE shares this opinion and attaches great importance particularly to the position of the angle of the mouth, stating that: "if the angle of the mouth is nearly even and shows very little sign of drooping 2 or 3 weeks following the onset of the paralysis, I think this is a good sign. It means that there are sufficient impulses getting through the nerve to supply tonus to the muscle". BIEMOND (1953) maintains that the prognosis of a paralysis is serious when striking hypotonia is obvious already in the acute phase; he adds, however, that this is not always the case.
In our patients with facial paralysis, the condition of the face *at rest* as found at each examination has been recorded, viz: lines of the forehead, position of the eyebrow, width of the palpebral fissure, depth of the nasolabial fold, deviation of the philtrum and position of the angle of the mouth. The situation is always compared with the unaffected half of the face, and the findings are recorded as follows:

R=L indicating an identical condition right and left
R>L indicating a wider palpebral fissure on the right
R<L indicating a less deep or smoothed out nasolabial fold on the right
R ↓ indicating a drooping angle of the mouth or eyebrow on the right
→R indicating deviation of the philtrum to the right

TABLE VII. MUSCLE TONE, EXTENT OF PARALYSIS AND ULTIMATE RECOVERY IN 35 CASES OF ACUTE FACIAL PARALYSIS (BELL'S PALSY).

P = partial paralysis + = no or only mild decrease in tone
T = total paralysis — = severe or total loss of tone

Nr	name	end 1st week		end 2nd week		ultimate recovery
1	Bl (4)	+	P	+	P	complete
2	JO (3)	+	P	+	P	complete
3	Ry (6)	—	P	+	P	complete
4	Sh (6)	+	P	+	P	complete*
5	VB (3)	+	P	+	P	complete
6	BS (4)	—	P	+	P	complete
7	Be (4)	+	P	+	P	complete
8	DW(6)	+	P	+	P	complete
9	HK (5)	+	P	+	P	complete
10	Hs (5)	—	P	—	P	*incomplete*
11	Ra (5)	+	P	+	P	complete
12	SM (4)	+	P	+	P	complete
13	Lb (4)	+	P	+	P	complete
14	Bo (3)	+	T	+	P	complete*
15	SJ (2)	+	T	+	P	complete*
16	Sh (3)	+	T	+	P	complete
17	Sh (3)	—	T	—	P	*incomplete*
18	Ho (2)	—	T	—	P	*incomplete*
19	Rs (2)	—	T	—	T	*incomplete*
20	Go (2)	—	T	—	T	*incomplete*
21	Bu (2)	—	T	—	T	*incomplete*
22	Rn (2)	—	T	—	T	*incomplete*
23	SB (5)	—	T	+	P	complete*
24	Pe (5)	—	T	—	P	complete
25	Kr (8)	+	P	+	P	complete*
26	Sr (8)	+	P	+	P	complete
27	Bu (8)	—	P	+	P	complete
28	MB (8)	+	P	+	P	complete
29	VS (7)	+	P	+	P	complete
30	Hb (8)	+	T	—	P	*incomplete*
31	Ml (7)	+	T	+	P	complete
32	Br (8)	—	T	+	P	complete
33	KB (7)	—	T	—	P	complete*
34	RK (7)	—	T	—	T	*incomplete*
35	Ne (8)	—	T	—	T	*incomplete*

* see explanation page 35

If necessary, these indications were supplemented by additions such as slight, marked, more or less than at previous examination, etc.
The tonus score in a patient with total right-sided paralysis reads as follows:

forehead : R smooth
eyebrow : R ↓
palpebral fissure : R>L
nasolabial fold : R<L
philtrum : →L
angle of the mouth: R ↓

Of course this is a relatively gross type of registration; we found it impossible, however, exactly to appraise the often very slight differences in tone, particularly when these varied from one examination to the next.

So as to establish whether a correlation exists between the tone of a paralysed facial half, the extent of paralysis and ultimate recovery, we have tabulated the data on the extent of paralysis of Table V with tone data at end of the 1st and 2nd week, obtaining Table VII.

The following can be stated with regard to our patients:
— those who were clinically cured (25), with the exception of two, showed no or only mild decrease in tone in the paralysed half as compared with the unaffected half of the face at the end of the second week. Compared with the condition at the end of the first week, the tone in these patients had either improved or remained unchanged; there was never a decrease.
— those who made an incomplete recovery (10), with one exception, had a flabby facial half at the end of the second week. Seven of these 10 patients showed this loss of tone already at the end of the first week.

The position of the angle of the mouth, to which TICKLE attaches such great importance, was also studied relative to the ultimate result. We confirmed that all patients showing no drooping of the angle of the mouth at the end of the second week, made a full recovery. The reverse was not true, for several patients showing unmistakable drooping at that time did, none the less, fully recover. In our opinion, however, the tone at one site cannot be expected to afford information on the face in its entirety. In the group of paralyses under discussion, there were several patients in

whom the various muscle groups of the face were affected to a different extent, or in whom several muscle groups recovered to a different degree after total paralysis.

Summarizing, we can say with regard to the not readily estimated tone that paralyses in which the tone is still normal or only slightly affected at the end of the second week, have a very favourable prognosis; poor tone at this time, however, particularly when this was also present at the end of the first week, indicates an unfavourable or at least a dubious prognosis in the majority of cases.

Aural pain

Many authors report that cases of Bell's palsy associated from the onset with severe pain (usually behind the ear) have a poor prognosis. This opinion is supported by Tumarkin (1936), Tickle (1948), Hilger (1949) and others. Taverner (1959) found that initial pain was significantly more common in the degeneration group than in that with conduction block. Waterman (1909), on the contrary, noted mastoid pain in 41% of patients who made a quick recovery and showed no changes in faradic excitability, and in only 30% of patients with a complete or partial reaction of degeneration. Sullivan (1952) and Kettel (1959) hold that initial severe pain behind the ear in complete paralysis warrants immediate decompression of the facial nerve as an emergency operation. Verjaal (1955), who generally rejects large-scale surgical neurolysis, agrees that the intervention is indicated "in acute cases of facial paralysis when violent pain deep in the ear suggests incarceration of the swollen nerve in the osseous canal".

We obtained data on the presence or absence of pain at the onset of Bell's palsy in 89 of our patients. The most common localization was behind the ear; in some patients the pain radiated to the occipital region, neck or shoulder. The pain was usually described as "twitching"; in some cases it was of the severe, tearing type. A few patients merely indicated a vaguely unpleasant sensation in the mastoid region. In some cases the pain preceded the onset of paralysis by 1-5 days; in others, it did not commence until 1-5 days after the onset of paralysis. In most cases the onset of pain coincided with the onset of paralysis.

Pain was present in 52 and absent in 37 of the 89 patients.
The relation between presence or absence of pain and ultimate recovery is shown in Table VIII.

Table VIII. Relation between recovery and initial pain in 89 patients with Bell's palsy

Complete recovery	32	pain	16 (severe in 4)
		no pain	16
Incomplete recovery	57	pain	36 (severe in 8)
		no pain	21

These data show that:
- the number of painless and painful cases was the same in the group who made a full recovery;
- in those who made an incomplete recovery, the number of painful cases exceeded that of the painless cases by some 25%;
- among the patients with aural pain, the proportion of severe pain was the same in the group of complete and that of incomplete recovery (1 : 4).

On the basis of these findings we cannot confirm the statement by Findlay and Kettel that the majority of cases of ischaemic palsy are painless, nor the suggestion that severe initial pain in this type of paralysis indicates a serious prognosis.

Aural pain in facial paralysis can also be a manifestation of aural *herpes zoster* (Ramsay-Hunt's syndrome). When the wellknown herpetiform efflorescences are lacking — or have been overlooked in the initial phase of the affection — confusion with Bell's palsy is easily possible. The typical localization of the herpes zoster pain, however, is in the external meatus or deep in the ear, whereas the pain in Bell's palsy is usually indicated as localized behind the ear or in the occipital or nuchal region. Apart from this, in typical cases of aural herpes zoster there is frequently

a functional disturbance in one of the other cerebral nerves, particularly the stato-acoustic nerve. In some cases, however, differential diagnosis from Bell's palsy may offer great difficulties or even be entirely impossible without inspection of the facial nerve.

Taste sensation

According to Tumarkin (1951) cases of Bell's palsy accompanied by marked pain and loss of taste have a much more unfavourable prognosis than when the onset of paralysis is asymptomatic. Park & Watkins (1949) stated, however, that loss of taste is of no reliable prognostic value in individual cases. Taverner (1959) reported that loss of taste as subjectively reported by the patient was not significantly different in the degeneration group as compared with the conduction block group.

In many of our patients the subjective taste sensation was noted to be at variance with the results of objective tests. Among 88 patients from whom we succeeded in obtaining reliable information, there were 40 who reported disturbances in the sense of taste in the initial days of facial paralysis. These disturbances consisted in loss of taste, alteration of taste (metallic, galvanic, sweetish) or difference in sense of taste between the right and the left half of the tongue. Some patients noted paraesthesia in the homolateral half of the tongue or a sensation as if that part of the tongue had been anaesthetized. The relation between presence or absence of subjective taste disturbances and ultimate recovery is shown in Table IX.

Table IX. Relation between subjective taste disturbance and recovery in 88 patients with Bell's palsy

Complete recovery	36	disturbance	17
		no disturbance	19
Incomplete recovery	52	disturbance	23
		no disturbance	29

In our cases, therefore, there was no correlation between the presence or absence of subjective taste disturbance in the initial days of facial paralysis and the degree of ultimate recovery.

An attempt was made to establish whether, and to what degree, a difference existed between the subjective taste sensation and the result of a taste test. The gustatory function was tested with the aid of solutions of sugar, sodium chloride and quinine, applied to the anterior part of the tongue by means of swabs. The side of the tongue homolateral to the facial paralysis was tested first. On 70 patients referred to us for Bell's palsy, data on subjective taste sensation and taste test at the time of the first examination are available for comparison. Of these patients, 51 reported no subjective taste disturbances; 18 did. The results of the taste test in these patients were as follows (Table X).

Table X. Comparison of subjective taste sensation and results of the taste test in 70 patients with Bell's palsy.

No subjective taste disturbance:	51	taste test unreliable	7
		taste test normal	21
		hemi-ageusia	18
		hemi-hypogeusia	5
Subjective taste disturbance :	19	hemi-ageusia	13
		hemi-hypogeusia	6

The patients with subjective taste disturbances, therefore, invariably also showed objective differences between left and right; in patients without subjective taste disturbances, objective changes existed in about half of the cases. These findings warrant the conclusion that a subjective disturbance in the sense of taste is a reliable finding, whereas the absence of subjective taste disturbances is of little or no significance.

The value attached by various investigators to taste examination and taste disturbances, varies greatly. Biemond (1961) points out that ageusia is an inconstant finding in facial paralysis, and attaches only moderate clinical significance to this phenomenon. Collier (1941) maintains that taste examination is only seldom of practical value. Lathrop (1952) regards the taste test as unreliable and affording only meagre information; Lathrop has abolished the test. Von Schulthess & Dubs (1957) studied the value of the taste test in connection with the localization of the nerve lesion in 17 patients with posttraumatic facial paralysis; they consider this method

of investigation unreliable because the test results did not correspond with the findings at operation. PARK & WATKINS (1949), VERJAAL (1954), KRISTENSEN (1957), KRARUP (1958, 1959), FELDMANN & MAIER (1959) and SULLIVAN & SMITH (1959), however, regard the taste test as important in the topical diagnosis of facial nerve lesions.

Our experience, based on a large number of operations in which the facial nerve was dissected free over its entire course in the mastoid, is that the point at which the chorda tympani curves away from the nerve shows considerable individual variations. In Bell's palsy, the site at which and the distance over which the nerve in the Fallopian canal is swollen, is likewise subject to considerable variation. Whether and to what extent the chorda tympani is involved in this process is therefore dependent both on the level of bifurcation and on the localization and spread of the facial nerve affection. In view of the variability of these factors, the presence or absence of taste disturbances cannot be expected to yield exact topical and certainly not prognostic indications in facial paralysis.

HYPERACUSIS (DYSACUSIS)

The term hyperacusis refers to abnormal sensitivity to sounds of the ear homolateral to the facial paralysis, which is ascribed to insufficiency of the stapedius muscle and resulting predominance of the tensor tympani muscle. Another theory is that the phenomenon must be regarded as a recruitment phenomenon resulting from an affection of the labyrinth; the labyrinthine disturbances and those of the facial nerve are believed to originate from a common cause (BRUNETTI & HAHN, 1951; BOTMAN, 1954). The latter theory deprives the phenomenon of hyperacusis of any value it might have in topical diagnosis.

MIEHLKE (1960) prefers the term dysacusis to hyperacusis because there is no improvement or perfection of auditory acuity but merely a disagreeable loudness of normal environmental sounds. The condition is also referred to as phonophobia or oxyakoia (BARRAUD, 1926). Some of our patients described the sounds they heard as hollow and reverberating.

BIEMOND (1961) writes that the symptom of hyperacusis occurs transiently only in acute paralysis, and even then as an inconstant phenomenon. VERJAAL (1954) also states that the phenomenon is often described as associated with facial paralysis but is infrequently observed in practice.

Only 6 out of 80 patients questioned by him in this connection mentioned this symptom.

We did not question all our patients as to hyperacusis. Relevant data are available on 62 patients, in 18 of whom the phenomenon occurred during the acute phase of facial paralysis, usually only for a few days. The relation between the presence or absence of hyperacusis and ultimate recovery is shown in Table XI.

TABLE XI. RELATION BETWEEN HYPERACUSIS AND RECOVERY IN 62 PATIENTS WITH BELL'S PALSY

COMPLETE RECOVERY	22	hyperacusis	2
		no hyperacusis	20
INCOMPLETE RECOVERY	40	hyperacusis	16
		no hyperacusis	24

In our series, therefore, hyperacusis in cases of incomplete recovery was about four times as frequent as that in cases of complete recovery. It must be sufficient to mention these findings; since they are data on a subjective sensation of inconstant occurrence and transient in nature, they warrant no conclusion as to the prognosis of the facial paralysis which is the basis or perhaps merely associated with it (recruitment phenomenon).

Objective demonstration of the action of the stapedius muscle is possible by examination of the acoustic *stapedius reflex* and determination of acoustic impedance. TSCHIASSNY (1953) and KRISTENSEN (1957) consider these methods very important in the localization of the nerve lesion in cases of facial paralysis. SULLIVAN & SMITH (1959), however, regard tests for stapedius muscle power as useless in determining the localization of the lesion, "since the tensor tympani muscle supplied by the trigeminal nerve evidently relieves the disability". MIEHLKE (1960) also regards the value of the dysacusis test as doubtful because of the uncertain origin of this phenomenon. We made no such examinations in our cases.

Lacrimation

Lacrimal secretion is as a rule studied by suspending a strip of filter paper bent to form a hook in the conjunctival sac of both eyes, comparing the extent to which and the speed with which lacrimal fluid is soaked up by these strips. Verjaal (1954) attaches great importance to this method of investigation, as do Maxwell & Magielski (1956). Collier (1944) and Lathrop (1952) consider the test unimportant and unreliable. Dalton (1960) finds it difficult to interpret this test.

Whatever the value of the lacrimation test may be, in any case it is exclusively of topical diagnostic interest. Theoretically, no decrease in lacrimation should be found in Bell's palsy because the affection is always localized in the vertical part of the facial nerve in the mastoid, i.e. past the bifurcation of the greater superficial petrosal nerve which governs the secretion of the lacrimal glands. Verjaal (1955) nevertheless found absence of lacrimation on the homolateral side in 8 of 82 patients with Bell's palsy. Dalton (1960) examined 97 patients and found unmistakable hyposecretion in 2 cases. Jacobs & Colony (1955) found no change in lacrimal secretion in 44 cases. Botman (1954) and Jongkees (1957) also failed to find changes in lacrimal secretion in the cases examined. Since we believed that no prognostic indications could be expected from the lacrimation test, our patients were not submitted to this examination.

Nasopalpebral reflex

Biemond, who attaches the greatest importance to electrical methods of examination in determining the prognosis of facial paralysis, also attaches some significance to the *nasopalpebral reflex* (percussion between the eyebrows, immediately above the root of the nose, causes simultaneous contraction of both orbicularis oculi muscles). In the case of facial paralysis, this reflex is diminished or lacking on one side; in the latter case the prognosis is believed to be unfavourable. In Ansink's thesis (1960) we find that examination of this reflex was introduced as a clinical aid in 1920, by Guillain, who believed that the prognosis of peripheral facial paralysis had so far been determined too exclusively on the basis of electrical findings. Ansink presents a detailed review of the various theories on the nature of these and other periocular reflexes. In the nasopalpebral reflex, we are in fact confronted with two different reflexes, viz: a pure muscle (skin) extension reflex and a response to a pain stimulus given by

percussion between the eyes. The mechanism is trigeminofacial, and a normal reflex course therefore requires an intact facial and trigeminal nerve (sensory portion).

The nasopalpebral reflex has a low threshold value. If the stimulus is very small but still just sufficient to cause a reaction, then this reaction is visible in the form of a horizontal nasad displacement of the inferior palpebra as a result of contraction of the inferior palpebral part of the orbicularis oculi muscle. According to Van Gilse (1936) this phenomenon is the last to disappear when facial paralysis increases, and the first to re-appear as recovery commences. It is believed to have some prognostic significance in that persistence of this phenomenon in the case of a paralysis which otherwise seems complete, opens perspectives of improvement, whereas disappearance of this phenomenon is believed to indicate an unfavourable course of the paralysis.

The nasopalpebral reflex was studied in the majority of our patients; the criterion used was the movement or immobility of the inferior palpebra. If no movement was observed, the reflex was called negative. The 35 patients seen shortly after the onset of paralysis (previously included in Table V) included 5 on whose nasopalpebral reflex no data are available. In one other patient (no. 1), moreover, no nasopalpebral reflex could be provoked either on the paralysed or on the intact side. Our data, therefore, refer to only 29 patients. In Table XII the outcome of the nasopalpebral reflex (+ or —) has been listed with an indication of the severity of paralysis at that time (Partial or Total); the table also mentions the ultimate recovery.

Consideration of the data on these patients shows that absence of the nasopalpebral reflex does not necessarily indicate that paralysis is complete. Several patients showed only partial paralysis at a given time, although the nasopalpebral reflex was absent. Another striking feature — which should be mentioned because it is not apperent from the table — was the fact that, in a number of patients (no. 5, 7, 9, 14, 15, 18), there was still a possibility of voluntary contraction in some part of the orbicularis oculi muscle (although greatly diminished) whereas the nasopalpebral reflex could no longer be provoked. Van Gilse's observation that the reflex movement of the inferior palpebra remains intact longest in the case of increasing facial paralysis, was not confirmed by our findings. Nor was the reverse (first reappearance of this phenomenon at beginning recovery)

TABLE XII. NASOPALPEBRAL REFLEX, EXTENT OF PARALYSIS AND ULTIMATE RECOVERY IN 29 CASES OF BELL'S PALSY.

P = partial paralysis
T = total paralysis
\+ = nasopalpebral reflex present
— = nasopalpebral reflex absent

Nr	name	end 1st week		end 2nd week		ultimate recovery
1	Bl (4)					
2	JO (3)					
3	Ry (6)	P	+	P	+	complete
4	Sh (6)					
5	VB (3)	P	—	P	+	complete
6	BS (4)	P	+	P	+	complete
7	Be (4)	P	+	P	+	complete
8	DW(6)	P	+	P	+	complete
9	HK (5)	P	—	P	+	complete
10	Hs (5)	P	+	P	+	*incomplete*
11	Ra (5)	P	+	P	+	complete
12	SM (4)	P	+	P	+	complete
13	Lb (4)	P	+	P	+	complete
14	Bo (3)	T	—	P	—	complete*
15	SJ (2)	T	—	P	+	complete*
16	Sh (3)					
17	Sh (3)	T	—	P	+	*incomplete*
18	Ho (2)	T	—	P	—	*incomplete*
19	Rs (2)	T	—	T	—	*incomplete*
20	Go (2)	T	—	T	—	*incomplete*
21	Bu (2)					
22	Rn (2)	T	—	T	—	*incomplete*
23	SB (5)	T	—	P	+	complete*
24	Pe (5)	T	—	P	+	complete
25	Kr (8)	P	—	P	+	complete*
26	Sr (8)	P	—	P	—	complete
27	Bu (8)	P	—	P	+	complete
28	MB (8)	P	+	P	+	complete
29	VS (7)	P	+	P	+	complete
30	Hb (8)	T	—	P	—	*incomplete*
31	Ml (7)	T	—	P	+	complete
32	Br (8)					
33	KB (7)	T	—	P	+	complete*
34	RK (7)	T	—	T	—	*incomplete*
35	Ne (8)	T	—	T	—	*incomplete*

* see explanation page 35

always true. In a number of patients in the period of beginning recovery from total paralysis, some muscles were capable of some degree of contraction again at a time when the nasopalpebral reflex could not yet be provoked. In a number of patients, moreover, it was seen that the possibility of movement was restored in part of the orbicularis oculi muscle (e.g. near the lateral corner of the eye) before this was seen in the inferior palpebral part of the muscle.

In the presence of total paralysis the nasopalpebral reflex was invariably absent. This was also ascertained in patients who reported later than a week after the onset of paralysis, and who were consequently not included in Table XII.

A nasopalpebral reflex positive at the end of the first week was never seen to revert to negative afterwards; the reverse, however, was frequently observed.

Of 20 patients who showed a positive nasopalpebral reflex at the end of the second week, 18 made a full recovery; in 10 of these patients at the end of the first week the reflex was negative. Of 9 patients who showed a negative reflex at the end of the second week, 2 made a full recovery.

Any relation between presence or absence of the nasopalpebral reflex and ultimate recovery is best shown in the following table.

TABLE XIII. RELATION BETWEEN NASOPALPEBRAL REFLEX (NPR) AND RECOVERY IN 29 PATIENTS WITH BELL'S PALSY

		Nr	Ultimate recovery complete	Ultimate recovery incomplete
END 1st WEEK:	NPR +	10	9	1
	NPR —	19	11	8
END 2nd WEEK:	NPR +	20	18	2
	NPR —	9	2	7

TABLE XIV. MUSCLE TONE, NASOPALPEBRAL REFLEX, EXTENT OF PARALYSIS AND ULTIMATE RECOVERY IN 35 CASES OF BELL'S PALSY.

P = partial paralysis
T = total paralysis

Tone: + = no or only mild decrease in tone
— = severe or total loss of tone

NPR: + = nasopalpebral reflex present
— = nasopalpebral reflex absent

Nr	name	end 1st week Tone	NPR	end 2nd week Tone	NPR	ultimate recovery
1	Bl (4)	+	P ?	+	P ?	complete
2	JO (3)	+	P ?	+	P ?	complete
3	Ry (6)	—	P +	+	P +	complete
4	Sh (6)	+	P ?	+	P ?	complete*
5	VB (3)	+	P —	+	P +	complete
6	BS (4)	—	P +	+	P +	complete
7	Be (4)	+	P +	+	P +	complete
8	DW(6)	+	P +	+	P +	complete
9	HK (5)	+	P —	+	P +	complete
10	Hs (5)	—	P +	—	P +	*incomplete*
11	Ra (5)	+	P +	+	P +	complete
12	SM (4)	+	P +	+	P +	complete
13	Lb (4)	+	P +	+	P +	complete
14	Bo (3)	+	T —	+	P —	complete*
15	SJ (2)	+	T —	+	P +	complete*
16	Sh (3)	+	T ?	+	P ?	complete
17	Sh (3)	—	T —	—	P +	*incomplete*
18	Ho (2)	—	T —	—	P —	*incomplete*
19	Rs (2)	—	T —	—	T —	*incomplete*
20	Go (2)	—	T —	—	T —	*incomplete*
21	Bu (2)	+	T ?	—	T ?	*incomplete*
22	Rn (2)	—	T —	—	T —	*incomplete*
23	SB (5)	—	T —	+	P +	complete*
24	Pe (5)	—	T —	—	P +	complete
25	Kr (8)	+	P —	+	P +	complete*
26	Sr (8)	+	P —	+	P —	complete
27	Bu (8)	—	P —	+	P +	complete
28	MB (8)	+	P +	+	P +	complete
29	VS (7)	+	P +	+	P +	complete
30	Hb (8)	+	T —	—	P —	*incomplete*
31	Ml (7)	+	T —	+	P +	complete
32	Br (8)	—	T ?	+	P ?	complete
33	KB (7)	—	T —	+	P +	complete*
34	RK (7)	—	T —	—	T —	*incomplete*
35	Ne (8)	—	T —	—	T —	*incomplete*

*. see explanation page 35

Although, with regard to a paralysis involving the face in its entirety, we would prefer to be very prudent in attaching prognostic significance to a symptom arising from only a constituent part of the face, the data obtained nevertheless seem to indicate:

— that presence of the nasopalpebral reflex is a favourable sign;
— that absence of the nasopalpebral reflex at the end of the first week indicates a dubious prognosis;
— that absence of the nasopalpebral reflex at the end of the second week constitutes an unfavourable sign, although not absolutely so.

SUMMARY

After a separate discussion of the extent of the paralysis, muscle tone, aural pain, taste sensation, hyperacusis, lacrimation and nasopalpebral reflex, the following can be stated as to the value of these findings in determining the prognosis of Bell's palsy:

— presence or absence of aural pain, hyperacusis, disturbed taste sensation and disturbed lacrimation are of little or no importance;
— the extent of the paralysis, the degree of muscle tone, and the presence or absence of the nasopalpebral reflex are valuable data;
— during the first 14 days, the paralytic features are still capable of considerable changes; partial paralyses can become total and vice versa; at the end of this period, the condition is as a rule well established.

The extent of the paralysis is the most reliable criterion. The value of the muscle tone and the nasopalpebral reflex as such is limited; these data are of importance, however, if they correspond with the extent of the paralysis, in which case they support prognostic conclusions based on the extent of the paralysis. Data on muscle tone, nasopalpebral reflex, extent of paralysis and ultimate recovery have been arranged again in Table XIV. Correspondence between extent of paralysis, muscle tone and nasopalpebral reflex at the end of the first week was seen in about 60%, and at the end of the second week in about 75% of cases. This is yet another argument in favour of the contention that the condition of paralysis is more firmly established at the end of the second week.

In acute cases of Bell's palsy, the following *general prognosis* can be derived from these data:
— of patients showing *partial* paralysis at the end of the second week, about 85% make a full recovery. This favourable prognosis is supported by normal or only slightly subnormal muscle tone and presence of the nasopalpebral reflex. In cases showing from the onset partial paralysis, good muscle tone and persistent nasopalpebral reflex, a full recovery can be predicted with a degree of probability approaching certainty;
— of patients showing *total* paralysis at the end of the first week, about 50% nevertheless make a full recovery; the muscle tone at that time has no obvious prognostic significance;
— for patients showing *total* paralysis at the end of the second week the chance of complete recovery is virtually zero.

Unfortunately, this general prognosis is of little value in the appraisal and treatment of *individual* patients. It is of no practical importance that a patient showing total paralysis after one week still has a chance of about 50% to make a full recovery; if we are to do anything for this patient, we must know to which 50% he belongs at a given time (or is likely to belong in the near future). The same holds true for patients with partial paralysis; of this group, too, a number do not make a full recovery. It is therefore of importance to know the severity of the functional impairment in each individual case, at a time sufficiently early to permit of measures counteracting a possibly imminent unfavourable course.

For this purpose, two definite questions were raised at the beginning of this study. We must now establish whether the discussed clinical data enable us to answer these questions:

First question: *Is this paralysis transient (reversible nerve block) or is it a degenerative disorder of conduction, so that recovery is dependent on regeneration of the nerve fibres affected?*

complete paralysis: the fact that patients showing complete paralysis at the end of the second week have virtually no chance of a full recovery, warrants the conclusion that complete paralysis at that time is based on degenerative disorder of conduction; at an earlier time, the question cannot be answered;

partial paralysis: the fact that the chance of a full recovery is virtually 100% if paralysis is partial from the onset, muscle tone good and nasopalpebral reflex persitent, warrants the conclusion that only a transient disorder of conduction exists in these cases; in the other cases the question cannot be answered.

Second question: *If the paralysis is still in the reversible stage at the time of examination, are there any signs (if so, which?) heralding an unfavourable development, i.e. irreversibility?*

Neither in complete nor in partial paralysis do clinical data afford any information in this respect.

The practical conclusion at the end of this chapter is that, on the basis of the *clinical* findings alone it is impossible to select individual patients with an unfavourable or dubious prognosis, at least not within the first two weeks after the onset of paralysis. At that time, unfortunately, we are too late to prevent an imperfect recovery.

ELECTRICAL TESTS

The main function of a peripheral motor nerve is the conduction of impulses from the CNS to the muscles with which the nerve corresponds. If the impulse conduction is disturbed at any level, the muscles experience a repercussion of this disturbance. An examination of the muscles, therefore, can give an impression of the disturbance in the function of the nerve.

Such an examination can be carried out in two different ways, viz: 1) as "spot diagnosis" examination, and 2) as "follow-up" investigation: comparison of the data obtained in a serial examination reveals whether a paralysis is stationary, improving or regressing. This is only true, however, if accurate *quantitative* methods are used, which are reproducible and comparable.

Several electrical tests are in use. According to the principle on which they are based, these tests are distinguished as

1) electromyography and
2) electro-diagnosis by stimulation.

ELECTROMYOGRAPHY

Electromyography can be defined as the recording of the electrical characteristics of muscles (RODRIQUEZ & OESTER, 1956); the records — obtained with the aid of electrodes and electrical registration apparatus of sufficient sensitivity — represent the changes in potential in the muscle fibres.

This description is incomplete because electromyography offers more. The electrical changes which occur in the muscle fibres afford information as to what goes on in the motor unit, and consequently it is the electrical characteristics of the *motor unit* that are studied by electromyography (DE GROOT, 1952).

A detailed *historical* review of the origin and development of electromyography can be found in "Clinical Electromyography" by MARINACCI (1955) and in "Electrodiagnosis and Electromyography" by LICHT (1956). Many investigators have contributed to the clinical development of electromyography and numerous important papers on the subject have been published, e.g. by ADRIAN & BRONK, ARIEFF, BAUWENS, BOWDEN, BUCHTHAL, DENNY-BROWN, DENNY-BROWN & PENNYBACKER, GASSER, GOLSETH, HOEFER & PUTNAM, HUDDLESTON, JASPER, KUGELBERG, LEFEBVRE, LINDSEY, LUNDERVOLD, MARX, PRITCHARD, PROEBSTER, RICHARDSON, RODRIQUEZ, THIEBAUT, WATKINS, WEDDELL, FEINSTEIN & PATTLE and WYNN PARRY. In the Netherlands, pioneer work was done by WERTHEIM-SALOMONSON (1920). In 1939 SIEMELINK wrote his thesis "Contribution to the knowledge of the electromyogram", and BOONE (1958), also as a thesis, wrote an electromyographic study entitled "The cure of peripheral facial paralysis". Other contributions were made by DIRKEN (1941), LORENTZ DE HAAS (1952) and SPOOR & VAN DISHOECK (1958). DEN HARTOG, MULLER & VAN DER TWEEL (1952) published a paper on electromyographic equipment.

All authors discussing the value of electromyography are unanimous in their opinion that this method of investigation is superior to all other methods of electro-diagnosis in assessing the functional status of the motor unit. Electromyography affords a means of obtaining precise information on all neuromuscular disorders associated with lower motor neuron lesions. It is capable of revealing minor degrees of muscle denervation even when clinical examination indicates normal motor function, and of demonstrating the presence of a few intact motor units in an otherwise paralysed muscle. In this way it makes it possible to differentiate paralyses, which are clinically indistinguishable, into reversible physiological interruptions of nerve conduction and more severe affections of the nerve. BAUWENS (1948) summarized these aspects as follows. "The numerical reduction in normal action potentials on maximal exertion, or their total absence (coupled with the presence or absence of denervation potentials), can be of great prognostic and diagnostic significance in peripheral nerve injuries; electromyography is of assistance in differentiating quantitatively between complete and partial denervation, and qualitatively between paralysis due to transient nerve block and that due to axon degeneration".

Electromyography is also unrivalled in indicating the first signs of *reinnervation* of paralysed muscles; this type of motor unit activity can be recorded about 8 weeks before muscle function can be clinically detected; this is earlier than can be achieved by any other electro-diagnostic test.

Electromyography provides us with a functional rather than with an anatomical (histological) report; in cases of complete degeneration, it is therefore impossible to distinguish between disruption of the nerve (neurotmesis) and severe damage to the nerve fibres not affecting the continuity of the nerve trunk itself (axonotmesis).

The number of publications on electromyographic examination in *peripheral facial paralysis* is still limited. Some authors — e.g. LATHROP (1952), MAXWELL & MAGIELSKI (1956), SULLIVAN & SMITH (1959), BOETTE (1960) and VON SCHULTHESS (1961) — recognize electromyography as the method of choice in early detection of signs of regeneration after paralysis but regard its value otherwise as strictly limited. MARTIN (1952) considers electromyography an aid of no greater reliability than other factors considered. According to COLLIER (1955), however, and also according to TAVERNER (1955) and WILLIAMS (1959), the prognosis of facial paralysis is indicated with accuracy by electromyography. KETTEL (1959) agrees with this but he regards electromyography as a useless guide in emergency, at least for the time being. Like TUMARKIN (1951), however, he has great hopes of further development of this method of investigation.

ELECTRO-DIAGNOSIS BY STIMULATION

These tests study the response of muscle to electrical stimuli. "Electrical stimuli are the most satisfactory because the phenomenon of excitation is itself of electrical nature" (STARLING). The stimuli are applied to the muscle either directly or indirectly (i.e. via the motor nerve or its terminal ramifications). The most proximal point from which the *facial* muscles can be indirectly stimulated is the site at which the facial nerve emerges from the stylomastoid foramen. In the case of facial paralysis of endotemporal origin, therefore, this point of stimulation always lies distal to the site of the nerve lesion. It would be ideal if it were possible also to apply a stimulus proximal to that site, so that the impulse conduction through the affected part could be studied. However, this is possible only when

the nerve is exposed in this region at operation. Under normal conditions, we must rely on indirect information on the nerve lesion, based on findings obtained from the extratemporal portion of the facial nerve and the facial muscles. Several electrical tests will be discussed in the next section.

Faradic-Galvanic test

The faradic-galvanic test procedure is based on a difference in excitability between the nerve (usually its terminal fibres in the muscle), and the muscle. Treshold values are determined for faradic (short-duration) and galvanic (long-duration) current, and the presence or absence as well as the character of muscular contraction is ascertained. Normal muscle is responsive both to faradic- and galvanic-current stimulation. Completely denervated muscle does not respond to a faradic current of tolerable intensity but only to direct stimulation by a galvanic current. This condition is described as "full reaction of degeneration" (RD). The reaction of a denervated muscle unlike the brisk contraction of normal muscle is sluggish. If the response to indirect stimulation or to direct faradic stimulation is not abolished (although obviously diminished), then the condition is described as "partial reaction of degeneration" (PRD). For detailed information on this method of electrical testing, we refer to textbooks of neurology (BIEMOND) and neurphysiology (LICHT), special reports (SEDDON, 1954) and publications by BOWDEN, BRAZIER, RICHARDSON & WYNN PARRY, BAUWENS, RITCHIE, WATKINS & BRAZIER.

We will attempt to determine whether, and to which extent, this "classic" method of electro-diagnosis is of value for the prognosis of peripheral facial paralysis.

In studying the numerous publications on facial paralysis it is obvious that opinions on the value of the faradic-galvanic test differ very widely. FOWLER (1939), PARK & WATKINS (1949), SULLIVAN (1952), ERSNER (1954) regard this test as most valuable. BIEMOND forms his prognostic conclusions chiefly on the basis of these electrical findings, but maintains that reliable prediction can only be made after 2-3 weeks. RODRIQUEZ & SKOLNIK (1954), ZACHARY & LOAF (1954), CAWTHORNE & HAYNES (1956) and WILLIAMS (1959) also believe that the faradic-galvanic test (if performed 2-3 weeks after the onset of paralysis) has a reliable prognostic significance. VERJAAL (1955) writes: "In the majority of cases, a prognostic

assessment on the basis of the faradic-galvanic test requires considerable experience, and even then the reliability of this prognostic aid is usually limited". McGOVERN & FITZ-HUGH (1956) maintain that serial testing by faradic-galvanic stimulation is of practical (if limited) importance. According to BREMOND & GARCIN (1959), the faradic-galvanic test can indicate a complete reaction of degeneration on the 14th day but this does not necessarily have a prognostic significance. SANDER (1934) reports on a case of facial paralysis associated with chronic otitis media in which — although a reaction of degeneration was seen for several days — recovery was complete within a few days after operation. TUMARKIN (1936) states that there is some evidence that the reaction of degeneration does not necessarily imply absolute death of the nerve. ANDREEVSKY & YOVANOVITCH (1959) also describe two patients with complete electrical reaction of degeneration, who nevertheless made a full recovery. BOETTE (1960) regards the value of faradic-galvanic stimulation as limited because it is a qualitative instead of a quantitative method of investigation; a full RD does not necessarily indicate irreversibility of the nerve lesion. VON SCHULTHESS (1961) states that the RD does not warrant any conclusions as to prognosis.

Many investigators do not rely on the results of combined faradic-galvanic testing or the character of the RD, but exclusively use faradic stimulation as a single test of VIIth nerve function. BUNNELL (1952) advises decompression of the facial nerve upon loss of faradic response but MARTIN (1951) regards loss of faradic response as an unreliable indicator. This opinion is shared by VIOLE (1944), TICKLE (1945), HALL (1951), LATHROP (1952), MAXWELL & MAGIELSKI (1956), FEINMESSER (1957), KETTEL (1959) and KIRSTEIN & SCHOPFER (1960). Several of these authors have reported on patients with facial paralysis who spontaneously recovered although they had lost their response to faradic stimulation. The majority agree that a positive response to faradic stimulation indicates a favourable prognosis, but maintain that nothing can be deduced with certainty from a negative reaction. Yet a positive faradic reaction as such does not necessarily indicate a favourable prognosis: VERJAAL, who made follow-ups on the paralysis in a large number of cases of Bell's palsy, states that the patients who failed to recover included several in whom faradic response remained intact for a long time. CAWTHORNE (1946) pointed out that a faradic response may persist in a severed nerve for as long as 14 days.

COLLIER (1959) also warns that persisting neurapraxia should not be mistaken for a more destructive lesion; she observed facial paralysis without evidence of degeneration for up to 11 weeks. It is not surprising that, in view of such divergent views on the prognostic value of faradic-galvanic testing, she reached the conclusion that "the time has come to abandon the faradic-galvanic reaction in the management of facial paralysis".

Apart from this assessment of faradic-galvanic stimulation as to its diagnostic and prognostic value in facial paralysis, we studied general evaluations of this test in publications by physiologists, neurologists and physiotherapists. STARLING writes: "In view of the fact that there is no fundamental difference between galvanic and faradic currents such a statement as "excitable to faradic, inexcitable to galvanic electricity", so often met with, is meaningless". GRODINS & OSBORNE (1942) also point out that, from a physiological point of view, there is no sharp line of demarcation between the terms "faradic" and "galvanic". "Galvanic current simply represents a faradic current of very low frequency". HUDDLESTON (1945), DUMOULIN & AUCREMANNE (1960) and BAUWENS (1961) comment on the classic method of examination of muscle and nerve by electrical stimulation in the statement that with this method it is possible only to obtain a rough estimate of the situation. The majority of investigators mention as a grave disadvantage of this test that the results of faradic and galvanic stimulation cannot be quantitatively expressed; the voltage of the faradic current is not measured and serial examinations are consequently not comparable (WATKINS & BRAZIER, 1945; BOWDEN, 1951; COLLIER, 1955; RITCHIE, 1954; RICHARDSON & WYNN PARRY, 1957; REBOUL c.s., 1960). Apart from this, the voltage of the faradic current is limited by the tolerance of the patient. COOKSEY (1941) advises that, when the patient does not tolerate a faradic current sufficient for adequate clinical examination, the test should be repeated under anaesthesia. POLLOCK (1944) and WATKINS (1947) maintain that the lack of reaction to percutaneous faradic stimulation of denervated muscle is exclusively to be ascribed to an insufficient intensity of stimulation; if the necessary strength of current were bearable to man, this current would produce contractions throughout the period of denervation. RITCHIE (1954) and RICHARDSON & WYNN PARRY (1957) also point out that it has been well established in practice that denervated muscle can respond to a faradic current.
In faradic stimulation it may also be difficult to obtain an accurate result

because of high *skin resistance*. SULLIVAN (1959) and COOKSEY (1941) report that, after the nerve had been exposed (in cases of facial paralysis), conductivity could sometimes be demonstrated upon stimulation although no reaction was seen when the nerve was percutaneously tested.
COOKSEY (1941), POLLOCK et al. (1944), WATKINS & BRAZIER (1945) and RICHARDSON & WYNN PARRY (1957) pointed out, furthermore, that the galvanic-faradic test is of no great value either, in demonstrating neural *regeneration* at an early stage. Return of voluntary motion often precedes return of faradic excitability (in 60% of cases according to RITCHIE).

This review of opinions on the significance of faradic-galvanic testing in the diagnosis and prognosis of peripheral nerve blocks in general, and of facial paralysis in particular, may be closed with the conclusion reached by BOWDEN, WYNN PARRY and RICHARDSON (1957): the faradic-galvanic test is often confusing; no objective data can be recorded for comparison; with the introduction of electronic apparatus, the faradic-galvanic test should be replaced by serial testing of electrical reactions with the aid of standard techniques in which the strength and duration of the stimulus can be measured (plotting of strength-duration curves; estimation of nerve conduction); in conjunction with the complete clinical picture, these quantitative methods are an aid in the diagnosis, prognosis and treatment of nerve injuries.
Bearing this conclusion in mind, and because it seemed hardly useful to enlarge the series of negative or positive points of view by yet another contribution, we have refrained from using the faradic-galvanic test in our series of patients.

Strength-Duration Curves

Detailed particulars on the Strength-Duration (Intensity-Duration or I/T) curves can be found in papers by POLLOCK (1944), WATKINS (1947), WYNN PARRY (1953, 1956), RITCHIE (1954) and RICHARDSON & WYNN PARRY (1957). Suffice it to present a summary of these publications.
Nerve and muscle are excitable tissues, which respond when electrically stimulated. The degree of response depends on the *strength* (intensity) and *duration* of the exciting stimulus. The electronic apparatus used, delivers a rectangular wave (square pulse) of current or potential, which permits of accurate measurement of both the intensity and the duration of the

stimulus. The amount of current or potential required to produce minimal muscle contraction is determined. The curve which relates duration (in milliseconds) with amount of current or potential (in milliampères or volts), is known as the strength-duration curve (hereafter referred to as I/T curve). The graphs are usually plotted with ordinates expressed directly in mA or V. and abscissae as logarithmic pulse durations.
If a square pulse is delivered either directly to a normally innervated muscle or to its motor nerve, the results (and the I/T curve) are substantially the same. According to WATKINS (1947) it can be demonstrated, with extremely sensitive recording instruments, that the curve in fact consists of at least two distinct excitabilities, one of which has slower characteristics than the other. The "rapid" curve has been demonstrated to result from indirect stimulation of muscle by excitation of the intramuscular nerve fibres, whereas the "slow" curve results from direct excitation of the muscle fibres themselves.
In this way the I/T curve obtained from *normally innervated* muscle is a smooth curve, which corresponds with the excitability characteristics of the nerve fibres (rapid curve). The I/T curve obtained from *denervated*

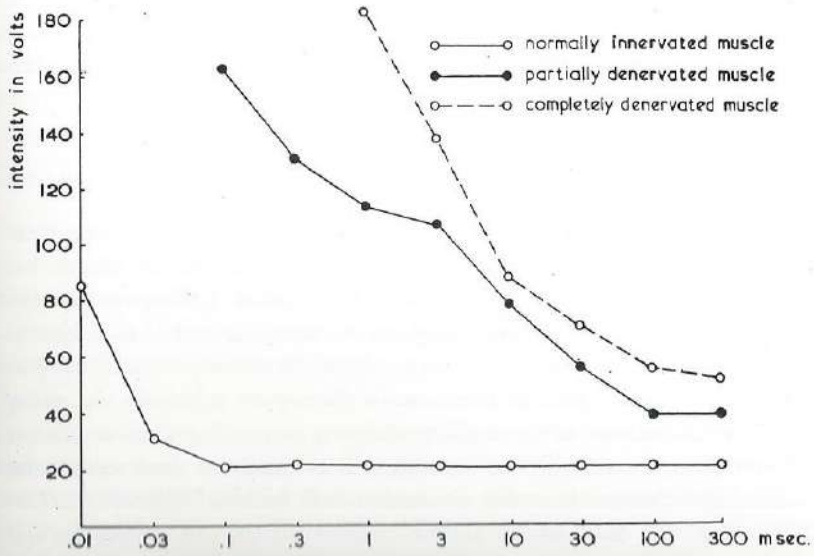

Fig. 2. Intensity-Duration curves (Constant voltage stimulator) (freely after: Richardson & Wynn Parry (1957). Ann. phys. Med., *4*, 3.)

muscle, however, is a steeply inclined curve, which corresponds with the excitability characteristics of muscle fibres (slow curve). In *partially denervated* muscle the I/T curve shows certain characteristic features, the most important of which is the appearance of a *kink* or discontinuity (see Fig. 2). This observation was first made by ADRIAN in 1917 and has been confirmed by subsequent investigations.

WYNN PARRY (1956), DALTON (1960) and BAUWENS (1961) point out that I/T curves are not infallible; they represent the excitability characteristics of the most excitable and most superficial fibres in the muscle. WYNN PARRY remarks that, in small or thin muscles such as the *facial* muscles, this objection does not hold true because the I/T curve then does afford information on the excitability characteristics of the whole muscle. RITCHIE (1954) reports that the distinction between normal and denervated muscle is so straightforward that it could be made with certainty in about 99% of 5100 curves examined by him. He adds, however, that at least 8-10 different duration points should be used for each curve in order to reveal minor discontinuities as very early signs of impeding denervation or commencing re-innervation; the recording and plotting of these curves, therefore, requires much time.

All investigators point out that the first signs of *re-innervation* in I/T curves precede the first clinical signs of recovery by some weeks. In a series of 54 patients with Bell's palsy, WYNN PARRY (1956) found this interval to average 21 days.

Nerve Conduction Test

In the previous section it was pointed out that in normally innervated muscle, the I/T curve is the same regardless of whether the muscle has been stimulated directly or indirectly. A normal I/T curve implies normal nerve conduction. Since the point of clinical interest is to determine whether the conductivity of the nerve itself is normal, reduced or lost, the most important part of the curve is that which represents the excitability characteristics of the rapid mechanism (nerve fibre), i.e. at stimuli of very short duration (0.3 to 0.1 msec.). It is chiefly in these stimulation values that changes in nerve conduction will become appreciable. This constitutes the basis of the nerve conduction test — a simple and sensitive test (RICHARDSON & WYNN PARRY) — in which the threshold intensity of stimulation of a peripheral nerve is measured.

Technique: the main nerve trunk which supplies the muscles under examination, is stimulated with a pulse of short duration at gradually increasing intensity, and the threshold intensity of stimulation for minimal muscle contraction is noted. The same procedure is repeated on the unaffected contralateral side, and the figures obtained are compared.

According to RICHARDSON & WYNN PARRY, loss of nerve conduction resulting from nerve lesions between the site of stimulation and the muscle can be due to any dysfunction (viz: neurapraxia); loss of nerve conduction caused by lesions proximal to the site of stimulation, however, implies lower motor neuron degeneration. In the presence of nerve conduction it is advisable to repeat the test at short intervals, as pointed out by GILLIATT & TAYLOR (1959). In patients with Bell's palsy, these authors contend that re-examination for up to 2 weeks may be necessary before it is concluded with certainty that degeneration of the distal part of the nerve will not occur.

There are still other electro-diagnostic tests by means of stimulation, e.g. measurement of the accommodation (adaption) of muscle and nerve to progressive currents (ZIEDSES DES PLANTES) and estimation of chronaxia. In our department chronaximetry was for some time systematically carried out in all cases of peripheral facial paralysis. This method of investigation, therefore, should at least be briefly described.

Chronaximetry

The chronaxia of a muscle is defined as the time during which a stimulus of twice the intensity of the rheobase*) must be applied to produce the minimal perceptible contraction of this muscle.

LAPIQUE found that chronaxia is an organ-specific datum. According to WERTHEIM SALOMONSON chronaxia is a true constant, which can be regarded as the criterion par excellence of excitability. HARRIS (1956), however, states that this view is not generally accepted by Anglo-American investigators.

BOURGUIGNON (1923) found a chronaxia of 0.2 to 0.6 msec. (condenser discharges) for the *facial* muscles; HARRIS found that the value of

*) The rheobase of a tissue is the minimal intensity of current of long duration (over 100 msec.) required to excite that tissue.

the frontal muscle in 10 normal subjects averaged 0.07 msec. with a voltage-stabilized stimulator and 0.18 msec. with a current-stabilized stimulator. According to our determinations (current-stabilized stimulator), the average chronaxia value of the orbicularis oculi and the orbicularis oris muscles in normal subjects is less than 0.3 msec.

Chronaxia increases after denervation, and in completely denervated muscle it is between 50 and 200 times that in normally innervated muscle. Chronaxia decreases again after reinnervation, but not as a rule until recovery is well advanced.

A few years ago, chronaximetry was carried out in our clinic in order to collect quantitative data on the condition of the facial nerve, to be used as criteria in deciding for or against surgical intervention in cases of peripheral facial paralysis. At that time these patients were usually referred to us not earlier than 4-6 weeks (sometimes even 2-3 months) after the onset of a paralysis, with a request for surgical treatment because conservative measures had failed. In virtually all these patients the paralysed facial muscles showed greatly increased chronaxia. The majority of these patients were submitted to an operation on clinical grounds. When no correlation could be established between the amount to which chronaxia was increased and the postoperative recovery, chronaximetry was abandoned and other methods of investigation were looked for.

It may well be that chronaximetry will prove to be valuable when serial determinations can be made from the first days after the onset of paralysis; we have no experience in this respect.

We regard it as a serious disadvantage of chronaximetry as a method of investigation in facial paralysis that electrical impulses of long duration (rheobase) are used in it. Our experience has shown that these current impulses in the face are experienced as particularly disagreeable by the majority of patients; in several adult patients and in most children, this test is impracticable for this reason.

For our study we had to make a choice from the abovementioned electro-diagnostic tests. Since it is generally agreed that electromyography is superior to all other methods of electro-diagnosis in obtaining precise information on neuromuscular disorders associated with lower motor neuron lesions, and also because experience with electromyography in peripheral facial paralysis is still limited, it was this investigation that we

selected as a basis for our study. We felt the need for a less elaborate test in addition, which would also have to be suitable for serial investigation and which would supply a more general picture of the paralysis. The nerve conduction test was chosen for this purpose because this test makes it possible both to study the function of all facial muscles together and that of every muscle separately, by electrical stimulation of the facial nerve at a single site.

Both methods of investigation will be exhaustively discussed in the following chapters, with reference to the data obtained by means of these tests.

ELECTROMYOGRAPHIC EXAMINATION

Instrumentation

The detection and recording of muscle or nerve potentials requires a special apparatus which, at the very least, should include electrodes, an amplifier with calibration device, and the recording apparatus, which consists of an oscilloscope with camera and loudspeaker. (Fig. 3)

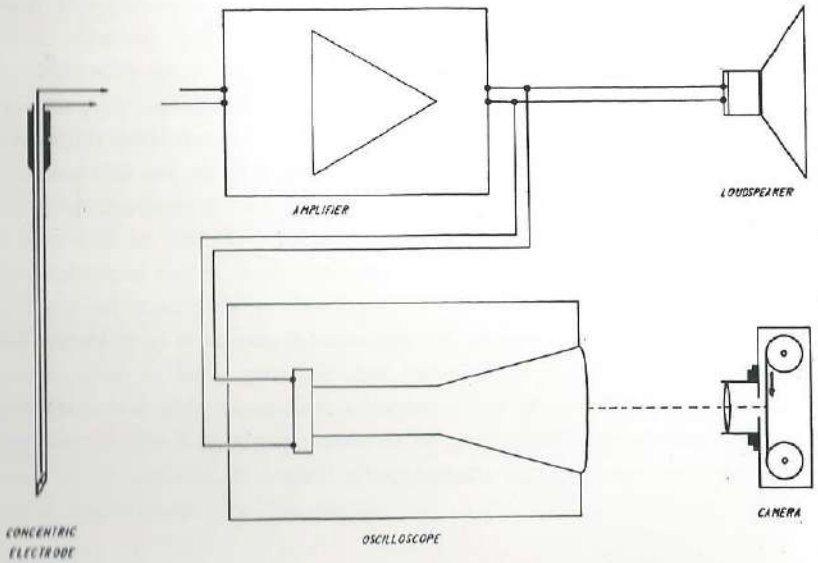

Fig. 3. Schematic drawing of Electromyographic recording apparatus.

Comprehensive data on these instruments can be found in publications by Siemelink (1939), Buchthal & Clemmesen (1940), Seyffarth (1941), Kugelberg (1947), Marx (1948), Bowden (1954), Buchthal & Rosenfalck (1955), Marinacci (1955), Licht (1955), Richardson & Wynn Parry (1957). Since an evaluation of an investigation such as ours,

and the conclusions based on it, are partly dependent on the arrangement and characteristics of the apparatus used, we consider it necessary to present some details on the various instruments.

Electrodes

In principle, distinction is made between two types of electrodes, viz: platelet (skin) electrodes and needle electrodes.

Electromyography via *platelet electrodes* is undoubtedly more agreeable to the patient because this mode of examination is not painful. Unlike the insertion of needle electrodes, moreover, it does not directly irritate or wound the muscle fibres. With platelet electrodes, however, only a general impression of muscle function can be obtained; recording of electrical phenomena of short duration, such as the spikes of the separate motor units or of fibrillations, is not feasible; traces of residual activity cannot be detected, nor can the preliminary phenomena of beginning regeneration be recorded. According to Hodes c.s. (1948), these are more readily established by a careful search for muscle movements than by electromyography with platelet electrodes. Fisher (1956) states that the information obtained with platelet electrodes can also be gathered, as a rule, by simpler methods such as inspection, palpation and the conventional electrical examinations. Another disadvantage of platelet electrodes is that they are likely to record, at the same time, the activity in adjacent muscle groups.

Whereas no detailed report can be expected from examination with platelet electrodes, this can certainly be obtained by examination with the aid of *needle electrodes*. For normal clinical electromyography, as a rule monopolar or concentric needle electrodes are employed.

The *monopolar needle electrode*, used by such authors as Gilson & Mills (1941), Jasper & Ballem (1949) and Forbes (1955), is a fine metal needle (often a common sewing-needle), insulated over its entire surface except the extreme point. The needle is inserted through the skin until it touches or enters the muscle. Beside the exploring electrode, a small platelet electrode is applied to the skin which serves as reference electrode. It can also be used as earth electrode, although for this purpose a third, generally much larger, electrode at some distance is often used.

The *concentric needle electrode*, designed by Adrian & Bronk who described it in 1929, consists of a hollow needle (e.g. a common injection needle) in which — centred and insulated from the jacket — a fine metal

wire extends as far as the point of the needle. With the wire in situ, the needle is obliquely ground down so that wire point and needle point come to lie in the same plane. To prevent formation of polarization currents, needle and wire are of the same type of metal. The central wire is the active electrode, the jacket is reference electrode and often also earth electrode.
Another type of electrode is sometimes used for special purposes. This is the so-called bipolar needle electrode in which the hollow needle (now exclusively serving as earth electrode) contains two insulated wire electrodes. The difference in potential between the two wire points is measured. With this electrode, potentials can be derived only from a very limited area. It is therefore unsuitable for normal clinical electromyography. Its use is restricted to the research laboratory.
Several investigators have maintained that there is only a very slight difference between the monopolar and the concentric needle electrode as to duration and amplitude of action potentials recorded; these values are slightly higher when the monopolar electrode is used. When the concentric electrode is used, the potential spikes recorded are sharper, and examination with a single needle is definitely less difficult, particularly in examining an area in which skin electrodes are not readily fixed, such as the face.

In our investigation we used single-core stainless steel concentric needle electrodes designed and constructed in our own laboratory. The core diameter was 0.15 mm., and that of the jacket was 0.45 mm. The point of the needle was ground down to an angle of about 30°. Insertion of the needle electrode was generally not too painful to the patient because the needle points were kept very sharp. Before and after use, the needles were immersed for 5 minutes in a solution of 1% iodine in 50% alcohol, after which iodine remnants were washed off in fixer (a sodium thiosulphate solution).

Amplifier

The minute changes in potential which arise in the muscle fibres and are received by the electrodes can be made perceptible by means of an amplifier. The amplifier must be of very high standard if it is to transmit, undistorted, the wide variations in frequency, amplitude and duration of the various fluctuations in potential. In our opinion the amplifier is the most important component of the electromyograph.

The amplifier used in our investigations was designed by our physicist, Mr E. De Boer Ph.D., and constructed at the physical laboratory of the department. It consists of a pre-amplifier, a filter unit, a power amplifier and a calibration oscillator. The pre-amplifier has two push-pull stages of amplification with Philips valves type E80F. Negative feedback has been used to stabilize the gain at the value of 1000. The pre-amplifier has been constructed on a shock-absorbing sub-chassis to protect it from microphony. In the filter unit, the frequency band of the signals can be cut both on the low and the high frequency side. The lower cut-off frequency can be set at 75, 150 or 300 c/s to ensure the desired suppression of slow fluctuations of the base line. The high frequencies can be cut off beyond 3000, 10.000 or 30.000 c/s. On most occasions, however, the full pass band of 75 to 30.000 c/s was used. The output voltage of the filter is fed to the oscilloscope. A power amplifier (2 W.) drives the monitor loudspeaker.
The circuit diagram of the complete amplifier is shown in fig. 4 (page 70). An enumeration of the chief characteristics of the amplifier system is given on page 71.

Recording apparatus

Visualization of the received and amplified fluctuations in potential has long been a serious problem. Although Lippman's capillary electrometer and the Einthoven string galvanometer were sensitive instruments with only slight inertia, they were incapable of recording vibration frequencies exceeding 2000 c/s. The same holds true for other electrodynamic or electromagnetic apparatus such as the ink writers; their inertia is so great that variations exceeding 100-125 c/s cannot be recorded with accuracy. Today, the only apparatus capable of recording the fluctuations in potential from 20-10.000 c/s occurring in electromyographic examination is the cathode-ray oscillograph introduced in physiology by Gasser & Erlanger (1922).

Oscilloscope

We used a Philips oscilloscope type GM 5656. Frequency range: 1-200.000 c/s. Sensitivity setting used: 100-300 mV./cm. (equivalent to 100-300 μV./cm. input to preamplifier). Time base frequency setting for visual inspection: 1 c/s.

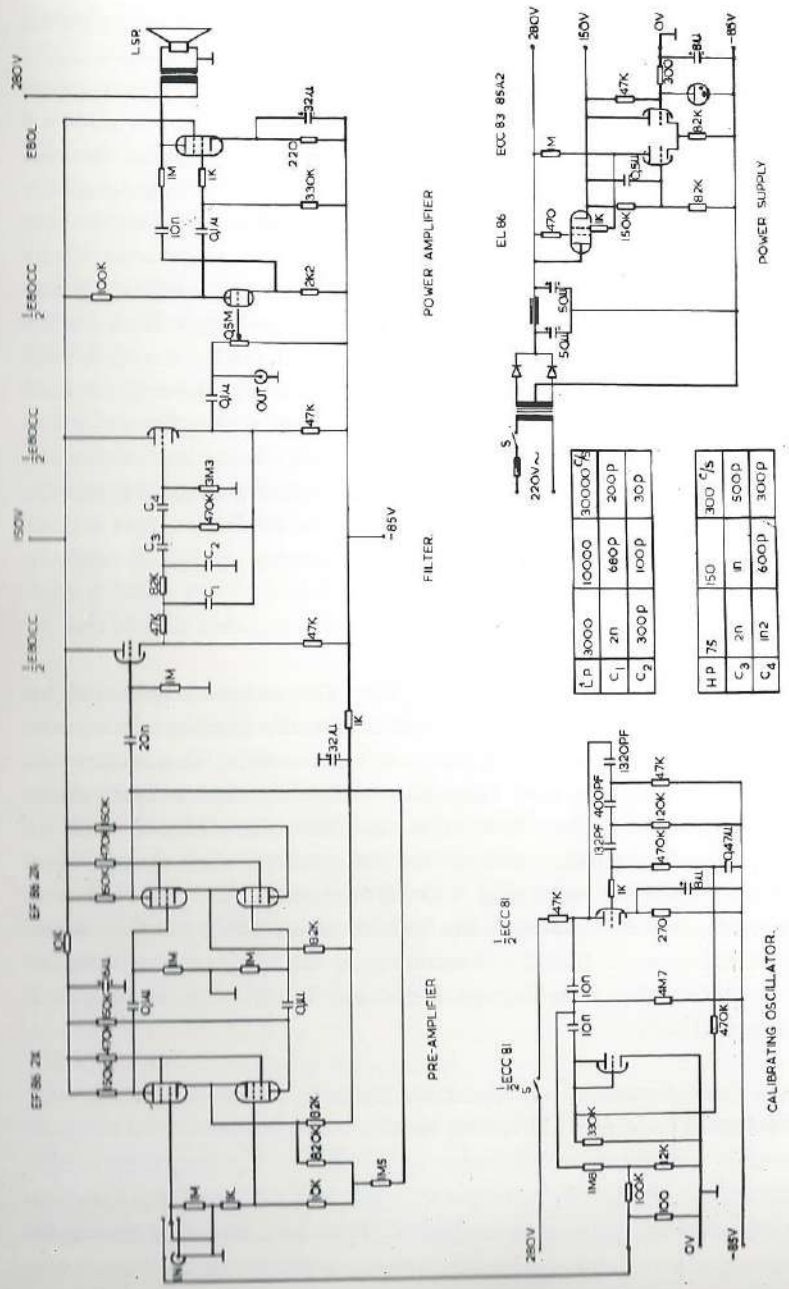

Fig. 4. Circuit diagram of the amplifier system.

Chief characteristics of the amplifier system

Preamplifier

Amplification	: 1000 x
Input	: not differential
Equivalent noise level at the input	: 1, 45 μV. over 75-30.000 c/s
Coupling time constant	: 0.1 sec.
Input resistance	: 1 MΩ
Input capacitance (excluding cable)	: 15 pF
Cable capacitance (excluding electrode)	: 230 pF
Electrode capacitance	: 120 pF

Filter unit

Low frequency cut-off	: 75, 150 or 300 c/s
Slope below cut-off	: 12 db/octave
High frequency cut-off	: 3000, 10.000, 30.000 c/s
Slope beyond cut-off	: —12 db/octave

Power amplifier

Maximum power	: 2W.
Maximum sensitivity	: 50 mW. at 250μW. input to preamplifier

Power supply

Supply voltages to preamplifier and filter unit (electronically stabilized)	: —85 V., + 150 V.

Camera

The camera used was a COSSOR film camera (model 1431) with lens 1 : 2.5 / f = 75 mm. Film speed variable between 0.05 and 25 inches per second. Normal setting 5 inches per second (i.e. 80 msec./cm.). The film used was Ferrania Pancro C7 35 mm.

Calibration

The calibration oscillator built into the amplifier provides a sinusoidal signal with a frequency of 1000 c/s and an amplitude of 1 mV. (peak to peak); see fig. 5. Normal adjustment of amplification: 50 μV. at the input gives a deflection of 1 mm. on the film. For observation of fibrillation the sensitivity is increased so that 15 μV. gives 1 mm. deflection.

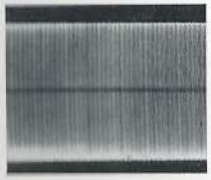

Fig. 5. Calibration signal.

Loudspeaker

The muscle potentials derived can be made audible by connecting a loudspeaker with the amplifier. The pitch of the sound perceived is dependent on the duration of the action potentials on which it is based (MARX, 1948). The intensity of the sound varies with the distance between the electrode point and the active motor unit or fibrillating muscle fibres; the shorter the distance the louder the sound.

The loudspeaker in this manner is a source of information as to the nature of the potentials derived; at the same time, however, it is a guide of the insertion of electrode needles, which makes it possible to "search about" with the needle point until it can be inserted into the centre of potential discharges. Personally, we regard the loudspeaker as indispensable in electromyographic examination, particularly because of the last mentioned possibility, and also because of the recognizable, characteristic sound of the fibrillation potentials. We found that a well-fitting *earphone* can be very useful in addition to the loudspeaker. Particularly when the examination room is not soundproof or when the whirring of the film camera interferes, it is yet possible with the aid of an earphone to follow exceedingly fine potential sounds of low intensity.

Some investigators such as KENDELL c.s., RICHARDSON, MARX & ISCH,

use a *tape recorder* in addition. This apparatus makes it possible at any given time to reproduce not only the picture of the potential fluctuations, but also their sound; this greatly facilitates the study and comparison of serial recordings.

General

If the muscle potentials are to be accurately reproduced, any disturbing influence of electrical or electromagnetic waves (light circuits, X-ray apparatus, etc.) in the vicinity of the examination room must be neutralized. In some cases this may necessitate the use of a FARADAY cage (PRITCHARD, 1929; HOEFER & PUTNAM, 1939; MARX, 1948). Generally, however, it is sufficient to ensure complete insulation and earthing of the apparatus, the electrode flexes and all electrical connections and cables. No further measures were required in our investigation; occasional disturbances were nearly always caused by loosened connections.

THE NORMAL ELECTROMYOGRAM

The insertion of a needle electrode in normal muscle tissue is associated with a burst of action potentials, varying in amplitude and duration from fibrillation potentials to motor unit potentials. They are called *pricking potentials* (BUCHTHAL and CLEMMESEN, 1941) or *insertion potentials* (WEDDELL, FEINSTEIN and PATTLE, 1944). They are the direct reaction to the mechanical irritation of the muscle fibres and in normal muscle tissue they are of very short duration; as soon as the needle stops moving they disappear.

In a normal muscle *at rest* no activity will be recorded by electromyographic examination, upon this all investigators are agreed. Complete electrical silence reigns, the oscilloscope shows an unbroken iso-electric base line (fig. 6) and the loudspeaker is silent. It is not always simple to obtain complete muscle relaxation; especially in the facial muscles this appears to be a difficult task for many patients, a fact which was also pointed out by RODRIQUEZ (1954), BOWDEN (1954) and MARSHALL (1959). However, when the patient receives a clear explanation of what is going to be done and the examination is carried out in the dorsal recumbent position so that all the other muscles of the body can relax, with a little patience one practically always succeeds in relaxing the muscles of the face. As an additional aid one can show the patient how the sounds from the loudspeaker become

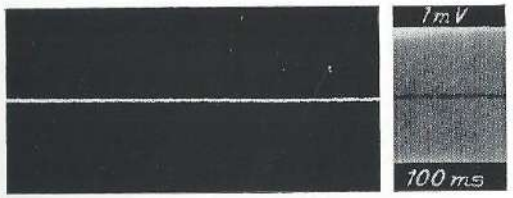

Fig. 6. Iso-electric base line.

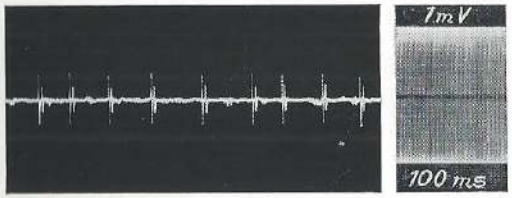

Fig. 7. Pattern of single motor unit potentials.

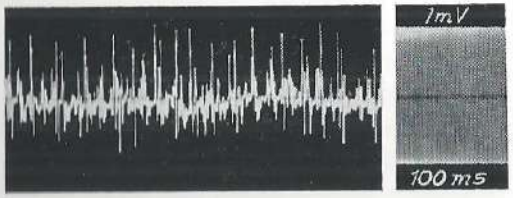

Fig. 8. Mixed pattern.

Fig. 9. Interference pattern.

stronger when he tenses his muscles and weaker when he relaxes them. As a rule he quickly learns what is wanted of him and with guidance from the loudspeaker he reaches complete relaxation. Only with a few nervous and anxious individuals we were unable to reach the desired relaxation in spite of all the aids.

When a very weak *muscle contraction* is evoked by only one motor unit, a train of single motor unit potentials appears on the oscilloscope screen at a frequency of 5-10 per second. Strengthening of the contraction is accomplished by increasing the discharge *frequency* of the active motor unit (temporal summation, MARX 1948) and by introduction (*recruiting*) of new motor units (spatial summation).

The strength of a voluntary muscle contraction will be determined chiefly by the total number of active motor units in the muscle (BUCHTHAL; MARX; BIGLAND & LIPPOLD); changes in the discharge frequency of the motor units result in only small variations in the muscle contraction (LINDSLEY, 1935). Depending upon the strength of the muscle contraction, different action potential patterns can be recorded electromyographically. In accordance with BUCHTHAL & CLEMMESEN (1941) one differentiates three mean patterns:

pattern of single motor unit potentials: a tracing of the discharging of one or only a few motor units in which the separate potential peaks are readily identified so that their discharge frequency can easily be counted. This is the recording of a weak contraction. (see fig. 7.)

mixed pattern: a tracing of the discharging of various motor units so that it is impossible to distinguish separately the appropriate potential peaks. Moreover, now the phenomenon comes into play that through the simultaneous discharging and summation of action potentials from two or more units potential peaks will be formed of more than normal amplitude. This is the recording of a moderately strong contraction (see fig. 8.).

interference pattern (= summation pattern): the number of active motor units of which the action potentials are picked up simultaneously by the electrode is now so great that a complex tracing of summated and interfering action potentials is recorded, in which the original base line has completely disappeared. This is the recording of a maximal muscle contraction (see fig. 9.).

Although the action potentials are not a direct estimate of muscle strength (BUCHTHAL, 1957), indirectly there is in the normal as well as in the paretic muscles such a narrow relation between number and discharge frequency of the active motor units and the muscle tension produced that the EMG-pattern obtained during maximum contraction effort can be used as a gauge for the innervation conditions (BIGLAND & LIPPOLD).

The action potential of a motor unit such as we record with the electromyograph is the resultant of the action potentials of the individual muscle fibres of that motor unit. To an impulse from the motor cell these fibres respond with an almost perfectly synchronous contraction. In the normal muscle about 90% of the potentials have a biphasic or triphasic appearance: there is an initial positive phase followed by a larger negative one which is mostly peak shaped. With the triphasic form follows a terminal positive phase. The steep negative deflection (*spike* potential) arises chiefly from the muscle fibres situated in the immediate neighbourhood of the needle point.
Yet the *form* of the action potential is not of essential importance; many investigators have determined that its shape depends on the derivation technique and the position of the electrode point in relation to the muscle fibres (BISHOP & GILSON, 1929); PRITCHARD, 1930; DENNY-BROWN & PENNYBACKER, 1938; MARX, 1948; JASPER & BALLEM, 1949; HIRSCHBERG & ABRAMSON, 1950; JARCHO c.s., 1952; LUNDERVOLD, 1953; RODRIQUEZ & OESTER, 1956; MARSHALL, 1959). Notably by BUCHTHAL and ROSENFALCK (1955) the importance of the type of electrode used is pointed out (see Table XV).

TABLE XV. DISTRIBUTION OF SHAPE OF ACTION POTENTIALS
(after BUCHTHAL & ROSENFALCK (1955). Acta psychiat. scand., *30*, 125.)

Muscles		total number of action potentials	number of action potentials in %				
			mono-	di-	tri-	tetra-	polyphasic
Frontal m. Orbic.oris sup.m Triangular m.	CONCENTRIC NEEDLE	289	2.5	46.5	41.0	4.0	5.5
	BIPOLAR NEEDLE	289	4.0	52.0	34.0	9.0	1.0

It appears that the sharp pointed negative phase is the most constant part of the potential figure; in fact it is considered representative for the potential discharge.

The *amplitude* (= voltage output) of the motor unit potentials, by which is understood the peak to peak distance between the highest positive and negative deflections, is chiefly dependant upon the distance from the active muscle fibres to the electrode point. With increasing distance the amplitude becomes smaller and also the spike form is less pointed (MARX & ISCH, 1951; BUCHTHAL, 1955). With ideal position of the electrode, thus in contact with the motor unit, the measured amplitude is a good gauge for the number of active muscle fibres. The difference in amplitude of action potentials recorded in various muscles is consequently explained to originate from an unequal number of muscle fibres per motor unit (SEYFFARTH, 1941; WEDDELL, FEINSTEIN & PATTLE, 1944; BOWDEN 1954). The amplitude of a motor unit action potential is constant and does not change with the increase or decrease of the muscle tension; the only factor that varies herewith is the discharge frequency.
The mean amplitude appears to be larger in the arm and limb muscles than in the tongue-, larynx-, eye- or facial musculature. For the muscles of the extremities the amplitude is usually between 1 - 2mV (JASPER & BALLEM, 1949). For the *facial* musculature FEINSTEIN (1946) gives values of 50 - 500 microvolts. BUCHTHAL & ROSENFALCK (1955) mention that by use of concentric needle electrodes the mean amplitude value of the facial musculature is 33% lower than that of the biceps brachial muscle.

The *duration* of a motor unit potential, by which is understood the total time lapse between the points where the potential wave turns off the base line of the oscilloscope and rejoins it again, is, like the amplitude and the form, dependent upon the type of electrode and the distance between the electrode point and the active muscle fibres. This duration according to most investigators amounts in skeletal muscles to 5 - 10 msec. (WEDDELL, c.s., 1944; JASPER, 1946; KUGELBERG, 1947; GOLSETH & FIZZEL, 1947; RODRIQUEZ & OESTER, 1956). BUCHTHAL & CLEMMESEN (1941) performed very accurate measurements with concentric needle electrodes and found for the human biceps brachial muscle and other skeletal muscles a mean

action potential duration of 7 - 14 msec. They noticed the time of the quick negative spike to be 4.64 ± 0.24 msec.

The duration of the motor unit action potential — according to THIEBAUT (1956) its most constant and best clarified parameter — is chiefly dependent on the number of active muscle fibres and is thus a guide for the size of the motor unit (PETERSEN & KUGELBERG, 1949; BUCHTHAL, 1955; RODRIQUEZ & OESTER, 1956). This duration varies considerably from muscle to muscle and is shortest in the eye-, larynx- and facial musculature. For the duration of the action potentials of the *facial* muscles FEINSTEIN (1946) reports 2 - 5 msec. PETERSEN & KUGELBERG (1949) found a mean value of 2.28 ± 0.03 msec. For adults between the ages of twenty to thirty years BUCHTHAL & ROSENFALCK (1955) computed a mean value of 4.4-6.2 msec. (this is 40 to 45% shorter than in the biceps brachial muscle: 7.7-10.7 msec.). The shorter duration of the action potentials of the facial musculature is explained by the number of muscle fibres per motor unit being so much smaller than in skeletal and trunk muscles.

The duration of the action potential of a motor unit is considerably longer than that of a single muscle fibril (0.5-2msec.). This is due to the fact that the contractions of the individual muscle fibres composing the motor unit are not perfectly synchronous (KUGELBERG, 1949; HIRSCHBERG & ABRAMSON, 1950; RODRIQUEZ & OESTER, 1956). BUCHTHAL & ROSENFALCK (1955) interpret this as "temporal dispersion in the summation of the action potentials from the fibres of the motor unit". This might be due to difference in time in which the nerve impulse reaches the motor endplates (caused by minimal differences in length of the preterminal branches of the nerve fibres or differences in propagation velocity of the impulse in these branches), differences in latent period at the myoneural junctions, and differences in distance between the individual muscle fibres and the electrode point.

Increased duration of the motor unit action potential (and in particular of the spike potential) is seen among others with progressing age, low intramuscular temperature and in neurogenic lesions (PETERSEN & KUGELBERG, 1949; PINELLI & BUCHTHAL, 1953; BUCHTHAL & ROSENFALCK, 1955; THIEBAUT, ISCH & ISCH-TREUSSARD, 1956). According to RODRIQUEZ & OESTER (1956) this is mainly attributed to a decrease of the conduction

velocity of the nerve impulse. In some cases this imperfect synchronisation is easily seen in a notching of the spike wave (see fig. 10).

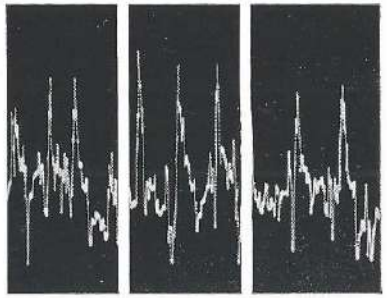

Fig. 10. Notching of the spike profile.

The discharge *frequency* of an individual motor unit varies in normal muscle between 3-5/sec. during minimal and 50-70/sec. during maximal contraction effort (LINDSLEY, 1935; BUCHTHAL & CLEMMESEN, 1941; DENNY-BROWN, 1949; BIGLAND & LIPPOLD, 1954; THIEBAUT, ISCH & ISCH-TREUSSARD, 1956). Higher frequencies were measured by a number of investigators, however, only during maximal tension of partially paralyzed muscles, cf. SEYFFARTH (1941): 90/sec.; or in the experimental animal cf. ADRIAN & BRONK (1928: 112/sec.; HOEFER & PUTNAM (1939): 200-450/sec.

The discharges of the various motor units in a muscle are asynchronous and independant of one another (SMITH, 1934; HOEFER & PUTNAM, 1939; MARX & ISCH, 1951). Only with maximal muscle tension, when the discharge frequencies of all the individual motor units have reached the extreme value of circa 50-70/sec., will large oscillations of a sinusoidal character develop by interference and summation, likewise in a frequency of about 50/sec. This discharge frequency is called "*the rhythm of Piper*". (In his original paper of 1912 PIPER stated that the rhythm of the contraction of muscle fibres during voluntary contraction as a rule amounts to about 50/sec., a frequency to be determined by the rhythm of impulses from the Central Nervous System). ADRIAN (1928) considered that Piper-rhythm can be explained from a synchronous activity of the motor units. This conception of the existence of a synchronisation tendency, as well as

the denomination "rhythm of Piper" has from the very beginning created much criticism and discussion (PRITCHARD, 1930; HOEFER & PUTNAM, 1939; SIEMELINK, 1939; BUCHTHAL & MADSEN, 1950; MARX & ISCH, 1951). SIEMELINK dedicated an eleborate experimental investigation to this problem. In his conclusion he denies the existence of a synchronisation tendency: "the waves of more than normal size are brought about merely and only as a result of accidental interference".

THE ELECTROMYOGRAM IN PERIPHERAL NERVE LESIONS

Peripheral nerve injury may cause dysfunction of nerve conduction and neuromuscular transmission, which may lead to paralysis of the motor units whose axons constitute the affected motor nerve. If all motor units are affected complete paralysis is present; if part of the motor units is preserved the condition is called partial paralysis or paresis. Paralysis, however, does not necessarily imply degeneration of the nerve axons, for reversible physiological block of conduction without degeneration is possible.

In *partial paralysis* when the patient tries to contract his muscle, electromyography reveals some motor unit action potentials, but because of the impossibility of recruiting sufficient motor units on maximal contraction effort no interference pattern can be produced. The amplitude of the action potentials lies within normal limits. The mean duration of the potentials may be somewhat longer than usual. The profile of the spikes may be ragged and the number of polyphasic action potentials is often increased. As mentioned already formerly it appears likely that these phenomena are due to asynchronous contractions of the muscle fibres within the motor unit, caused by variations in propagation velocity of the nerve impulse in the preterminal axon fibres or by variations in transmission at the myoneural junctions. (GOLSETH & HUDDLESTON, 1949; BOWDEN, 1954; SHEA & WOODS, 1955). According to some authors the discharge frequency of the preserved motor units on maximal contraction effort is higher than in normal muscle (THIEBAUT, ISCH & ISCH-TREUSSARD, 1956; EATON & LAMBERT, 1957). LEFEBVRE & GREMY attach prime importance to this

phenomenon for the diagnosis of paralysis of peripheral nerves. According to them it gives the impression that the remaining motor units through an increased discharge frequency (temporal summation) endeavour to compensate for the inability to recruit other motor units (spatial summation).

Severe involvement of a nerve may stop nerve conduction and lead to *complete paralysis* and denervation of the muscles supplied by that nerve. This condition will be followed by complete neuraxial cylinder degeneration distal to the point of injury. Voluntary contraction effort will not reveal any action potentials and complete electrical silence will be present during the first period after onset of the paralysis.
After some time, however, the denervated muscle fibres start producing some electrical activity again. Spontaneous action potentials of short duration and low voltage occur, repeating rhythmically at low frequency. These potentials are called *"fibrillation potentials"* or *"fibrillations"*. Since fibrillation of denervation will play an important part in the following chapters we consider it necessary to give a detailled description of this phenomenon.

Fibrillation

The *history* of fibrillation is covered in detail in the publications of MARINACCI (1955) and LICHT (1956). SCHIFF (1851) is considered to be the first to observe that denervated voluntary muscle is in a constant state of surface agitation. He noted that from 3-5 days after he had sectioned the hypoglossal nerve in dogs and rabbits spontaneous twitches could be seen in the denervated tongues; this condition he described as fibrillation. He noticed that the fibrillation ceased as the muscle atrophied and also when nerve regeneration took place. SCHIFF associated fibrillation with minute muscular contractions due to degeneration of the hypoglossal nerve. Further study convinced him that this activity was present in all muscles deprived of their innervation.
The credit of the first clinical observation of fibrillation is given to PROEBSTER (1928) who described spontaneous action potentials which he recorded in the affected muscles of a boy who had suffered a brachial plexus injury and in a patient with long standing poliomyelitis. PROEBSTER noticed that this fibrillation activity could not be recorded from the unaffected healthy muscles. In 1928 an important contribution to electro-diagnosis was made by ADRIAN and BRONK. With the introduction of the coaxial needle

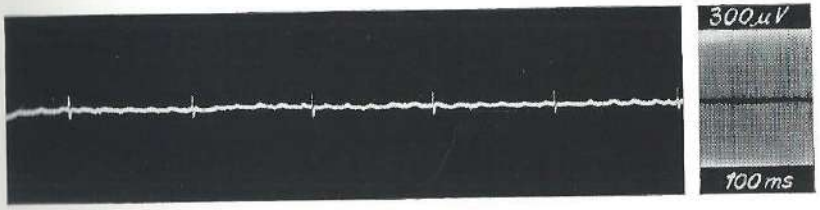

Fig. 11. Rhythmically repeated single fibrillation potentials.

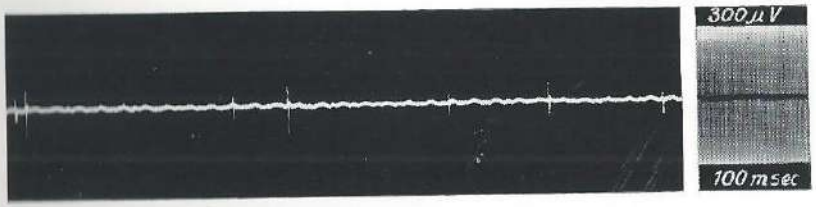

Fig. 12. Two fibrillation potentials rhythmically repeated at different frequencies.

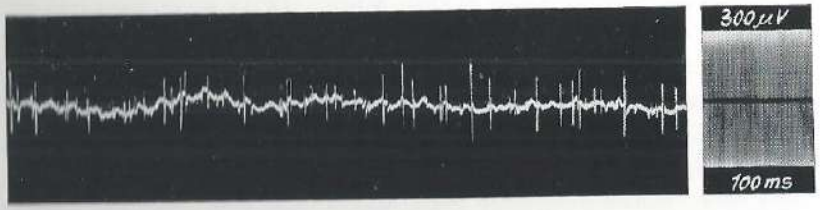

Fig. 13. Profuse fibrillation.

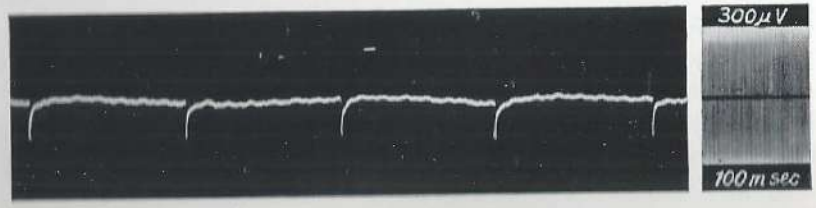

Fig. 14. Rhythmically repeated single positive sharp waves.

electrode they made it possible to pick up action currents developed by very small portions of muscle and even by single muscle fibres. With the help of these improved recording methods further observations of neuromuscular action potentials and fibrillation of denervation were made and reported by LINDSEY (1935), BROWN (1937), DENNY-BRAWN & PENNYBACKER (1938), HOEFER & PUTNAM (1939), ECCLES (1941), WEDDELL, FEINSTEIN & PATTLE (1944), GOLSETH & FIZZELL (1947) and by many other investigators.

Fibrillations cannot be detected through the intact skin, but only in the bared muscle. In man they are visible only in the tongue in cases of hypoglossal nerve paralysis. They are best seen when the exposed surface is illuminated obliquely (LANGLEY & KATO, 1915). The surface of the muscle shows a quivering of inconstant rhythm without any obvious centre of activity. The twitching of the various muscle fibres is not coördinated. With concentric needle electrodes fibrillations are recorded electromyographically as very fine mono- or diphasic (mostly diphasic, rarely triphasic) spikes. They are repeated with a constant and perfectly regular rhythm at a frequency varying from 2-30/sec. Usually, however, the discharge rate is less than 10/sec. (cf. fig. 11-12). The amplitude (voltage output) of the fibrillation potentials is low. It may range from 10-300 μV, but most investigators report values between 50 and 150 μV. In experimental studies BERGAMINI (1955) obtained a mean amplitude of 135.5 μV. The duration of fibrillation potentials is short, usually from 1-2 msec. BUCHTHAL reports values from 0.5-3msec. JASPER & BALLEM (1949) found the negative spikes to be 0.5-1.5 msec. in duration.
Because of their extemely short duration fibrillations are easily recognised in the loudspeaker by the characteristic sharp high pitched clicking sound they produce. MARINACCI (1955) compares the sound produced by large numbers of fibrillations with a "frying sound" or the "sound of fine rain falling on a tin roof".

From experimental study it is known that fibrillation action potentials are due to contractions of individual muscle fibres. Much debated is the question from which point along the muscle fibre the excitation wave of fibrillation originates. JARCHO c.s. (1954) studied this problem in the gracilis muscle of the rat; they concluded that fibrillation potentials originate within or in the immediate neigbourhood of the motor end-

plates and are propagated thence along the length of the muscle fibres.

Fibrillation potentials are present only when the nervous connection between a muscle fibre and its supplying motor nerve has been lost, in other words: in denervated muscles in the presence of lower motor neuron degeneration. Numerous authors have confirmed this statement (ARIEFF; BOWDEN; BUCHTHAL; DENNY-BROWN & PENNYBACKER; DUMOULIN & AUCREMANNE; GOLSETH & FIZZELL; HIRSCHBERG & ABRAMSON; HUMBERT, DEHOUVE & LAGET; JASPER & BALLEM; LEFEBVRE & GREMY; LORENTZ DE HAAS; MARSHALL; MCGOVERN & FITZ-HUGH; RICHARDSON & WYNN PARRY; RODRIQUEZ & OESTER; SHEA & WOODS; SKOLNIK; SOLANDT & MAGLADERY; THIEBAUT, ISCH & ISCH-TREUSSARD; WEDDELL, FEINSTEIN & PATTLE; WIESENDANGER). These authors unanimously concur and consider fibrillation as a phenomenon characteristic of denervated muscle. MARINACCI writes in his monograph (1955) "fibrillation is a crying of denervated muscle because of its loss of innervation", a somewhat theatrical but nevertheless a clear description. No less evident is the statement of BERGEL, discussing the report of GOLSETH & HUDDLESTON (1949). He says: "in the potentials associated with denervation fibrillation we learned to recognise one of the most reliable pathognomonic sigs of clinical medicine".

Fibrillation potentials are of two types: (1) those aroused by mechanical irration (insertion of a needle electrode, movement of a needle-point owing to squeezing or passive stretching of the muscle), (2) spontaneous potentials which are repeated rhythmically when the needle is stationary in the muscle.

In comparison to the very short burst of insertion potentials evoked by insertion of a needle in normal muscle, the initial manipulation of the electrode in denervated muscle induces a prolonged activity. The discharge frequency, at first increasing up to about 30/sec., decreases after a while and gradually settles down to a regular rhythm at low frequency (2-10/sec.). Minor movements of the point of the needle may be sufficient to elicit a new shower of fibrillations.

Some authors have reported that the onset of sustained spontaneous fibrillation is preceded by the appearance of insertion type fibrillations (KUGELBERG & PETERSEN; WEDDELL, FEINSTEIN & PATTLE; BOWDEN; MARX & ISCH).

Both provoked and spontaneous fibrillations are thought to be due to a greatly increased irritability of denervated muscle fibres, or as LANDAU (1951) stated: "low threshold rather than fibrillation per se is the significant factor in denervated muscle".

Since the experimental work of FRANK, NOTHMANN & HIRSCH-KAUFFMAN (1922) and DENNY-BROWN & PENNYBACKER (1938) it is believed that spontaneous fibrillation is due to periodic twitch contractions of denervated muscle fibres, sensitized by neural atrophy to the small amounts of acetylcholine in the normal circulation. SCHÆFFER & LICHT (1926) noted that the administration of small doses of acetylcholine enhances fibrillary contractions in denervated muscle. The same effect is produced by physostigmine (eserine) as reported by LANGLEY & KATO in 1914-15. The action of this drug upon muscle fibre contraction is not a direct one, but it inhibits the natural breaking down of acetylcholine by the enzyme choline-esterase. Thus it acts as "anti-cholinesterase" and potentiates the action of acetylcholine. Analogous effects are seen from the administration of prostigmine (neostigmine), first reported by LEVINE, GOODFRIEND & SOSKIN (1942) working on rats and confirmed later on by WEDDELL, FEINSTEIN & PATTLE (1944) in rabbits and man. Prostigmine is a synthetic compound closely allied to physostigmine, but with relatively less intestinal and cardiac effects, and thus safer for clinical use (MICKS).

Positive sharp waves

In addition to fibrillations a second feature of denervation may occur: monophasic *positive* discharges, which are known as "positive sharp waves", also referred to as "V-waves" or "saw tooth waves". They have a most characteristic constant shape: a rapid initial positive deflection (less than 1 msec. duration) followed by a slower declining negative phase (as long as 15-50 msec. duration) returning to the base-line (cf. fig. 14). Their frequency may range from 2-50/sec.; their voltage is very variable and they produce the sound of a dull thud in the loudspeaker. With the usual recording techniques positive sharp waves have never been observed in normal muscle, but only in denervated muscle accompanied by fibrillations. However, they do not occur in all cases and are seen much less frequently than fibrillation potentials. From experimental study JASPER & BALLEM (1949) assume that positive sharp waves represent non-propagated local potentials, set up in hyperirritable denervated muscle by mechanical

stimulation (electrode insertion; needlepoint injury), an explanation supported by MARINACCI ("mechanically induced denervation activity"). Though the exact nature of these discharges is still unknown they are considered to be a sign of denervation, of diagnostic value similar to fibrillation of denervation (JASPER & BALLEM; KUGELBERG & PETERSEN; MARINACCI; RODRIQUEZ & SKOLNIK; BUCHTHAL; RICHARDSON & WYNN PARRY; ROSSELLE c.s.; MARSHALL; DUMOULIN & AUCREMANNE; THIEBAUT, ISCH & ISCH-TREUSSARD; WIESENDANGER).

Time of onset of fibrillation

In the *experimental animal* the earliest moment at which fibrillation potentials can be detected after denervation varies widely with the animal used. In rats JONES, LAMBERT & SAYRE (1955) observed fibrillation potentials in only one area 49 hours after section of the sciatic nerve. Thereafter fibrillations increased progressively in number and after 75 hours the multitude of fibrillation potentials made identification of the individual spikes impossible. LANGLEY & KATO (1915) stated that in cats fibrillation of denervation does not commence until the 5th day after nerve section. Their observations have been confirmed by DENNY-BROWN & PENNYBACKER (1938): for the first 4 days following nerve section no fibrillation was observed; its first appearance was in the form of scattered single and very small momentary indentations of the surface of the muscle, each accompanied by a very small single diphasic action current. LANDAU (1951) reports that in rabbits sustained fibrillations can be demonstrated by the 4th or the 5th day after nerve section. FEINSTEIN, PATTLE and WEDDELL (1945) found that in thyroïdectomized rabbits, in which the metabolic rate was lowered about 30%, fibrillation was delayed in onset and did not appear for about 14 days; the administration of dessicated thyroid advanced the time of onset of fibrillation. From these findings they concluded that in their opinion fibrillation was dependant upon metabolic factors. They suggested that the variation in time of onset of fibrillation is due to varying metabolic factors relative to the size of the animal (rat 4 hours; guinea pig 2 days; rabbit 4-5 days; cat 5 days; monkey 8 days; man 18 days).

In man nearly all investigators agree that the time of onset of fibrillation potentials is from 2 tot 3 weeks after nerve injury (BAUWENS, 1948; JASPER

& BALLEM, 1949; RODRIQUEZ & SKOLNIK, 1954; BUCHTHAL, 1955; LEFEBVRE & GREMY, 1955; RODRIQUEZ & OESTER, 1956; MCGOVERN & FITZ-HUGH, 1956; SHEA & WOODS, 1956; EATON & LAMBERT, 1957; RICHARDSON & WYNN PARRY, 1957). BOWDEN (1954) found in a series of 10 patients that the mean time of onset of fibrillation was 19 days ± 2.0 for the large muscles of the arm, and 26 days $\pm$ 2.5 for the smaller muscles of the hand. Some authors mention onset of fibrillation at an earlier moment: ARIEFF (1948) 8 days after nerve injury; HUMBERT, DEHOUVE & LAGET (1958) 8-15 days after nerve injury. TAVERNER (1955), studying 55 cases of *facial paralysis* (Bell's palsy) with poor recovery, reports that in patients seen sufficiently early profuse fibrillation was usually detected by the 7th to the 10th day. In some patients scanty fibrillation potentials were detected as early as the 4th day. WEDDELL, FEINSTEIN & PATTLE (1944) studying 75 cases of peripheral facial paralysis, report that in their series onset of fibrillation never exceeded 14 days after the onset of the paralysis.

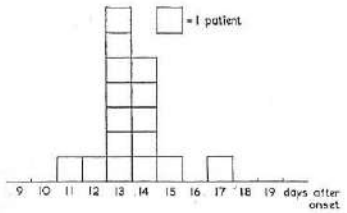

Fig. 15. Onset of fibrillation in 16 patients with Bell's palsy.

Personal observations: in 16 patients with Bell's palsy in whom fibrillations were found at a given time, we had occasion to carry out serial electromyographic examinations from the onset of paralysis. The graph (Fig. 15) shows that the time at which in these patients the first fibrillations were detected was an average of 13 days after the onset of paralysis.

If denervation is minimal or when electromyography is performed soon after the onset of paralysis it is often very difficult to find fibrillation potentials. Extensive exploration of the muscle by thorough probing with the needle electrode into a number of different directions is necessary. Fibrillations will be even harder to find if the muscle to be explored is cold or has been immobilised for a long time, if the blood supply to the muscle is insufficient, if the patient has hypothyroidism (FEINSTEIN, PATTLE & WEDDELL) or if he has taken quinine or quinidine (SOLANDT & MAGLADERY). On the other hand, preliminary warming of the denervated muscle, massage, passive or active motion (HAASE, 1961), mechanical

irritation and the administration of thyroid will increase the number and perhaps also the frequency of spontaneous fibrillations, according to RICHARDSON & WYNN PARRY particularly after the denervated muscle has been stimulated by interrupted electrical current. The same effect is seen after the administration of acetylcholine, physostigmine (eserine) and prostigmine (neostigmine), as has been mentioned in detail in the earlier part of this chapter.

Persistence of fibrillation

Spontaneous fibrillation continues as long as muscle tissue is present and power of contractility is preserved. It may persist for a variable time up to many years after injury of the axon. Much mentioned is the case-history reported by FEINSTEIN, who recorded fibrillation action potentials from the paralysed facial musculature of a man, aged 45, who sustained this paralysis at birth. A still more exceptional case has been described by MARINACCI. This author found fibrillation potentials in a woman, aged 59, afflicted with poliomyelitis when she was 5 years old.
If reïnnervation does not take place atrophy and fatty degeneration of the muscle fibres will occur with a corresponding decrease in fibrillary activity. When atrophy is complete fibrillation potentials cannot be found (TOWER). The denervated muscle has ondergone morphological changes and is no longer recognizable as muscle. Fibrosis will develop, a symptom which may be felt on insertion of the needle electrode.

From the foregoing one may conclude that fibrillation is a physiological response of skeletal muscle to denervation. This fibrillation continues as long as muscle tissue is present. It is, however, of utmost importance to keep in mind the statement of GOLSETH & FIZZELL (1947), that fibrillating skeletal muscle is *viable* contractile muscle, though denervated, having the capacity for reïnnervation. Whether fibrillation is present or not will be conclusive in cases where surgical intervention is considered (decompression, nerve graft); if fibrillation voltages are not present such intervention will be of no value, because there will be no muscle tissue to reïnnervate.

RECAPITULATION OF ELECTROMYOGRAPHIC FINDINGS IN PERIPHERAL NERVE LESIONS

Clinically, the terms complete and incomplete paralysis (paresis) are used in accordance with the severity of the paralysis. Electromyographic examination in these situations can yield the following findings.

Clinical complete paralysis

If the nerve lesion is so severe as to entail degeneration of all nerve fibres, then the muscles supplied by that nerve show complete electrical silence during the period following the onset of paralysis, both during relaxation and during maximal contraction effort. Some fibrillation potentials and/or positive sharp waves can be detected after 12-14 days; fibrillating muscle fibres are found, initially widely scattered through the muscle but in an increasing number of localizations after a longer duration of the paralysis.
Clinical complete paralysis can also exist without degeneration of nerve fibres, i.e. in the case of reversible conduction block (neurapraxia). In such cases electromyographic examination as a rule reveals one or several discrete motor unit action potentials at maximal contraction effort; apparently, however, this is not sufficient for visible muscle contraction.
Differentation between clinical complete paralysis caused by nerve degeneration and paralysis resulting from reversible conduction block can be very difficult or even impossible in an early stage of the paralysis. Serial electromyographic examination finally decides in these cases; in the case of reversible block, motor unit potentials re-appear at a given time, while fibrillation is found after some time in the case of degenerative paralysis.

Clinical incomplete paralysis (paresis)

The qualification "clinical incomplete or partial paralysis" comprises the entire range of variations between the normal situation and total paralysis. This also holds true for the electromyographic features. In the case of severe but not complete paralysis, maximal contraction effort is associated with no more than a pattern of single motor unit potentials; less severe affections can be associated with a mixed pattern but a complete interference pattern is not as a rule attained.

If, in the paretic muscles, denervation potentials (fibrillations, positive sharp waves) are found in addition, this indicates degeneration of part of the nerve fibres; the extent of fibrillation observed (discrete or profuse) gives an impression of the extent of these degenerations.
It is understandable that mixed lesions in widely diverse forms can be found in this way — particularly in nerves which supply a large number of muscles, such as the facial nerve. Dependent on the severity of the nerve lesion it is possible, not only that merely part of the muscles is affected but also that the extent of paralysis varies in the muscles affected (in one muscle more motor units may be affected than in the other); also, the affection can be transient in some motor units (reversible conduction block), whereas in others the axons degenerate.

Regeneration

According to many investigators reïnnervation is initiated by a progressive decrease in the number of fibrillation potentials; however, it is difficult to measure the amount of decrease with any degree of accuracy. In any case complete cessation of fibrillation is rare.
The first reliable sign of re-innervation is the appearance of base line disturbances upon voluntary contraction effort; after a short time it becomes obvious that these are caused by discrete low-voltage motor unit potentials (100-200μV.) of considerable duration (up to 15-20 msec.). These potentials are highly polyphasic and are quickly exhausted; initially they cannot be sustained for more than a few seconds at a time. The complex polyphasic appearance and the protractedness of these potentials is explained by asynchronous discharge of the individual muscle fibres due to differences in length and conduction velocity between the newly regenerated terminal axon branches, and differential end-plate delay.
As re-innervation proceeds these action potentials (called "*nascent*" motor unit potentials by WEDDELL c.s., 1943) increase in voltage and become less complex; this indicates improved synchronization of the component muscle fibres. Normal diphasic or triphasic forms eventually occur. The number of motor unit action potentials meanwhile increases; patients who make a good recovery may finally show a partial interference pattern or occasionally even a complete interference pattern of motor unit activity at maximal contraction effort. In patients making a fair recovery, only a

partial interference (mixed) pattern can be attained. If recovery is poor, only discrete motor unit potential discharges are possible.

According to JASPER & BALLEM (1949) and MARINACCI (1955), visible voluntary movement does not appear until the voltages of the nascent potentials are of the order of 500-600 μV. The average time between appearance of these potentials and the first detectable clinical contraction in the muscle, is about 8 weeks (WYNN PARRY, 1953; LICHT, 1954; RODRIQUEZ & SKOLNIK, 1954; MCGOVERN & FITZ-HUGH, 1956).

Fibrillations of the insertion type have been left undiscussed in this review. According to some authors, these mechanically aroused potentials precede the appearance of spontaneous fibrillations; both are regarded as a sign of axon degeneration. We have seen spontaneous fibrillation in many cases in which we had neither seen nor heard insertion fibrillations at insertion of the electrode needle. On the other hand, if unmistakable insertion fibrillations were found, sustained spontaneous fibrillation as a rule also existed; in these cases there was no appreciable separation between the two fibrillation types. In our study, we exclusively considered the presence or absence of rhythmically repeated fibrillation potentials without considering the question of whether these were "spontaneous" or "aroused by mechanical irritation".

ELECTROMYOGRAPHIC EXAMINATION OF PATIENTS WITH FACIAL PARALYSIS

Electromyographic examination of the facial musculature requires special care. Most muscles, and particularly the frontal and the orbicularis oculi muscle, consist of a very thin layer of muscle fibres, the thickness of which does not exceed one to a few millimetres; the surface area of the muscles, however, is relatively large. Consequently a search of these thin muscle shells with the aid of needle electrodes is a subtle procedure which must be effected with great prudence, the more so because most of these muscles

are localized in virtually direct contact with the faciocranial bones; touching the periosteum with a too far advanced needle is exceedingly painful. Very thin electrodes (Ø 0.5 mm or less) are therefore used in examining these muscles. These electrodes must be inserted at a very sharp angle, the needle being kept almost parallel to the skin surface.

Even when the patient has been carefully instructed as to what will happen, insertion of needles into the face is an alarming experience for most patients; especially in the vicinity of the eyes, this must be done with great caution lest defence movements of the patient surprise the examining physician. This risk of such a surprise is smaller when the patient's head is supported, which also facilitates the investigator's control over his movements when inserting the needles. For this reason, and also to promote the muscular relaxation required for examination (less readily ensured in the facial muscles than elsewhere in the body), we carry out all examinations with the patient in *dorsal recumbent* position.

Lest hypothermia of the paralysed muscles (patients are often examined in the out-patient clinic) conceals some phenomena such as fibrillation potentials (see page 87.), examinations were carried out routinely under an infra-red lamp placed above the patient's face at a distance which ensures a temperature of 37°C. at the level of the forehead. To facilitate detection of possible fibrillation potentials, moreover, the majority of patients receive a prostigmine (neostigmine) injection; adults are given 1 mg prostigmine intramuscularly 10-15 minutes before examination is started.

Before the electrode is inserted, the skin is cleansed with acetone. The patients do prefer this agent to alcohol; because it evaporates very swiftly, it causes only very brief irritation of the eyes.

The muscles examined are as a rule the frontal, orbicularis oculi, quadratus labii superioris and quadratus labii inferioris muscles. The orbicularis oris muscle is not included in the examination because in this sphincter muscle fibres can be innervated from across the median line, through nerve rami of the unaffected facial nerve; this impedes correct interpretation of the electromyogram of this muscle. For the same reason, only the lateral half of the frontal muscle is always examined. For examination of the orbicularis oculi muscle, the electrode is inserted next to the lateral corner of the eye. If necessary, the needle is advanced from this point to either the pars superior or the pars inferior. For examination of the quadratus labii superioris muscle, the needle is inserted into the nasolabial fold next to

the nostril. The quadratus labii inferioris muscle is examined via an insertion vertically below the corner of the mouth and about 1 cm. above the inferior margin of the mandible. Since this muscle is partly covered by the triangular muscle, these two muscles have been regarded as an entity in interpreting the electromyogram.

Results

It has been shown in the previous chapter that degeneration of nerve fibres gives rise to fibrillations in the muscle fibres communicating with the axons affected. The fluctuations in potential associated with the fibrillations, can be electromyographically recorded; these fibrillations therefore constitute an objective datum characteristic of denervated muscle. In the chapter on degeneration and regeneration it was pointed out that paralyses associated with nerve fibre degeneration can only be cured by virtue of regeneration of the dysfunctional axons and/or collateral sprouting of axons that have remained intact. In this respect it is pointed out that a recovery made by this mechanism is seldom, if ever, complete; nearly always there are sequelae (incomplete return of voluntary or affective movement, synkinesis, contracture).

If these remarks are correct, the conclusion is justified that recording of fibrillations at electromyographic examination indicates the necessity of bearing in mind the serious possibility of incomplete cure of the existing paralysis.

Of the 220 patients with peripheral facial paralysis whom we treated in the course of 3 years, 197 were electromyographically tested; this was done at the first examination and, if necessary, on several subsequent occasions until we believed to have attained sufficient understanding of the situation. In the case of patients seen shortly after the onset of paralysis, this meant that electromyographic examinations were carried out every other day during the period in which possible fibrillations might first be observed (10th to 20th day); if no fibrillations were found, the examination was repeated at longer intervals up to 6 weeks after the onset of paralysis. In the case of patients seen late (e.g. six weeks or more) after the onset of paralysis, a single electromyographic examination was as a rule sufficient. In patients in whom the first signs of incipient clinical recovery remained absent longer than usual, electromyography was carried

TABLE XVI. RELATION BETWEEN PRESENCE OR ABSENCE OF FIBRILLATION AND ULTIMATE RECOVERY IN 159 PATIENTS WITH PERIPHERAL FACIAL PARALYSIS.

		fibrillation	synkinesis	contracture	incomplete return of voluntary movement	complete return of voluntary movement without synkinesis or contracture
Bell's palsy	92	+ 69	67	57	62	2
		— 23	3	3	2	20
Surgical trauma	17	+ 17	15	11	13	—
			no recovery at all 2			
Head injury	19	+ 18	16	12	14	1
			no recovery at all 1			
		— 1	—	—	—	1
Acute otitis media	8	+ 2	—	—	—	2
		— 6	—	—	—	6
Chron. otitis media	9	+ 9	9	7	9	—
External otitis	1	+ 1	1	1	1	—
Aural herpes zoster	5	+ 4	4	3	3	—
		— 1	—	—	—	1
Tumours	4	+ 4	1	1	1	—
			no recovery at all 3			
Inf. mononucleosis	1	+ 1	—	—	—	1
Uncertain cause	3	+ 3	2	2	1	—
			no recovery at all 1			
Total	159	+ 128	115	94	104	6
			no recovery at all 7			
		— 31	3	3	2	28

out again later so as to ensure early detection of such regenerative signs as might exist.

In the group of 197 patients thus examined, there were 159 who could be followed up at regular intervals for at least 1 year after the first examination. The data on the incidence of fibrillations in these patients, and on the condition at the final follow-up, are presented in Table XVI.

These data warrant the following conclusions:

— of 128 patients in whom fibrillations were found, 6 (5%) made a full recovery without sequelae; in 7 (5%), complete paralysis persisted; 115 patients (90%) made an incomplete recovery with residual symptoms. Synkinesis was seen in 100% of these 115 patients; contracture*) occurred in 94 (75%) and incomplete return of voluntary movement was seen in 104 (80%). In 11 patients, therefore, return of movement was complete but there was nevertheless synkinesis or contracture, although usually only in a mild degree.

— of 31 patients in whom no fibrillations were found, 28 (about 90%) made a full recovery without sequelae; residual symptoms were seen in 3 patients, all suffering from Bell's palsy. In 1 of these, return of voluntary movement was complete but, like the other 2, this patient showed a very slight contracture and synkinesis.

We believe that these data convincingly indicate the necessity of bearing in mind the possibility of an incomplete recovery when fibrillations are found. On the other hand, in the absence of fibrillations, complete recovery can be expected with some certainty.

In a small number of cases recovery differed from expectations based on the electromyogram. Six patients in whom fibrillations were found, nevertheless made a full recovery without sequelae. This may be explained by faulty interpretation of the electromyogram; on the other hand it is possible that the number of degenerate axons was so small that such regenerative sequelae as existed failed to give rise to clinically appreciable symptoms; the EMG report on 4 of these patients does in fact mention that the number of fibrillation potentials was very small ("minimal", "limited"). Three patients in whom no fibrillation was found, ultimately

*) Like LIPSCHITZ (see chapter *"Degeneration and Regeneration"*), we found associated movements in all contracture cases; the reverse was not always true in our patients.

showed residual symptoms. Our observation must have been faulty in these cases; in following the course in these paralyses, we have probably been more intent on the increasing motor unit activity than on the denervation potentials which must also have existed.
However, the number of cases (9 out of 159) in which an incongruence between electromyographic findings and the condition at the final follow-up occurred can be regarded as so small as to be hardly incompatible, in our opinion, with the prognostic significance of the presence or absence of fibrillations.

Discussion

In 1955, Taverner reported the results of an electromyographic study which he made in 96 cases of Bell's palsy of recent onset. He found fibrillations in 51; 49 made an incomplete recovery. No evidence of denervation was found in 45 patients; 44 of these recovered completely. Taverner mentioned three mistakes in assessing the prognosis of these patients.
The following table compares our findings with those of Taverner.

Bell's palsy	Number of patients	Fibrillation	Complete recovery	Errors
Taverner	96	+ 51	2	2
		— 45	44	1
Our data	92	+ 69	2	2
		— 23	20	3

Our findings, therefore, are in agreement with those of Taverner although we have a few more errors. We can completely confirm his conclusion that: "modern electromyography provides an accurate guide to the prognosis in Bell's palsy". However, we would prefer not to confine this conclusion to Bell's palsy only but to extend it to all forms of peripheral facial paralysis, particularly those of traumatic origin.

Feinstein (1946) reports on 12 cases of traumatic facial paralysis (*head injury*), examined electromyographically. In 4 of these there was no evidence of denervation and they all made a complete recovery. In the other cases there was interruption of a varying number of axons; the patients recovered but all showed associated movements. Comparison shows an unmistakable agreement between Feinstein's findings and ours:

Head injury	Number of patients	Fibrillation	Associated movements	Complete recovery
Feinstein	12	+ 8	8	—
		— 4	—	4
Our data	19	+ 18	17	1
		— 1	—	1

The fact that the presence or absence of fibrillation potentials is of great signification in assessing the prognosis of peripheral nerve lesions, has also been pointed out by other investigators, e.g. Golsch (1949): "If no fibrillation occurs after paralysis for 3 weeks, there is either a good prognosis or simulation"; and Marshall (1959): "The presence of fibrillation in the electromyogram is of bad import, whereas its absence promises a good degree of recovery no matter how severe the paralysis". Marinacci (1955) stated: "The clinical application of electromyography depends as a whole upon fibrillation of denervation rather than upon motor unit activity"; Haase (1961) maintained that: "The basis for present day clinical electromyography is the fibrillation of denervation voltages found in denervated muscle". In 1949 Bercel put it in a different but none the less clear manner: "In the potentials associated with denervation fibrillation we learned to recognize one of the most reliable pathognomonic signs in clinical medicine. The electromyographer goes after this sign with the tenacity of a bulldog and, if it does not appear spontaneously, he will warm up the muscle or try to bring it out by injection of neostigmine".

Yet the possibilities of electromyography would only be partly used if it would be confined to the search for fibrillations. Apart from the presence

or absence of fibrillation, after all, it is of great significance whether or not an attempt at muscle contraction reveals a discharge of motor unit activity and, if it does, to what extent. This is also pointed out by HIRSCHBERG & ABRAMSON (1950), who stated: "The relative number of fibrillation potentials and of motor unit potentials allows one to make an appropriate evaluation of the degree of paralysis in any part of the muscle". There is of course a great difference between the finding of a few fibrillations in a muscle still capable of a strong discharge of action potentials at a contraction effort, and the finding of these fibrillations in complete paralysis, in which even maximal contraction effort fails to produce action voltages. In the former case, only a few muscle fibres are denervated and very good if not complete recovery can be expected; in the latter case the prognosis must be considered very gloomy.

It is a firm neurological principle, however, that anomalous phenomena guide the examination, and in this respect the fibrillations constitute an objective datum of paramount importance in assessing the functional status of the final common path (GOLSETH & HUDDLESTON, 1949).

With a view to therapeutic measures to be taken, importance must be attached to the time at which the fibrillation potentials are obtained. So far as we know from the literature, TAVERNER breaks all records in the statement that "In patients seen sufficiently early, profuse fibrillation activity was usually detected by the 7th to the 10th days; in some patients scanty fibrillation potentials were detected as early as the 4th day, becoming profuse after 7 days". As has been indicated in detail in a previous chapter, nearly all investigators agree as to a time of onset of fibrillation potentials 2-3 weeks after nerve injury *in man*. It was pointed out in particular that WEDDELL, FEINSTEIN & PATTLE (1944), studying 75 cases of peripheral facial paralysis, found that the onset of fibrillation never exceeded 14 days after the onset of paralysis. Our average time (see page 87.) was 13 days, which agrees well with the above data. This means that KETTEL (1959) is correct in his statement that "electromyography is useless as an emergency guide". For, not only are we in the dark as to the condition of the nerve fibres during the first two weeks, but *when* the first fibrillations are found (on the 13th or 14th day) it is too late; the axons supplying the fibrillating muscle fibres have already degenerated.

This doet not mean, however, that all axons will degenerate. When electromyography at a given moment reveals fibrillations and also motor

unit action potentials at a contraction effort, the conclusion is reached that part of the nerve fibres is degenerated while other fibres have remained intact. In our opinion it is not unreasonable to assume that, in addition to the degenerate and the intact nerve fibres, there is a third category of fibres showing a disturbance in nerve impulse conduction which, however, is not yet irreversible (neurapraxia). Appropriate treatment affords a good chance of functional recovery of these fibres. By the same reasoning we believe that the finding of fibrillations in clinically and electromyographically complete paralysis need not necessarily mean that all nerve fibres will degenerate. We find corroboration in this respect in MATTHEWS' statement (1959) that: "Degeneration is not an all-or-none phenomenon; some fibres may degenerate and others remain intact". COLLIER has also pointed out that lesions may be mixed — some axons undergoing degeneration while others remain in a condition of reversible block and are capable of recovery. Support was also found in the views of FEINSTEIN (1946), BOWDEN (1951) and COLLIER (1959) that a condition of reversible block may persist as long as 6-12 weeks after the onset of facial paralysis. Also, the *rapid* return (within a few days or a week) of at least some function after decompression of the facial nerve (claimed by ear-surgeons such as CAWTHORNE, DUEL, HALL, JONGKEES, KETTEL, SANDERS, SOKCIC and SULLIVAN) can only be explained if it is assumed that the nerves affected have contained a number of undegenerated axons for which surgical decompression was life-saving.

SUMMARY

It has been repeatedly pointed out that the crucial point is to know the severity of the functional nerve impairment in each individual case of facial paralysis, at a time sufficiently early to permit of measures counteracting a possibly imminent unfavourable course. On the basis of the previously discussed clinical data — e.g. extent of paralysis, muscle tone and nasopalpebral reflex — only a general prognosis can be made. Yet, in its generality, this prognosis affords a few definite data, viz: virtually no chance of full recovery when paralysis is complete at the end of the second week, and virtually 100% chance of full recovery when paralysis is partial from the onset, with good muscle tone and persistent nasopalpebral reflex. Clinical examination, however, affords no objective prognostic data appreciable or measurable in the *individual* patient himself. Electromyographic examination does provide such data. Now let us consider

whether the questions previously raised can be answered with the aid of electromyography.

First question: *Is this paralysis transient (reversible nerve block) or is it a degenerative disorder of conduction so that recovery is dependent on regeneration of the nerve fibres affected?*
In view of the fact that fibrillations are not demonstrable until 10-14 days after the onset of paralysis at the earliest, no answer can possibly be given to this question before the end of the second week, regardless of whether complete or partial paralysis is involved. If fibrillations are found at or after this time, this fact indicates that at least some nerve fibres have already degenerated and that incomplete recovery must be expected. Absence of fibrillations after the abovementioned time, particularly to the extent to which the interval since the onset increases, warrants the hope that the nerve lesion is of a reversible nature. Certainty as to whether a paralysis is or is not transient, can be attained only in the negative sense because the only objective datum available — fibrillation — is as such a sign of degeneration.

Second question: *If the paralysis is still in the reversible stage at the time of examination, are there any signs (if so, which?) heralding an imminent unfavourable development, i.e. irreversibility?*
The answer is: no. Neither in partial nor in complete paralysis does electromyography afford any information in this respect.

The practical conclusion at the end of this chapter is that, on the basis of electromyographic findings, *it is possible* to select individual patients with an unfavourable or at least dubious prognosis, but not before the 10th-14th day after the onset of paralysis. Since the phenomenon which marks these patients is as such a sign of axon degeneration, it is at that time unfortunately too late for prevention — by any means now known — of incomplete recovery. However, if it is borne in mind that degeneration is not an all-or-none phenomenon (as previously explained in detail), and if suitable measures are taken upon the first alarming sign (the first fibrillations), then it is probably still possible to protect a number of nerve fibres from degeneration or at least to promote regeneration of axons which have already given way. This is what KETTEL (1954) described as "the key to the understanding of the effect of decompression".

ASSESSMENT OF NERVE CONDUCTION

INTRODUCTION

COLLIER (1959) maintains that, during the first few days after the onset of facial paralysis, information on the excitability of the nerve can only be obtained by testing for *nerve conduction*. RICHARDSON & WYNN PARRY (1957) confirm that assessment of nerve conduction is the most important test in the electro-diagnosis of facial paralysis; it is the first electrical examination to be made. Nerve conduction can be demonstrated in that muscle contraction is produced by stimulation of the nerve trunk at any point in its peripheral course. In the nerve conduction test, the threshold value of excitation is determined. This is done by measuring the intensity of the stimulus minimally required to produce muscle contraction. This requires a stimulator capable of delivering stimuli of measurable duration and strength. In this way nerve testing can be done on a quantitative basis. The functional state of the nerve can be established by comparing the results of examinations repeated, if necessary, every day.
If a nerve lesion blocks the conduction of nerve impulses, stimulation of the nerve at a site proximal to the lesion fails to elicit a contraction in the appropriate muscles; this loss of nerve conduction can result from any dysfunction of the nerve, viz. neurapraxia. However, if stimulation of the nerve *distal* to the lesion fails to cause muscle contraction, this implies degeneration of the peripheral part of the nerve (BOWDEN, RICHARDSON & WYNN PARRY, COLLIER).

RICHARDSON & WYNN PARRY (1957) contend that *degenerative nerve lesions* result in cessation of conduction in the peripheral part within about 72 hours. BOWDEN (1951), examining nerve lesions in the limbs, reported that: "When nerve fibres had been completely interrupted by damage or disease, stimulation of the nerve trunk was ineffective if it was applied above the level of the lesion. However, during the first two or even three

days after infliction of the damage it was possible to elicit a contraction in the appropriate muscles by stimulating the nerve trunk below the lesion, but there was a gradual rise in the threshold of effective stimuli of short duration; by the 3rd or 4th day nerve conduction failed below the lesion and there was clear evidence of degenerative lesion of the nerve fibres". LANGLY & KATO (1915); LONGET; ERLANGER & SCHOEPFLE (1946) and LANDAU (1951) demonstrated in various experiments on animals that conduction in the distal segment of a severed peripheral nerve disappears after a maximum period of 2-5 days. LANDAU (1953) applied electrical stimulation to the peripheral portion of human ulnar and median nerves after traumatic severance. He concluded that, as in other mammals, motor function was completely abolished 3-5 days (91-128 hours) after the lesion was produced. In another patient the buccal branch of the facial nerve was cut in the parotid region and complete loss of excitability was seen on the fourth day (after 91 hours). GILLIATT & TAYLOR (1959) carried out a similar investigation in a number of patients following complete severance of the facial nerve in excision of a neurinoma. Response to nerve stimulation was not only visually estimated but also electromyographically recorded with the aid of coaxial needle electrodes. Their results completely confirm LANDAU's observation. In their patients, the visual muscle twitch in response to nerve stimulation disappeared within 3-4 days; in all cases the electromyographic response persisted for a further 48-72 hours. Similar data have been presented by HINES, WEHRMACHER & THOMSON (1945): 3-4 days; WATKINS (1947): a minimum of 4-5 days; MURPHEY (1949): 2-4 days; BOWDEN (1951): 3-4 days; BUNNELL (1952): 3-4 days; MARINACCI (1955): 4-5 days; RODRIQUEZ & OESTER (1956): 2-3 days; WILLIAMS (1959): 2-4 days.

If injury to a nerve causes *transient block* of conduction of the nerve impulses, stimulation of the nerve trunk above the site of the block fails to elicit any muscle contraction; stimulation *below* the level of the lesion, however, results in brisk contraction of the appropriate muscles throughout the period of paralysis. COLLIER (1955) maintains that, if in facial paralysis muscle contraction results when the facial nerve is stimulated at its exit from the stylomastoid foramen, any lesion proximal to the foramen is non-degenerative or causes only a minimal degree of degeneration. RICHARDSON & WYNN PARRY (1957) contend that, in cases of facial paralysis, maintenance of normal nerve conduction beyond the third day carries a good

prognosis, and that beyond the week an excellent one. "Rapid recovery is to be expected in those cases since the lesion is mainly a neurapraxia". This opinion is shared by GREINER et al. (1960), who state that preservation of excitability of a nerve beyond the third day is conclusive of a transient block (neurapraxia).

However, we should beware of over-optimism so shortly after the onset of paralysis. This has been emphasized by such authors as LANDAU (1953), who points out that degeneration can hardly be expected to be as acute or as complete in Bell's palsy as in traumatic nerve injury. This is stressed also by WILLIAMS (1959) and GILLIATT & TAYLOR (1959), who point out that degeneration is certainly not always so rapid in Bell's palsy as in cases of nerve severance. GILLIATT & TAYLOR state that re-examination up to 2 weeks may be necessary in some cases before it can be ascertained that degeneration is not going to occur. FEINSTEIN (1946) and BOWDEN (1951) indicate that a conduction block may persist for as long as 6-12 weeks! Similar findings have been recorded by COLLIER (1959), who found complete clinical paralysis without evidence of degeneration after up to 11 weeks; she warns against confusing a persistent neurapraxia with a more destructive lesion.

CLINICAL ASSESSMENT OF NERVE CONDUCTION

The main nerve trunk which supplies the muscles under examination is stimulated with a pulse of short duration at gradually increasing intensity, until the minimal visible muscle contraction is obtained; the amount of current required to produce this contraction — the *threshold intensity* — is recorded. An identical procedure is repeated on the normal side, and the two threshold values are compared. To obtain reliable data, adequate equipment (stimulator, electrodes) is a prerequisite. A constant testing technique is equally important.

For assessment of nerve conduction in *facial paralysis,* the stimulus is applied to the facial nerve in the region of the stylomastoid foramen. GILLIATT & TAYLOR (1959) stimulated the facial nerve percutaneously, immediately in front of the ear; a saline pad (Ø 1 cm) was used as stimulating electrode; the anode was a large metal plate strapped to the neck; the stimulus used was a pulse of 0.1 msec. duration. BOTELHO et al. (1952) cemented the active electrode (circular silver cup, Ø 0.7 cm) to

the skin anterior to the external auditory meatus; the duration of each stimulus was 0.5 msec. or less. LANDAU (1953) applied stimuli of 0.1 msec. duration, and RICHARDSON & WYNN PARRY tested with 0.3 msec. pulses.

PERSONAL OBSERVATIONS

Stimulator

We used a current-stabilized electronic stimulator, designed and constructed at the physical laboratory of our department. The circuit diagram of the stimulator is shown in fig. 18. The chief characteristics of the stimulator are:

pulse duration	: adjustable from 0.1 to 100 msec.
current strength	: variable from 0.1 to 20 mA.
maximum voltage swing allowed at the output terminals	: 80 V.

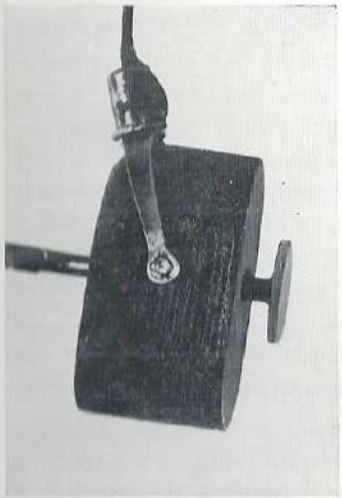

Fig. 16. Pertinax block with stimulating electrode.

Electrodes

The stimulating electrode used was a circular metal platelet (Ø 1 cm), centred in and slightly protruding from a block of pertinax (see fig. 16); the block is flexibly attached to a normal earphone headband. A large copper plate strapped to the nape of the neck is used as reference (earth) electrode.

Procedure

In order to ascertain good contact between the electrodes and the skin, and to reduce the skin resistance, the nape and the preaural region are cleansed with acetone and well greased with electrode paste. The reference electrode is strapped onto the nape, and the active electrode is placed

anterior to the external meatus in such a way that the upper margin of the pertinax block rests against the inferior aspect of the zygomatic arch. In this position the facial nerve — embedded in the parotid gland — is caught between the pertinax block and the lateral surface of the ramus of the lower jaw. The design of the electrode block is such as to ensure that the platelet electrode lies over the site at which the facial nerve trunk divides into its terminal rami (see fig. 17).

Fig. 17. Correct position of the stimulating electrode.

The facial nerve is percutaneously stimulated with a square pulse of 0.3 msec. duration, at gradually increasing intensity. The amount of current required to obtain the minimal visible muscle contraction is recorded.

The test is first carried out on the normal side. The threshold values of the affected and those of the unaffected contralateral side are compared for the orbicularis oculi and the orbicularis oris muscles. Because the difference in nerve conduction values (of the affected and of the unaffected contralateral side) of the two muscles are as a rule similar, in this study only the determinations for the orbicularis oculi muscles were quantitatively elaborated.

Normal values

The threshold intensity is dependent on a wide variety of factors, e.g. skin resistance, skin temperature, thickness of the layer of soft parts (skin, subcutis, parotid gland) between active electrode and nerve, and the

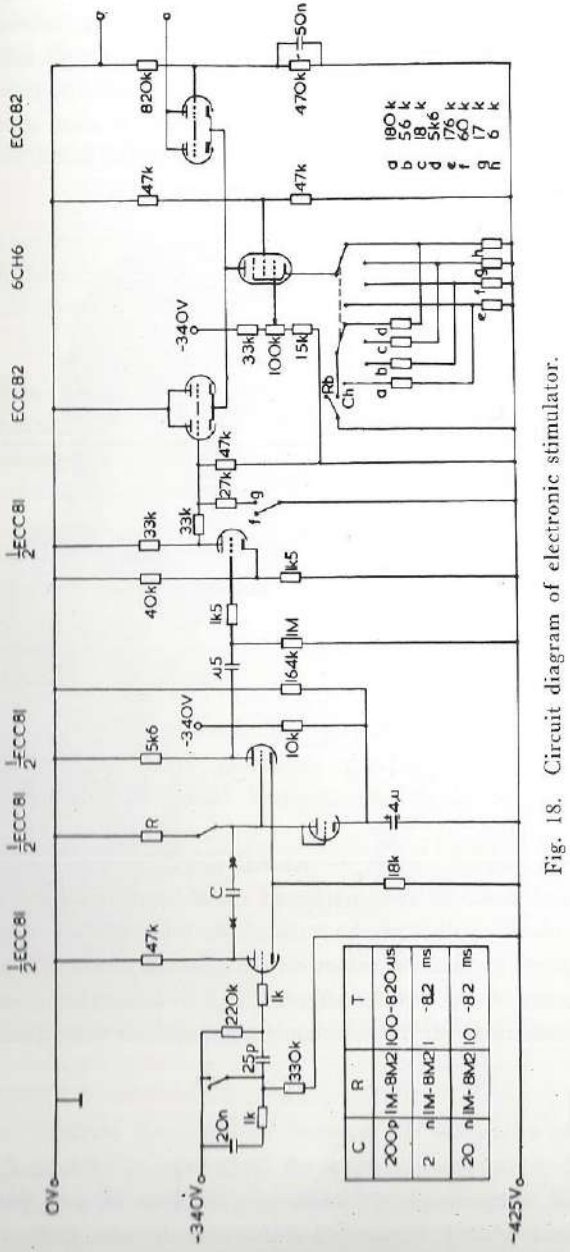

Fig. 18. Circuit diagram of electronic stimulator.

position of the electrode. These extrinsic conditions, and probably also the anatomical course of the facial nerve and the site of its division into the terminal rami, can show individual variations so marked as to make it impossible to set a standard normal threshold intensity value as criterion for all determinations. The average threshold intensity for the orbicularis oculi muscle on the unaffected side found in 459 tests carried out in 141 patients, was 6.5 mA.; the spread of normal values was from 2.4 to 16.2 mA. — extremes which clearly illustrate the above considerations. Not only does the normal threshold intensity vary from patient to patient, but it also varies from test to test in the same patient and in the same nerve, under carefully controlled identical conditions. The range of variation is usually limited (cf. Table XVII, patients A and B); in some cases, however, determinations vary considerably (cf. patient C).

TABLE XVII. THRESHOLD INTENSITY VARIATIONS OF THE UNAFFECTED FACIAL NERVE AT DIFFERENT TESTS.

Patient A	*Patient B*	*Patient C*
7.5 mA.	6.7 mA.	8.2 mA.
8.0 —	6.9 —	5.8 —
7.0 —	7.3 —	4.0 —
8.0 —	6.6 —	7.0 —
8.0 —	7.3 —	9.0 —
8.0 —	7.8 —	9.8 —
7.2 —		7.8 —
		9.3 —

In normal test subjects it was found that such fluctuations occur simultaneously and in the same proportion on both sides; this would seem to suggest that the patient's emotional condition, the skin resistance, the temperature of the skin or of the environment, and other *general* factors are responsible for this. In any case, the L/R difference is not appreciable influenced as a result.

The only possibility to reduce the number of variables to a minimum is to determine the nerve conduction in both facial nerves at every test, and to compare the values obtained on the affected side with those obtained on the unaffected contralateral side. If such an investigation is made in normal subjects, differences between right and left are nearly always

found. This is understandable because it is virtually impossible to place the active electrode in exactly identical positions on both sides, and minor anatomical differences between the two facial halves are always possible. The differences found, however, are as a rule very small. The nerve conduction of the facial nerves was determined in 20 normal test subjects aged 20-30. An average threshold intensity difference (left-right) of 0.4 mA. was found, with a standard deviation of 0.2 mA. A zero hypothesis (H_0) of symmetrical distribution of current strength differences with respect to zero was verified in the course of statistical elaboration of the data obtained.*)

Applying the WILCOXON *test for symmetry* (with correction for ties, normal approach), a two-sided tail probability of 0.0872 was found, by which the hypothesis H_0 is not rejected.

This means that no significant nerve condition difference between the two sides was found.

DETERMINATIONS IN PATIENTS WITH FACIAL PARALYSIS

Assessments of nerve conduction were carried out in 141 patients with facial paralysis. Table XVIII shows the distribution of these patients according to the aetiology of the paralysis.

TABLE XVIII. CAUSES OF FACIAL PARALYSIS IN 141 PATIENTS SUBMITTED TO ASSESSMENT OF NERVE CONDUCTION

Bell's palsy	76
Head injury	17
Surgical palsy	14
Chronic otitis media	10
Acute otitis media	6
External otitis	1
Aural herpes zoster	9
Uncertain	8
	141

*) Statistical analysis of the data was carried out under the direction of Drs W. R. VAN ZWET and Drs P. VAN DER LAAN of the Statistical Department (Head: Prof. Dr J. HEMELRIJK) of the Amsterdam Mathematical Centre. A review of the statistical methods used is given in an appendix. We will take this opportunity to express our gratitude to Drs VAN ZWET and Drs VAN DER LAAN for their interest and help.

As has been pointed out, the maximum output of the stimulator is 20 mA. This extreme strength of current is used only when no muscle contraction is seen upon application of lower intensities. Doubtless because of their brief duration (0.3 msec.), these 20 mA. impulses are well tolerated by all adults without exception; in children the examination sometimes offered difficulties. At higher intensities, contraction of the masseter muscle occurred as a result of direct stimulation; at each pulse this caused a twitch of the mandible and the cheek, which had a disturbing effect. This impeded assessment of muscle contractions around the mouth. By asking the patient to lock his jaws (pull on the masseter muscle), we largely neutralized this disturbing influence.

In 71 patients — who were followed up for at least 1 year after the onset of paralysis — no reaction of the facial musculature was seen at a given moment to a pulse strength of up to 20 mA. Table XIX shows the distribution of these patients according to the cause of paralysis.

TABLE XIX. CAUSES OF FACIAL PARALYSIS IN 71 PATIENTS SHOWING NO NERVE CONDUCTION AT PULSE STRENGHTS UP TO 20 mA.

Bell's palsy	33
Head injury	12
Surgical palsy	10
Chronic otitis media	4
External otitis	1
Aural herpes zoster	5
Uncertain	6
	71

At the time when this absence of nerve conduction at 20 mA. pulse strength was observed, none of these patients had been surgically treated. Patients who had had an operation were not included in this series (to eliminate the possible objection that the loss of nerve conduction could be a result of the operation — a subject to be discussed later). None of these 71 patients made a full recovery without residual symptoms. Electromyographic exami-

nations were made in 64 of these cases; profuse fibrillations were found in all. These data definitely suggest that absence of nerve conduction upon application of pulses of up to 20 mA. intensity is indicative of a *degenerative* character of the conduction disorder. This is in agreement with the views of RICHARDSON & WYNN PARRY (1957).

Any attempt at determining the earliest time, after the onset of paralysis, at which absence of nerve conduction can be encountered, is seriously thwarted because only a limited number of patients were examined at an early time. Of this series, no more than 15 patients were seen within 3 weeks of the onset of paralysis; 12 of these reported within 2 weeks and only 8 were seen within 1 week. The distribution of these patients according to the cause of paralysis and the first day on which absence of nerve conduction was found, is shown in Table XXI.

TABLE XXI. CAUSES OF FACIAL PARALYSIS AND FIRST DETECTION OF LOSS OF NERVE CONDUCTION IN 15 PATIENTS EXAMINED WITHIN 3 WEEKS OF THE ONSET OF PARALYSIS.

Cause	1st day of examination	1st detection of loss of conduction
1. Bell's palsy	2	13
2. — —	4	16
3. — —	7	11
4. — —	12	**12**
5. — —	12	**12**
6. — —	14	**14**
7. Surgical palsy	1	4
8. — —	5	**5**
9. — —	6	**6**
10. — —	6	**6**
11. — —	12	**12**
12. — —	18	**18**
13. Aural herpes zoster	1	11
14. Uncertain	4	**4**
15. —	17	**17**

(The figures printed in bold face have only a limited value; they indicate that absence of nerve conduction was observed at the first examination. It is highly probable that, at least in some of these patients, nerve conduction had been lost at an earlier date.)

Although this is only a small group of patients, the data nevertheless show that absence of nerve conduction is possible as early as the 4th day after injury of the facial nerve. This is in accordance with the observations of BOWDEN, BUNNELL, GILLIATT, HINES, LANDAU, MARINACCI, MURPHEY, RODRIQUEZ, WATKINS and WILLIAMS, as mentioned in an earlier part of this chapter. In none of the patients with Bell's palsy included in this table was loss of nerve conduction seen before the 11th day.

Our series of 141 patients includes 45 who — without operation — were clinically cured (see for our interpretation of "cured", page 35.). In 31 cases there was *complete* recovery, without residual symptoms. In 14 cases a clinical *complete** recovery was made, i.e. with residual symptoms so mild as to escape the notice of the patient and his environment.
The distribution of these cases according to the cause of paralysis is shown in Table XXII.

TABLE XXII. CAUSES OF FACIAL PARALYSIS IN 45 PATIENTS MAKING A SPONTANEOUS RECOVERY.

Bell's palsy	30
Acute otitis media	5
Surgical palsy	3
Head injury	3
Aural herpes zoster	2
Uncertain	2
	45

The differences in nerve conduction (between the affected and the unaffected facial nerves) in a homogeneous group of 30 Bell's palsy patients were statistically*) compared with corresponding data in the previously

*) See footnote page 108.

mentioned group of normal test subjects. This was done in 3 phases, viz. at the end of the 1st week (6th-8th day), end of the 2nd week (12th-14th day) and end of the 3rd week (17th-21st day) after the onset of paralysis. After each of the 3 intervals, WILCOXON's *two-sample test* showed that the conduction differences in the patients with Bell's palsy did not significantly differ from those in the normal test subjects. The two-sided tail probability after each interval was 0.242, 0.174 and 0.072, respectively (level of significance 0.05). In none of the phases, therefore, was the zero hypothesis(H_0) rejected.
This clearly indicates a very good prognosis in cases in which the threshold intensity values of the paralysed facial nerve show no abnormal difference from those of the intact facial nerve. The actual permissible extent of this difference will be discussed in a subsequent section.

Eliminating 71 patients with loss of nerve conduction and incomplete recovery, and 45 patients with nerve conduction within normal limits, who spontaneously recovered, a group of 25 patients remains out of the total of 141 patients. Of these 25, 2 failed to report after the first examination; data available on 3 others are insufficient; the remaining 20 show the following distribution according to the cause of paralysis.

Bell's palsy	11
Chronic otitis media	6
Head injury	1
Surgical palsy	1
Acute otitis media	1
	20

The patients whose paralyses were caused by head injury, surgical intervention and acute otitis media, and 5 patients with chronic otitis media, were admitted as emergency cases and immediately submitted to operation. The patient with acute otitis media underwent surgery elsewhere. The 6th patient with chronic otitis media was initially given conservative treatment based on the diagnosis Bell's palsy (found erroneous in retrospect); he was surgically treated when no improvement was seen; at operation the

cause of the paralysis was found to be a chronic otitis. In 10 cases of Bell's palsy, decompression of the nerve was carried out (in 1 of them partly in view of a relapse of paralysis). In these patients, nerve conduction was still measurable at the time of the operation, although pulse strengths required were considerably higher than those on the unaffected side. In 4 patients, nerve conduction was absent at follow-up 2 weeks after the operation; in 1 patient it did not disappear until more than a month after the operation; in 3 cases, no postoperative follow-up determinations were made; nerve conduction always remained intact in the 2 remaining patients and in the patient not surgically treated.

This is perhaps the appropriate moment to deal with the relatively widespread view that surgical decompression is more likely to damage the facial nerve than to have a favourable effect (BOONE seeks a probable explanation in obstruction of the drainage of venous blood). This is merely a bold statement not supported by sufficient evidence, as demonstrated by the following nerve conduction assessments carried out preoperatively and postoperatively in 4 of the 5 abovementioned cases of emergency surgery for facial paralysis resulting from chronic otitis media (no postoperative determinations were made in the 5th case). In all these patients, the facial nerve was thoroughly dissected out in the mastoid.

TABLE XXIII. PREOPERATIVE AND POSTOPERATIVE NERVE CONDUCTION VALUES IN 4 PATIENTS SURGICALLY TREATED FOR CHRONIC OTITIS MEDIA WITH FACIAL PARALYSIS (contralateral NC. values in parentheses).

	Preoperative		Postoperative	
Nr.	Days before operation	Nerve conduction values	Nerve conduction values	Days after operation
1.	1	6.8 mA. (5.8)	6.2 mA. (6.0)	25
2.	4	5.3 mA. (3.4)	5.1 mA. (4.8)	27
3.	3	18.0 mA. (5.0)	15.0 mA. (6.0)	16
			11.5 mA. (7.0)	31
4.	1	14.8 mA. (8.2)	13.0 mA. (6.8)	20

This table shows that none of these patients had deterioration of nerve conduction post or propter operationem.

It has been pointed out that the object of these investigations was to establish whether the prognosis of cases of peripheral facial paralysis can be predicted on the basis of nerve conduction determinations, and particularly to discern the cases with a dubious or unfavourable prognosis. All this — if possible — at a time when the course of the process can still be influenced. We have discussed 45 patients who were clinically cured; these were not significantly different from normal test subjects in terms of nerve conduction. On the other hand we saw a group of 71 patients who all made an incomplete recovery; in all these cases, nerve conduction was not demonstrable at a pulse strength of up to 20 mA. at a given moment. These data would seem to warrant the conclusion that absence of nerve conduction upon application of pulses up to 20 mA. indicates an unfavourable prognosis, whereas normal nerve conduction values suggest the possibility of a good recovery.

These two extremes — loss of nerve conduction at stimuli up to 20 mA. and nerve conduction values between normal limits — are far apart. This gap is not bridged all at once because the excitability of a nerve is not abruptly lost completely. As pointed out in the first part of this chapter, even the most severe nerve lesions are associated with *diminution* of excitability (so that ever stronger stimuli are required); in the case of severe, e.g., traumatic nerve lesion, this is a rapidly progressive process, excitability disappearing within 2-4 days. It can be imagined that, in the case of less abrupt affections such as vascular damage (Bell's palsy), this progression is slower or begins at a later time; this is also suggested by observations of previously mentioned investigators (FEINSTEIN, 1946; BOWDEN, 1951; COLLIER, 1959), showing that neurapraxia can persist for a long time. Our own observations (although made in a limited group of patients) show that loss of nerve conduction in cases of Bell's palsy was not seen before the 11th day after the onset of paralysis.

If these remarks are correct, differences in excess of normal variations should be demonstrable at a given time between the nerve conduction values on the affected and those on the contralateral side. This was investigated in Bell's palsy patients who had to undergo an operation because of a (clinically) expected unfavourable turn in the course of the

paralysis (nerve conduction assessments *were left undiscussed* in this respect). Unfortunately, as always, the number of patients who could be followed from shortly after the onset of paralysis, is only small. Only 13 of the surgical patients were seen within 3 weeks; 10 were seen within 2 weeks and 7 within 1 week.

On the basis of the data available, a comparison was made between nerve conduction differences (between the affected and the normal facial nerve) in spontaneously recovered patients, and those in patients in whom a clinically unfavourable course subsequently necessitated an operation; this was done at three different times in the clinical course (6-8 days, 12-14 days, and 17-21 days after the onset of paralysis). After each interval, but especially after the two lastmentioned intervals, WILCOXON's *two-sample test**) revealed that the nerve conduction differences in the subsequently surgically treated cases were significantly higher than those in the group of spontaneous recoveries, with one-sided tail probabilities of 0.047, 0.0014 and 0.0003, respectively (level of significance 0.05). No significant difference was found between the group of spontaneous recoveries and the group of normal test subjects. These findings warrant the statistical conclusion that nerve conduction determinations certainly give an indication of the severity of a disturbance in nerve function, particularly when carried out more than one week after the onset of paralysis.

In practice, cardinal importance must be attached to answering the question: what is the maximum permissible difference in nerve conduction between the affected and the unaffected facial nerve not yet conclusive of an imminent or already begun unfavourable course of the nerve conduction disorder? For advocates of decompression of the nerve (in suitable cases), the question is better phrased: at what difference in nerve conduction does a surgical indication exist?

We made an attempt at answering this question on the basis of a study of nerve conduction differences in patients with Bell's palsy, with reference to ultimate recovery. For this purpose we compared data on patients examined within 3 weeks of the onset of paralysis; after this interval (and sometimes earlier), electromyography yields such information, as a rule, that nerve conduction assessment is no longer essential. Data were available on 13 previously mentioned patients with incomplete recovery (and

*) See footnote page 108.

TABLE XXIV. NC. DIFFERENCES IN 39 PATIENTS WITH BELL'S PALSY WITHIN A WEEKS AFTER THE ONSET OF PARALYSIS (NR. 1-26 COMPLETELY RECOVERED; NR. 27-39 INCOMPLETELY RECOVERED).

Days after the onset of paralysis.

Nr.	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
1	-2.2		-0.8		**3.5**		0.5			1.2				0.3						0.6
2	-1.0					0			0.5				0.5							
3		0												0.8						
4		-1.8							0.8											
5		0.3			1.5				-0.7			1.3						**4.6**		
6		0			-0.9						0.3								0.2	
7			0.3				1.5		0.5				1.8							2.9
8			0.7			-1.2				0.7			1.0							
9				-1.1				0.3				-0.2								
10				-0.2							0.2								2.4	
11				1.3					1.7							0.7				0.7
12				0.1			0.9					**3.6**			2.0			**4.2**		
13				0.8											-0.4					
14					0.3							1.0							2.1	
15					2.5					0.5				-1.1			2.8			
16					-0.3					0.4										
17						2.9		1.0		2.8										
18						2.3							**5.2**							**4.9**
19				2.3							**5.5**			**3.3**				**4.1**		
20						0								-0.3						
21							-1.5			-0.4							-0.1			
22							0			0.5						-0.7			-0.5	
23							1.0													1.0
24											-1.4		-1.7				-2.5			
25												2.2				0.7				
26														-0.2						**4.0**
27	-1.5		-0.9		**4.6**			**4.6**			**4.7**				**7.2**					**12**
28	2.8		-0.2		**5.2**			**5.9**				**7.9**				**4.8**			**13**	
29	0.7					**3.9**			**11**			**>20**								
30		-0.9								**3.7**			**3**		**8.6**			**>20**		
31		1.4		-2.3		-1.2		-0.5		-1.2		-1.0				1.8	**6.8**	**7.8**		
32			1.5			1.0		2.5		1.5		1.5				0.5				
33						**4.3**						**4.6**								
34									**4.1**											**5.6**
35											**3.8**									
36											**>20**									
37														**>20**						
38															**11.6**			**>20**		
39															**4.4**					

subsequently treated by operation) and 26 clinically cured (spontaneously cured) patients. An attempt will be made to establish whether a critical difference in pulse strength can be found which indicates the maximum number of incompletely cured patients, as early after the onset of paralysis as possible, while leaving aside as many completely cured patients as possible. The data available are presented in Table XXIV. (Patients nr. 1 through 26: spontaneous cure; patients nr. 27 through 39: incomplete recovery).

The figures presented indicate the nerve conduction differences (referred to as NC. difference) between the unaffected and the injured facial nerve. The table shows that the investigation lacks systematic order throughout; this makes it impossible to compare the data from day to day. Upon the advice of statisticians of the Mathematical Centre, therefore, a comparison was made between maximal NC. differences (between unaffected and affected nerves) during specified *periods* after the onset of paralysis, in the group of incompletely cured patients and in the spontaneous recovery group, respectively. Three periods were set for this purpose, viz. 1st through 7th, 8th through 14th, and 15th through 21st day. To attain some understanding as to possibilities, a few NC. difference limits during the first period were studied. Table XXV shows the results of this study.

TABLE XXV. SOME NC. DIFFERENCE LIMITS AND THE CONSEQENT PROGNOSIS IN 27 PATIENTS (20 CURED, 7 INCOMPLETELY RECOVERED) WITHIN 7 DAYS OF THE ONSET OF PARALYSIS.

NC. difference	Unfavourable prognosis for 20 cured patients	Unfavourable prognosis for 7 incompletely cured patients
≧ 2.5 mA.	3	4
≧ 3.0 mA.	1	4
≧ 3.5 mA.	1	4
≧ 4.0 mA.	—	3

At NC. differences below 2.5 mA. an increasing number of clinically cured patients are given an unfavourable prognosis; the first incompletely cured patient to be involved does not appear until a NC. difference of 1.5 mA. is determined. As the NC. difference is increased to 4.5 mA. and over, an increasing number of incompletely cured successively leave the range of unfavourable prognostic indications.
At critical NC. differences $\geqq$ 3 mA. and $\geqq$ 3.5 mA., one patient (out of 20) would erroneously receive an unfavourable prognosis; in 4 patients (out of 7), however, the prognosis was correctly indicated as unfavourable even before the end of the first week. A more careful consideration of Table XXIV, which includes the NC. differences during the 2nd and 3rd periods, very definitely suggests that the NC. difference of 3.5 mA., determined in patient nr. 1 on the 6th day, must result from faulty determination; all determinations prior to and after his date were within normal limits. This indicates the necessity of duplicate determinations, or at least of a follow-up determination on short notice as soon as deviating results are obtained. Such a check would probably have revealed the incorrectness of this inexplicably large NC. difference. This would mean that none of the clinically cured patients would have had an unfavourable prognostic indication.
At a critical NC. difference of 3 and 3.5 mA., no indication of an unfavourable prognosis existed during the first week in 3 of the 7 patients who ultimately made an incomplete recovery; 2 of these 3 patients were still without unfavourable signs during the 2nd week, and 1 remained free of unfavourable indications even during the 3rd week; in this patient it was only on the 26th day that the NC. difference was so marked as to suggest an unfavourable prognosis. This patient was submitted to an operation, which revealed that he was not suffering from Bell's palsy but from facial paralysis as a result of (latent) chronic otitis. The question arises as to whether this patient could be included in the group under discussion; the fact that he showed all features of Bell's palsy prior to the operation, and was treated as a Bell's palsy patient, decided us in favour of including him. Apart from anything else this case clearly demonstrates just how difficult (or even impossible) it is to diagnose Bell's palsy with absolute certainty.

If, summarizing the above considerations, the starting-point were that an unfavourable prognostic indication is an indication for surgical decompression, then 1 patient out of 20 (about 5%) would erroneously be

indicated as a surgical case at a critical NC. difference of 3.0 or 3.5 mA. during the *first week* after the onset of paralysis; at the same time, 4 patients out of 7 (about 60%) would be correctly submitted to surgical treatment during this period.
Statistical analysis*) of these data shows that a confidence interval for the probability p of a faulty decision in the group non-surgical cases (20) and the group surgical cases (7) is $0 < p < 0.27$ and $0.20 < p < 0.88$, respectively (level of significance 0.05).

If we adhere to a critical NC. difference of 3.0 - 3.5 mA. for the subsequent periods also, there would be 4 patients out of 25 (about 15%) who underwent unnecessary surgery during the *first two weeks,* whereas 8 patients out of 10 (about 80%) would rightly be submitted to an operation. The confidence interval for the probability p of a faulty decision in the two groups is then $0 < p < 0.37$ and $0.44 < p < 0.97$, respectively (level of significance 0.05). If from the onset we had accepted a NC. difference of 4 mA. as criterion, then only 2 patients out of 25 (about 8%) would have been erroneously submitted to operation during the first two weeks; however, 2 others would have been erroneously excluded from operation. With a NC. difference of 3.0 or 3.5 mA. as criterion, 6 patients out of 26 (about 25%) would have undergone an unnecessary operation during the *first three weeks;* if patient nr. 1 is regarded as an unmistakably faulty observation, then this is 5 out of 26 (about 20%). On the other hand, 12 out of 13 patients (about 90%) would have undergone a necessary procedure in the course of this period.

If surgical indications were determined exclusively on the basis of NC. determinations, one patient would be erroneously treated by operation during the first week (about 5%); through the first two weeks there would be 4 such patients (about 15%), and the total over the first 3 weeks would be 6 (about 25%). It was found that 4 patients were of the *complete** recovery category (with slight ultimate imperfections); the other 2 were *complete* cures without any residual symptoms. The total number of patients studied is too small to warrant further conclusions on the basis of these data.
If no patient is to undergo an unnecessary operation, a NC. difference

*) See footnote page 108.

$>$ 5.5 mA. must be accepted as criterion, but on that basis within the first week no patient out of 7 (0%) would undergo surgery, within the first 2 weeks 3 patients out of 10 (about 30%) and within 3 weeks 9 patients out of 13 (about 70%).
To elucidate this, the various results will be presented in a tabulated form (Table XXVI).

TABLE XXVI. SOME NC. DIFFERENCE LIMITS AND THE CONSEQUENT SURGICAL INDICATION IN 39 PATIENTS WITH BELL'S PALSY (26 CURED, 13 INCOMPLETELY RECOVERED) AT THE END OF THE 1ST, 2ND AND 3RD WEEK AFTER THE ONSET OF PARALYSIS.

NC. difference within	Faulty surgical indication for clinically cured patients			Correct surgical indication for incompletely cured patients		
	1 week	2 weeks	3 weeks	1 week	2 weeks	3 weeks
$\geqq$ 2.5 mA.	3(20)	6(25)	9(26)	4(7)	9(10)	12(13)
$\geqq$ 3.0 mA.	1(20)	4(25)	6(26)	4(7)	8(10)	12(13)
$\geqq$ 3.5 mA.	1(20)	4(25)	6(26)	4(7)	8(10)	12(13)
$\geqq$ 4.0 mA.	—(20)	2(25)	5(26)	3(7)	6(10)	11(13)
$>$ 5.5 mA.	—(20)	—(25)	—(26)	—(7)	3(10)	9(13)

In an attempt to establish a critical NC. difference which on the one hand indicates as many incompletely cured patients (unfavourable prognoses) as possible and, on the other hand, leaves an optimal number of spontaneous cures (favourable prognoses) undisturbed, the above described tests showed that NC. differences of 3.0 and 3.5 mA. are optimal (and to the same extent). As surgical indication, we would prefer a NC. difference $\geqq$ 3.5 mA. so as to ensure a minimum of unnecessarily performed operations, also in series of patients larger than ours.

The limited number of patients available for study, and the limited number of data per patient, also explains why a correct prediction could

not be made in all cases. Statistically, the nerve conduction differences described prove to be of undoubted value as surgical indication, but they are not exact as such in each individual case; this is obvious from the number of patients who would have undergone an unnecessary operation. Observations in duplicate would enhance the accuracy, as would determinations at more or less fixed dates after the onset of paralysis (because there is probably a correlation between the values determined and the course of time). For all this it is a conditio sine qua non that patients can be examined at the earliest possible date; this is decisive for the possibility of using nerve conduction as a prognostic aid.

Precise information such as that yielded by electromyography, cannot be expected from nerve conduction tests. The nerve is stimulated by a given current strength, and the magnitude of the minimum stimulus sufficient to cause excitation, is determined. The nerve as a whole is stimulated, and a muscle contraction follows when a sufficient number of nerve fibres responds. A seemingly normal behaviour of nerve and muscle, however, frequently masks the presence of telltale deviations (BAUWENS, 1961). Determination of threshold levels on the basis of visual examination only is another source of possible inaccuracies. This is demonstrated by the previously mentioned investigations of GILLATT & TAYLOR (1959) who found that, upon electrical stimulation of severely injured nerves, an electromyographic response persisted for 48-72 hours beyond the disappearance of the visible muscle twitch. In our opinion, however, it is certainly possible to perfect the technique of examination and improve diagnostic possibilities. In this respect we are referring to:

Improvement of the active electrode, e.g. by shaping it into a globe to fit the space below the osseous external auditory meatus, between the mandibular ramus and the mastoid process; this ensures its position immediately next to the stylomastoid foramen and closer to the facial nerve, also because the layer of soft parts between the electrode and the nerve is thinner at this site. In the majority of cases the facial nerve trunk proper is stimulated at this site, before it divides into its various rami. Another possibility is to use this site, not for percutaneous but for subcutaneous stimulation with needle electrodes perforating the

skin and passing to a point near the nerve trunk (MURPHEY); this eliminates the skin resistance factor.

Recording of the effect of stimulation by means of electromyography (so-called provoked electromyogram); in this way, nerve conduction testing can be combined with electromyographic investigation.

In addition to a 0.3 msec. stimulus, stimuli of shorter duration, e.g., 0.1 msec. This has been tried out in a number of patients; our preliminary impression is that a difference in nerve conduction measured with stimuli of 0.1 msec., is relatively greater than that measured with stimuli of 0.3 msec.. Deviations from the normal are more striking when stimuli of 0.1 msec. are used.

SUMMARY

Although nerve conduction testing can be considerably perfected, the relatively crude method we have used in patients with peripheral facial paralysis would nevertheless have revealed an unfavourable prognosis in 4 patients out of 7 within the first week, and in 8 patients out of 10 within the first two weeks. So far, this has not been feasible either by clinical investigation or with the aid of electromyography.

Like the chapters on "Clinical investigation" and "Electromyographic examination", this chapter should end in an attempt to establish whether (and, if so, to what extent) nerve conduction assessment can give information on the severity of the functional impairment of the facial nerve in individual cases. And again we do this on the basis of the same questions, viz.:

First question: *Is this paralysis transient (reversible nerve block) or is it a degenerative disorder of conduction so that recovery depends on regeneration of the nerve fibres affected?*

As long as the NC. difference between the affected and the unaffected facial nerve remains < 3.5 mA., it can be assumed that the paralysis is still transient (reversible). If the paralysed side shows absence of nerve conduction upon application of pulses up to 20 mA., then the condition can be regarded as irreversible and the nerve fibres as degenerated.

Second question: *If the paralysis is still in the reversible stage at the time of examination, are there any signs (if so, which?) heralding an imminent unfavourable development, i.e. irreversibility?*

A nerve conduction difference $\geqq 3.5$ mA. can be regarded — with some reserve — as a sign heralding an unfavourable course.

The practical conclusion at the end of this chapter is that, in peripheral facial paralysis, it *is possible* on the basis of nerve conduction assessments to select the majority of patients with an unfavourable or at least dubious prognosis within two weeks (and in a few cases within one week) of the onset of paralysis. Since nerve conduction assessment can reveal signs of an imminent or incipient unfavourable course at an early time (provided the patient is examined soon after the onset of paralysis), it seems possible to influence this course by taking suitable measures.

DISCUSSION

In the introduction to this study it has been stated that, at the present state of knowledge, it seemed impossible in the vital early stages of facial paralysis to distinguish such patients as are likely to have an unfavourable or at least a dubious prognosis. It appeared that the decision as to the therapeutic approach to be adopted, would be greatly facilitated if some reliable data could be collected, on the basis of which the severity of the functional disorder of the nerve in each *individual* case could be assessed at a time sufficiently early to permit of measures counteracting a possibly imminent serious course. In search of such data, we have considered in turn the findings obtained by clinical investigation and those obtained with the aid of special electrical tests, notably electromyography and assessment of nerve conduction. So as to ensure both sufficient and uniform data, this problem was studied in patients suffering from facial paralysis of the type known as Bell's palsy.

The data considered in the chapter on *clinical investigation* were the extent of the paralysis, muscle tone, aural pain, taste sensation, hyperacusis, lacrimation and the nasopalpebral reflex. Of these, only the extent of paralysis, muscle tone, and presence or absence of the nasopalpebral reflex appeared to be of practical value in determining the prognosis of the affection. The most reliable datum is the extent of the paralysis; the muscle tone and the nasopalpebral reflex are important if they correspond with, and thus support, the prognostic conclusions based on the extent of the paralysis. A study of these data revealed that, during the first two weeks after its onset, a paralysis is still capable of considerable changes; partial paralyses may become complete, and complete paralyses may recede to partial. At the end of this two-week period, however, the severity and the course of the disorder are as a rule well established. This is demonstrated by the fact that patients who show complete paralysis at the end of the

first week, still have about 50% chance of full recovery, whereas this chance is almost zero when the paralysis is still complete at the end of the second week. Patients showing partial paralysis at the end of the second week have about 85% chance of making a full recovery. For patients showing partial paralysis from the onset, good muscle tone and persistence of the nasopalpebral reflex, the chance of a complete cure is virtually 100%.
It seems possible, therefore, to reach a general prognostic prediction on the basis of clinical investigation. But unfortunately, this cannot be done before the end of the second week after the onset of paralysis, and even then it is impossible, in cases of partial paralysis, to select patients likely to make only an incomplete recovery. A general prognosis of this type is therefore of little practical value in the appraisal and treatment of the *individual* patient, at least not at a time sufficiently early to permit of measures to ward off an unfavourable turn of events.

Electromyography affords precise information on neuromuscular disorders in peripheral nerve injuries. This method makes it possible to divide clinically indistinguishable paralyses into reversible, physiological interruptions of nerve conduction, and more severe affections of the nerve, associated with axon degeneration. In cases of the latter category, electromyography reveals so-called "fibrillations" — rhythmically repeated potential discharges resulting from spontaneous contractions of single muscle fibres. These fibrillations are considered pathognomonic of denervated muscle fibres. Although electromyographic findings permit of assessment of the functional condition of the facial nerve in individual cases of facial paralysis, and therefore of selection of patients with a serious prognosis, this cannot be done until 10-14 days after the onset of paralysis, as fibrillation potentials cannot be recorded earlier. At that time, however, an imperfect recovery can no longer be prevented because the phenomenon which marks these patients — fibrillation — is as such a sign characteristic of axon degeneration, against which current medical and technical methods fail. Degeneration, however, need not be an all-or-none phenomenon; if the first fibrillations are regarded as an indication of a severe affection of the nerve, and if measures are taken accordingly, then a proportion of the nerve fibres can probably still be saved from degeneration, or at least regeneration of affected axons can be promoted.

Further information on the prognosis of peripheral facial paralysis in the

individual patient can be obtained by *assessment of the nerve conduction.* This electrical test indicates the threshold stimulation intensity of the facial nerve for minimal muscle contraction. Changes in the conductivity of the nerve are manifested by differences in stimulation values between the affected and the contralateral side of the face. Data so far obtained warrant the conclusion that absence of conductivity upon excitation of the affected nerve with current strengths up to 20 mA. implies degeneration of the nerve fibres distal to the site of the lesion. In severe (traumatic) nerve injuries, this may be the case as early as on the fourth day after infliction of the injury. On the other hand, as long as NC. differences do not amount up to 3.5 mA., it can be assumed that the disorder arises from transient (reversible) conduction block, and a complete cure can be expected. If the NC. difference between the affected and the contralateral facial nerve is 3.5 mA. or more, this should be interpreted as a warning against an imminent unfavourable course. On the basis of the above criteria of nerve conduction, it was found possible to select the majority of patients with facial paralysis likely to have an unfavourable prognosis within 2 weeks, and a by no means negligible number of such patients within 1 week of the onset of paralysis. It seems possible — at least when one acts immediately in response to the first indication of an incipient or imminent unfavourable course — to influence the course of facial paralysis.

It can be stated in summary that, if a given patient with facial paralysis can be studied from the onset, the above discussed data warrant the following conclusions with regard to prognosis:
A complete cure can be expected if the paralysis is *partial,* with good muscle tone and intact nasopalpebral reflex, and if subsequent days show no aggravation, but perhaps even improvement. Electromyography provides a detailed survey of the extent of the nerve disfunction but affords no further prognostic data. Nerve conduction assessment in this case indicates no abnormal difference between the affected and the intact facial nerve.
If an initially partial paralysis subsequently becomes *complete* paralysis, or if it is complete from the onset, no prognostic information can be obtained from clinical findings. Electromyography may help in these cases in that this method of precision may show that some muscle activity is still possible, in which case there is an indication that at least some nerve fibres are still functioning; this is a favourable prognostic sign. Nerve conduction determ-

inations can be decisive in these cases; as long as the NC. difference between the paralysed and the contralateral nerve remains under 3.5 mA., it can be assumed that the nerve affection is reversible, and that complete recovery is among the possibilities.
If the paralysis remains clinically complete and electromyographically subtotal or complete, and if the NC. difference increases to 3.5 mA. or more (verified by duplicate determinations or repeated investigation), then there is a strong indication of imminent unfavourable development. An unfavourable development is confirmed when the NC. difference shows a further increase (rapidly or more gradually, dependent on the severity of the disorder). If at a given moment there is no longer any response even at a stimulus strength of 20 mA., then we know — empirically — that the nerve fibres have degenerated. If this process is rapidly completed, then nerve conduction is absent before electromyographic signs of degeneration (fibrillations) are demonstrable; after all, the earliest time at which these fibrillations are found was, as has been pointed out, the 11th day. If progression is of the more gradual type, then fibrillations may be found before nerve conduction is entirely absent. Fibrillation, like the absense of a response at a stimulus strength of 20 mA. or more, indicates an established fact: degeneration of nerve fibres exists.
In practice, of course, a whole range of variations on this theme can be encountered. We have found, that only the measurable and comparable quantitative data afforded by assessments of nerve conduction make it possible to follow a paralysis step by step from the onset, thus to ensure early warning of an imminent or incipient unfavourable course.

The object of our investigation — to find a method of selecting such patients with peripheral facial paralysis as are unlikely to make a spontaneous and complete recovery, in the vital early stages of the paralysis — seems to have been attained. However, we have repeatedly expressed our conviction that, if suitable measures are taken at the first alarming sign, it is probably possible to counteract an imminent unfavourable course of the paralysis, and to prevent nerve degeneration. Consequently we feel we must not conclude this study without discussing our therapeutic point of view.
As has been pointed out in the introduction to this thesis, there is no fixed rule of approach in the treatment of facial paralysis from causes as mentioned in group III (Bell's palsy, acute otitis media, external otitis, paralysis *of delayed onset* after operations on the temporal bone, fractures of

the temporal bone and external violence to the middle ear and the mastoid bone). The treatment of choice varies in accordance with the therapist's personal views and experience. Some advocate conservative treatment exclusively; others prefer early surgical intervention. Most investigators, however, start with conservative measures but switch to surgery when insufficient or no improvement is seen.

The lastmentioned approach is the one accepted in the Amsterdam University Ear-, Nose- and Throat Department (Head Prof. Dr L. B. W. Jongkees). We are advocates of conservative therapy whenever and as long as we believe there is a possibility of spontaneous and complete recovery. If we believe there is no longer such a possibility, or if conservative measures show insufficient or no effect, we resort to surgical exploration of the affected facial nerve. The reasons why we believe that success can be expected from surgery (particularly from decompression of the nerve in Bell's palsy) need not be discussed here because they are not within the scope of this study. The thinking is on the lines of preventing axon degeneration in order to ensure better functional restoration. Detailed considerations on these questions can be found in the various papers by JONGKEES and in BOTMAN's thesis (1954). Our views in this respect agree with those of CAWTHORNE, COLLIER, DUEL, FOWLER, KETTEL, MARTIN, MIEHLKE, VON SCHULTHESS, SOKCIC, SULLIVAN, TICKLE and many others.

There is still no consensus of opinion as to the criteria for changing the initial therapeutic approach, and the time at which surgery should be resorted to. This disagreement is largely a result of the lack of methods of investigation generally accepted as reliable, to assess the severity of the nerve affection and, therefore, the prognosis of the paralysis. Consequently, treatment is of necessity based on clinical experience and emperical indications. In Bell's palsy — to take the most important aetiology as example — it has so far been the general point of view that the facial nerve should be decompressed if insufficient or no improvement in facial nerve function is seen within 6-8 weeks of the onset of paralysis. Although undoubtedly this has a very favourable effect on the course, no investigator has so far been able to give his patients a complete cure by an operation at this time. This is freely admitted by KETTEL (1959) who, in his monograph, states: "In all cases synkineses, ranging from extremely slight to rather pronounced were noted". This is entirely understandable, for the indication for surgical intervention is based on absence of improvement at a time when this would

normally have started already; at a time, therefore, when the therapist is confronted with an established fact. It is generally agreed, therefore, that an operation should be performed at an earlier time but, as KETTEL says it: "To find sound indications for surgical intervention is as important as it is difficult".

Considering whether the data evaluated in this study may lead to a surgical indication at an earlier time, the following can be said:

Clinical investigation has shown that, for patients showing complete paralysis at the end of the second week after its onset, the chance of full recovery is virtually zero. Although we have found that clinical findings warrant no individual but only a general prognosis, this general prognosis is so definite for the abovementioned patients that we believe ourselves justified in using it as a basis for surgical indication. Patients showing complete paralysis on the 14th day after its onset, therefore, should in our opinion be submitted to an operation at this time, on the basis of the clinical findings.

Electromyographic examination (unlike clinical investigation) affords information on the severity of the nerve disorder in the *individual* patient. The first sign indicating an unfavourable course, however, does not occur until 10-14 days after the onset of paralysis. This first sign consists of fibrillation, and this indicates axon degeneration. Recovery of these patients, therefore, cannot be complete even when an operation is performed at the first alarming sign (fibrillation); residual symptoms must be expected. This is clearly demonstrated by our findings as presented in Table XVI. However, although we agree with KETTEL that "electromyography is useless as an emergency guide", it nevertheless seems possible to save the nerve fibres, at that time still capable of recovery, by an immediate operation; after all, degeneration need not be in all-or-none phenomenon. The requirement set by JAMES & RUSSELL (1951) — "If decompression really helps, it should be done within two weeks of the onset, when there is first evidence of nerve degeneration" — can therefore be met, at least in a number of cases. Immediate decompression as soon as fibrillation voltages occur has also been advocated by NAKATA (Tokyo, 1958) and recently by NEIGER (1961).

Assessment of nerve conduction is the method of investigation so far found to give the best and earliest information on the severity of the nerve disorder in the individual patient. If, as has been discussed in detail, a

NC. difference of 3.5 mA. or more between the affected and the unaffected nerve is regarded as an alarming sign — and therefore as an indication for operation — then it seems possible to operate in the majority of cases of Bell's palsy likely to have a serious prognosis within 2 weeks of the onset of paralysis (about 80%), and in a by no means negligible number (about 50%) even before the end of the first week.

It appears, therefore, that surgery on the basis of nerve conduction assessment affords the best chance of succesful counteraction against an imminent unfavourable course, and of preventing neuraxon degeneration.
In the discussion of nerve conduction assessment, however, is has not been concealed that — operating on the indication of a NC. difference of 3.5 mA. or more — a number of patients can be included who are in fact not suitable subjects (as shown in retrospect by their spontaneous recovery). In our group of patients with Bell's palsy, their number would have been 1 patient out of 20 (about 5%) over the first week after onset of paralysis, and 4 out of 25 (about 15%) over the first two weeks after onset. It is generally agreed that about 80% of patients with Bell's palsy makes a spontaneous and full recovery (post or propter conservative therapy). There is also virtually general agreement with the statement of PARK & WATKINS (1949) that: "If decompression were done within a few hours after the onset of facial weakness, it would be theoretically possible that in no case would a complete reaction of degeneration develop". At the same time, however, most investigators agree with TUMARKIN (1936) when he states: "How dare we advise operation when we know that any given patient has a four to one chance of spontaneous recovery". Very rightly nobody has had the regrettable audacity of resorting to immediate operation in all his patients, in which case the rate of injustified surgery would have been about 80%. On the other hand, however, it requires almost superhuman self-control not to resort to an operation in cases followed up from the onset without ever seeing any sign of improvement of the paralysis (and intuitively regarded as almost certain failures), simply because there is no 100% established surgical indication. Many a surgeon would gladly accept the risk (of course restricted to a minimum) of a number of unnecessary operations if this would be counterbalanced by the knowledge that a surgical cure could be obtained in the majority (if not all) of the patients whose recovery would otherwise probably be incomplete. The experienced ear-surgeon does not agree with the frequently

made objection that a major surgical procedure is involved, which entails grave risks for the patient or for the facial nerve; this does not constitute an argument against operation in this instance. To the patient, the operation is a minor nuisance which entails only a few days in hospital and no or hardly any aftercare.

In our group of Bell's palsy patients the rate of unjustifiable operations (according to the above considerations) was found to be 1 : 20 (about 5%) over the first week, and 4 : 25 (about 15%) over the first two weeks after the onset of paralysis. As has been pointed out, it should be emphasized that these figures hold true only if we act exclusively (blindly, so to speak) on the outcome of nerve conduction assessments. Nobody would attempt to regard electrical findings of any kind as detached from clinical observations, however. These technical methods are of value in the management of peripheral nerve injuries *only* if correlated with a full-scale clinical investigation (BOWDEN). This also holds true for facial paralysis. The ultimate object is to attain complete or at least optimal restoration of the function of the facial muscles. Whenever a paralysis is confined to mild paresis or has shown unmistakable signs of spontaneous improvement, precipitate surgical intervention is avoided even when some single electrical test result would point in this direction. In this respect, mention has been made of the necessity of duplicate determinations and repeated investigation. An entirely different situation exists, however, when an increased NC. difference between the affected and the unaffected nerve is observed in patients with severe or total clinical paralysis. If an expectant attitude is taken in the face of such a correlation between clinical and electrical findings, then the chance of a favourable outcome will soon be reduced to zero.

After these remarks, let us once again consider — on the basis of Table XXIV — all patients who should have been submitted to operation in accordance with the critical NC. difference of 3.5 mA. Patients numbers 27 through 39, who made an incomplete ultimate recovery, all showed subtotal or total paralysis at the time of determination of the abovementioned NC. difference. Subsequent NC. determinations also demonstrated a difference of more than 3.5 mA. in all cases; the majority of patients showed a progressive increase in this difference. In all these patients, therefore, an unmistakable correlation between clinical features and the results of nerve conduction assessment existed.

Of patients numbers 1 through 26, 4 would have been incorrectly indicated for surgery within the first 2 weeks, on the basis of the critical NC. difference alone. However, one of these (patient no. 1) would have been eliminated at repeated investigation which, like all subsequent assessments, would have revealed a normal NC. difference. A second patient (no. 18) was found to be virtually cured at the time when an increased NC. difference was found; he would have been excluded from surgery on clinical grounds. Patients numbers 5 and 26, who still showed a NC. difference of more than 3.5 mA. in the course of the third week, would also have been excluded on clinical grounds, because at that time patient no. 26 showed only a moderate remnant of paralysis, while paralysis was no longer visible at all in patient no. 5.

According to this procedure, on the basis of clinical investigation supplemented by nerve conduction assessments, 2 of our group of 39 patients would have undergone an unjustified operation. This is about 5-10% of the patients who made an ultimate clinical recovery. Comparing this figure with the large number of patients (about 80%) who made an incomplete ultimate recovery and who, by the same procedure, could have been selected for operation within 1 and 2 weeks of the onset of paralysis, we find that a risk of this kind should be considered acceptable.

It seems useful to present a few case histories to illustrate our line of thought.

BELL'S PALSY

Mr B., age 28, felt a stabbing pain behind the *left* ear on March 29th, 1960. On March 31st, waking up, he noticed facial hemiparesis and a foul metallic taste in the mouth. He felt completely fit otherwise. There was no history of illness, common cold, ear symptoms or emotional upset. We first saw the patient on 2nd April 1960. He reported aggravation of symptoms since the onset; the face was more flaccid and eating and drinking was difficult; lacrimation had increased; there was no dysacousia.
Physical examination. In repose, the forehead was smoother on the left side; eyebrow L. ↓; palpebral fissure L>R and filled with lacrimal fluid; nasolabial fold L=R; philtrum slightly → R; line of the mouth straight. Upon maximal effort, all muscles were still capable of some contraction. The nasopalpebral reflex was absent on the left. The left anterior part of the tongue had lost the sense of taste for sweet, salt and bitter. Further neurological, otological,

general physical and laboratory findings were normal. Mastoid X-rays showed no anomaly. The condition was diagnosed by elimination as acute peripheral facial paralysis (left sided) of the Bell's palsy type. Assessment of nerve conduction: *NC. difference* L/R: -2.2 mA. *Electromyographic examination:* pattern of single motor unit potentials in all muscles.
The patient was treated by the routine procedure used in our clinic for acute Bell's palsy, i.e. by bed rest, application of thermogenic pads to the left mastoid region, 100 mg. nicotinic acid 4 times daily for 1 week, and daily left-sided stellate ganglion block on 5 consecutive days. The patient was instructed as to active exercise of the paretic muscles.
April 4th: complete paralysis despite treatment instituted; *NC. difference* L/R: -0.8 mA.
April 6th: slight functional restoration in corr. supercilii muscle; *NC. difference* L/R: 3.5 mA. *Electromyogram:* pattern of single motor unit potentials in all muscles; no denervation potentials.
April 8th: slight functional restoration in orbicularis oculi muscle; *NC. difference* L/R: 0.5 mA.
April 11th: no obvious objective progress; subjectively, less bothered by difficulties of eating and drinking and lacrimation of the left eye; *NC. difference* L/R: 1.2 mA. *Electromyogram:* no denervation potentials.
April 15th: unmistakable functional improvement in all muscles; *NC. difference* L/R: 0.3 mA. *Electromyogram:* no denervation potentials; mixed pattern in all muscles.
April 22nd: marked functional improvement in all muscles; *NC. difference* L/R: 0.6 mA.
May 10th: slight residual paresis on the left; *Electromyogram:* interference pattern in all muscles; no denervation potentials.
One-year follow-up April 27th, 1961: complete subjective and objective recovery.

This patient, who showed complete paralysis on the 5th day after onset of symptoms, ultimately made a full recovery. The electromyographic examination (no denervation potentials) and the nerve conduction assessment (NC. difference L/R within normal limits) are in accordance with this (the abnormal NC. value on the 7th day appears to be based on faulty registration, as all subsequent determinations yielded normal control values). *N.B.* This is the same patient who was discussed on page 118 with reference to the NC. difference which did not fit the clinical features.

Mrs SB., age 58, noticed her messy drinking during the evening meal on 8th April 1960; her companions noticed that her face was slightly drooping on the *left*. She felt completely fit and had no history of a cold, infectious diseases or fever, unusual emotions or ear symptoms; 3 weeks previously she had an eczema-like affection on both arms for a few days. We saw this patient on 12th April 1960. The paresis had subjectively increased; patient had difficulties speaking, eating and drinking; the left eye was constantly lacrimating

and she had an insipid taste in the mouth; no pain in the mastoid region; no dysacousia.

Physical examination. In repose, the face was slightly asymmetrical; forehead L=R; palpebral fissure L>R; nasopalpebral fold effaced on the L; philtrum →R; corners of the mouth L=R. Upon maximal effort, no muscular contraction was seen. The L. nasopalpebral reflex was absent; the left anterior part of the tongue did not taste sweet, salt and bitter. Further neurological, otological, general physical and laboratory findings were normal. Mastoid X-rays showed normal pneumatization on both sides. The condition was diagnosed by elimination as acute peripheral facial paralysis on the left side, of Bell's palsy type. Assessment of nerve conduction: *NC. difference* L/R: 0.3 mA. *Electromyographic examination:* complete electrical silence at rest as well as on maximal contraction effort.

The patient was treated by bed rest, application of thermogenic pads to the L. mastoid region, 100 mg. nicotinic acid 4 times daily for 1 week, and daily left-sided stellate ganglion block on 5 consecutive days.

April 16th: complete paralysis; *NC. difference* L/R: 1.5 mA.; *Electromyogram* unchanged since April 12th.

April 18th: no subjective improvement; slight objective functional restoration in corrugator supercilii and orbic. oculi muscles; *NC. difference* L/R: 0.5 mA.

April 22nd: at maximal effort, movements are possible in all muscles; *NC. difference* L/R: 1.8 mA.; *Electromyogram:* pattern of single motor unit potentials in all muscles; no denervation potentials.

April 25th: further increase in muscular function; weak positive nasopalpebral reflex on the left.

April 29th: very good improvement; sense of taste restored to anterior left half of tongue; *NC. difference* L/R 2.9 mA.; *Electromyogram:* mixed pattern in orbic. oculi and quadratus labii sup. muscles.

May 11th: functional restoration almost complete, except for quadratus labii inf. muscle; *NC. difference* L/R: 0.4 mA.; *Electromyogram:* some fibrillations in quadratus labii sup. and inf. muscles.

July 10th: face symmetrical; still slight paresis of quadratus labii inf. muscle; *NC. difference* L/R: 2.5 mA.

One-year follow-up April 28th, 1961: complete subjective recovery: nobody notices anyting abnormal. Objectively, still some very slight paresis of the quadratus labii inf. muscle and slight synkinesis (narrowing of the left eye as the teeth are bared).

This patient, who showed complete paralysis on the 5th day after onset of symptoms, made a clinically complete* recovery. Careful observation reveals a few minor residual symptoms; the electromyographic findings (some fibrillations) were in accordance with this. The NC. difference L/R remained within normal limits.

Mr Gs., age 23, noticed a foul taste in the mouth on 30th May 1960. On the evening of the same day he noticed a stiff feeling in the *right* half of the face, drooping of the mouth and lacrimation in the right eye. He went to see the family doctor on the next morning and was immediately referred to us. We

therefore saw this patient on 31st May; apart from the facial paresis he felt completely fit. The past history comprised no common cold, emotional upsets, infections (particularly no ear affections) or any other abnormalities. There was no pain behind the ear. Speech, eating and drinking were impeded and the patient described a taste in the mouth as after dental filling; lacrimation in the right eye was troublesome; no dysacousia. *Physical examination.* Inspection of the face showed that the right side of the forehead was smooth; eyebrow R. ↓; palpebral fissure R>L and filled with lacrimal fluid; nasolabial fold R=L; philtrum →L; corner of the mouth R.↓. At maximal effort only the frontal muscle showed some contraction. Absence of the R. nasopalpebral reflex; the right anterior part of the tongue was insensitive to sweet, salt and bitter tastes. Further neurological, general physical, otological and laboratory findings were normal, as were the mastoid X-rays. The condition was diagnosed by elimination as right-sided subtotal acute peripheral facial paralysis, Bell's palsy type.

Assessment of nerve conduction: *NC. difference* R/L: -1.5 mA. *Electromyographic examination:* pattern of single motor unit potentials in the frontal, quadratus labii sup. and inf. muscles.

The patient was treated by bed rest, application of thermogenic pads to the R. mastoid region, infra-red irradiation of the R. mastoid region 4 times daily, 100 mg. nicotinic acid 4 times daily for 1 week and daily right-sided stellate ganglion block for 5 consecutive days. He was instructed to carry out active exercises with the still functioning muscles.

June 2nd: complete paralysis despite treatment instituted: *NC. difference* R/L: -0.9 mA.

June 4th: condition unchanged; *NC. difference* R/L: 4.6 mA.

June 7th: paralysis remains total; face entirely atonic; *NC. difference* R/L: 4.6 mA. *Electromyogram:* after thorough probing of the muscles, a single train of motor unit action potentials only in the frontal and the orbic. oculi muscles at maximal effort of contraction.

June 10th: no clinical change; *NC. difference* R/L: 4.7 mA. *Electromyogram:* no residual activity; no denervation potentials.

June 14th: no clinical change; *NC. difference* R/L 7.2 mA. *Electromyogram:* electrical silence at maximal contraction effort; scanty sustained fibrillation in all muscles examined.

June 16th: decompression of the facial nerve in view of clinically and electromyographically complete paralysis and denervation potentials in all muscles examined. At operation, the nerve was found to show, from the genu externum to the bifurcation of the chorda tympani, the oedematous swollen features typical of Bell's palsy. Postoperative treatment included infra-red irradiation of the right half of the face and massage of the facial muscles, carried out daily by the patient himself.

July 22nd: subjective improvement. Slight objective functional restoration in frontal, corrugator supercilii and quadratus labii sup. muscles. Therapy supplemented by active exercise of the muscles.

August 24th: further improvement; contraction also in orbic. oculi muscle. Weak positive nasopalpebral reflex on the right.

September 28th: further improvement, but incipient synkinesis and contracture; still considerable lacrimation.

One-year follow-up June 25th, 1961: at rest, asymmetry due to marked con-

tracture (nasolabial fold deeper on the right: right angle of the mouth elevated, palpebral fissure smaller on the right); marked synkinesis, active muscle function gravely restricted.

This patient, who showed complete facial paralysis on the 3rd day after onset of symptoms, ultimately made an incomplete recovery with relatively severe residual symptoms. Electromyographic examination (fibrillation in all muscles) and the nerve conduction test (NC. difference R/L > 3.5 mA. from the 5th day after onset) are in accordance with this.

Mr v. H., age 31, noticed a tedious pain behind the *right* ear when in bed on the evening of 8th May 1960. Shaving on the following morning, he noticed a strange sensation in the right cheek; breakfast was normal. His colleagues at the office noticed that his face was drooping. On the same day this patient saw his family doctor, who referred him to us. We saw the patient on May 10th; apart from the paretic face, the patient felt completely fit. The past history revealed no cold, emotional upsets, infections (particularly ear affections), fever, dizziness or any other symptoms or events which might explain this sudden development. The patient stated that, in his opinion, the condition had obviously exacerbated since the onset; the stabbing behind the right ear had not diminished; the patient found it difficult to pronounce labial letters; food was tasteless but eating or drinking offered no difficulty; the lacrimation in the right eye, however, was troublesome; there was no dysacousia.
Physical examination: Inspection of the face showed an effacement of the folds on the right side of the forehead; eyebrow R ↓; palpebral fissure R=L; nasolabial fold R<L; philtrum →L; right corner of the mouth ↓. At maximal effort there was still some contraction in the frontal, corrugator supercilii, orbic.oculi and the caput angulare of the quadratus labii sup. muscles. The right-sided nasapalpebral reflex was absent. The right anterior part of the tongue was insensitive to sweet, salt and bitter tastes. Further neurological, otological, general physical and laboratory findings were normal, as were the mastoid X-rays. The condition was diagnosed by elimination as acute peripheral facial paralysis on the right, of the Bell's palsy type. Assessment of nerve conduction: *NC. difference* R/L: 0.7 mA.
The patient was treated by bedrest, application of thermogenic pads to the right mastoid region, infra-red irradiation of the right mastoid region 4 times daily, and 100 mg. nicotinic acid 4 times daily for 1 week, with daily right-sided stellate ganglion block for 5 consecutive days. The patient was instructed in active exercising of such muscles as were capable of this.
May 12th: complete paralysis despite therapy instituted (surprisingly, the patient himself believed he noticed improvement).
May 15th: clinically complete paralysis; *NC. difference* R/L: 3.9 mA.
May 18th: very slight functional restoration in the frontal and corrugator supercilii muscles; no other change; face still entirely flaccid; persistent stabbing behind the ear.
May 21st: no further functional restoration; slight contraction in frontal and corrugator supercilii muscles. *Assessment of nerve conduction:* loss of nerve conduction at stimuli of up to 20 mA. *Electromyogram:* pattern of single motor

unit potentials in the frontal muscle. Scanty fibrillations in the frontal, orbic.oculi, quadratus labii sup. and inf. muscles.
May 22nd: decompression of the facial nerve in view of subtotal paralysis and degenerative symptoms in all muscles examined. At operation the nerve was found to show the typical oedematous swollen aspect characteristic of Bell's palsy.
May 30th: condition unchanged; slight contraction in frontal and corrugator supercilii muscles; patient is actively exercising these muscles; the other still paretic muscles are given electrical treatment 3 times a week.
June 7th: increased function in frontal and corr. supercilii muscles; incipient functional restoration in orbic. oculi and quadratus labii sup. muscles.
June 21st: further increase in muscular function; slight contraction also in the zygomatic muscle.
July 12th: further improvement; palpation of quadratus labii inf. muscle shows palpable contraction.
August 30th: all muscles resorted to function; nasopalpebral reflex on the right weak positive; slight synkinesis visible.
One-year follow-up May 15th, 1961: almost complete facial symmetry at rest, partly because of slight contracture in the nasolabial fold. Unmistakable synkinesis, which is not very disturbing unless vivid facial expressions are produced; active muscle function definitely limited. Result is good in the circumstances, and the patient himself is satisfied.

This patient, who showed total facial paralysis on the 4th day after onset of symptoms, made an incomplete recovery with unmistakable residual symptoms. This is in accordance with electromyographic (fibrillations in all muscles examined) and nerve conduction findings (NC. difference R/L > 3.5 mA. from the 7th day after the onset).

Mr M., age 50, felt a vague stabbing pain behind the *right* ear on the night of 10th October 1960. On 12th October, brushing his teeth in the morning, he noticed that the cheek was flaccid; at breakfast he had difficulty drinking. He felt fit and had no history of infections, diseases or emotional upsets. However, he had been very cold when waiting for somebody on a station platform on 9th October. Patient was treated with vitamin B1 and salicylic acid powders but was referred to us when the paresis failed to improve. We first saw this patient on 24th October 1960. The paralysis had shown considerable subjective exacerbation; speech, eating and drinking were impeded, and the right eye was constantly lacrimating. No change in the sense of taste had been noticed; there was no dysacousia; the pain in the right mastoid region had disappeared the day before we saw the patient.
Physical examination. The right half of the face was completely atonic at rest. The right side of the forehead was smooth; right eyebrow ↓; right palpebral fissure wide and full of lacrimal fluid; right nasolabial fold effaced; philtrum →L; right corner of the mouth ↓. At maximal effort there was no contraction of muscles. The right anterior part of the tongue was insensitive to salt, sweet and bitter. Further neurological, otological, general physical and laboratory findings were normal. X-rays of the mastoid bones were likewise normal. The

condition was diagnosed by elimination as peripheral facial paralysis of the Bell's palsy type.

Electromyographic examination: electrical silence in all muscles at maximal contraction effort. At rest, a few insertion fibrillations were found in the quadratus labii sup. muscle, but there was no sustained fibrillation. *Assessment of nerve conduction:* loss of nerve conduction at a stimulus strength of up to 20 mA.

October 26th: condition unchanged; *Loss of nerve conduction* at 20 mA. pulses; *Electromyogram:* complete paralysis of all muscles examined; scanty sustained fibrillations in the quadratus labii sup. and inf. muscles. On the basis of the clinically and electromyographically total paralysis, and the first signs of axon degeneration in the electromyogram, surgical intervention was resorted to.

October 28th: decompression operation. The nerve showed oedematous swelling from the stylomastoid foramen to just before the genu externum; aspect of the nerve typical of Bell's palsy. Upon consultation with the neurologist, the patient was given electrical treatment every day after the operation.

November 30th: condition unchanged; no visible muscle function yet.

December 28th: no functional restoration yet; electrical stimulation continued.

February 18th, 1961: slight functional restoration in the orbic.oculi and quadratus labii sup. muscles; some synkinesis of the corner of the mouth upon blinking.

March 19th: unmistakable but limited function in all muscles except the frontal muscle. Synkinesis. Mild contrature in the right nasolabial fold. Electrotherapy discontinued and replaced by active exercises.

One-year follow-up October 15th, 1961: at rest, reasonable symmetry of the face, particularly because of contracture in the nasolabial fold. Synkinesis, not very disturbing when the facial expression is quiet. Active muscle function greatly impaired.

This patient, who showed total paralysis on the 13th day after onset of symptoms, made an incomplete recovery with unmistakable residual symptoms. This is in accordance with the electromyogram, in which denervation potentials were found, and the nerve conduction test which showed that the paralysed nerve did not respond to stimuli of up to 20 mA. current strength.

SURGICAL PALSY

Mrs VB., age 57, underwent surgical stapedolysis on 16th February 1960 (*right* side). At operation it was found that the horizontal portion of the facial nerve canal partly obstructed vision on the oval window. In an attempt to improve access to the oval window, the facial nerve canal was accidentally opened; afterwards, a few fragments of tissue were visible at this site. With the disturbing thought of probably having injured the facial nerve, the surgeon terminated the operation. Postoperatively, the facial nerve function proved to be undisturbed, and patient was discharged on February 22nd. The right half of the face was entirely paretic when she woke up on the following morning.

We saw this patient on 29th February 1960. She reported some subjective improvement. She felt completely fit but, since the onset of paralysis, had felt stabbing pain in and behind the right ear. Speaking, eating and drinking were difficult; she had a sweet taste in the mouth and was much troubled by lacrimation. The past history revealed no possible causes of the paralysis other than the ear operation.

Physical examination. Completely asymmetrical face and flaccid right half. No contraction at maximal effort. Absence of the nasopalpebral reflex on the right; the right anterior part of the tongue was insensitive to sweet, salt and bitter. General physical, otological and neurological findings, and laboratory data, were normal except for otosclerosis, the surgical scar and the facial paralysis. X-rays of the temporal bone by the VAN DEN BREKEL technique revealed no distinct defect in the facial nerve canal. The condition was diagnosed as acute peripheral facial paralysis of delayed onset after surgery of the middle ear.

Assessment of nerve conduction: loss of nerve conduction at stimulus strengths of up to 20 mA. *Electromyographic examination:* electrical silence in all muscles despite maximal contraction effort. No denervation potentials.

In view of the total loss of function, probably as a result of surgical trauma, and after consultation with the surgeon, who urged this, surgical exploration of the facial nerve was resorted to.

March 1st: surgical inspection of the facial nerve. There was a small osseous defect in the bony facial nerve canal above the oval window; the surface of the nerve was roughened at this site but it was hardly swollen. The further course of the nerve in the mastoid showed nothing unusual. The nerve sheath was split, and the nerve was covered with a piece of amniotic membrane.

March 8th: condition unchanged. *Loss of nerve conduction* at 20 mA. pulses. *Electromyogram:* no sustained fibrillations. Upon consultation with the neurologist, the patient was given electrical treatment daily.

March 30th: still complete paralysis. *Loss of nerve conduction* at 20 mA. pulses. *Electromyogram:* complete paralysis and sustained fibrillations in all muscles examined.

May 2nd: clinically complete paralysis. *Electromyogram:* one train of single motor unit potentials in the orbic.oculi muscle.

June 13th: unmistakable but limited function in the frontal, orbicularis oculi, zygomatic and quadratus labii sup. muscles. Positive nasopalpebral reflex on the right. Electrotherapy discontinued and replaced by active muscle exercises.

August 22nd: unmistakable function in all muscles, some of which showed almost normal contraction. However, also generalized synkinesis and unmistakable contracture formation.

One-year follow-up April 27th, 1961: at rest, some facial asymmetry; normal quiet facial expressions hardly disturbed; synkinesis and contracture mild and not very disturbing. Active muscle function right and left virtually equal. Subjectively, the patient is satisfied. We consider this an incomplete recovery but, in the circumstances, a good result.

This patient, who developed a facial paralysis 7 days after an ear-operation (which paralysis was found to be complete 7 days after the onset),

made an incomplete recovery with unmistakable residual symptoms, although of limited severity. The preoperative nerve conduction assessment (loss of nerve conduction at 20 mA. pulses) and the postoperative electromyographic findings (denervation potentials) are in accordance with this.

Mr V., age 48, on July 7th, 1960, underwent an ear operation because of exostosis in the *right* external auditory meatus. During the first few postoperative days he constantly felt some pain in the right ear; the first change of dressings was made on July 15th. On July 16th the patient's wife noticed that "something was wrong" with the right eye. On July 17th slight facial paresis was observed which gradually increased and became complete paralysis. The patient was not febrile and felt fit apart from a stabbing pain in the right ear.

We first saw the patient on July 22nd, on the 15th postoperative day and the 5th day after onset of paralysis. Speech, eating and drinking were impeded. The sense of taste was undisturbed. The right eye showed mild lacrimation, particularly at reading. Apart from the ear operation, the history revealed nothing which could explain the paralysis.

Physical examination. The right half of the forehead was smooth and unlined; right eyebrow ↓; palpebral fissure R=L; nasolabial fold R<L; philtrum →L; right corner of the mouth ↓. At maximal effort, slight narrowing of the lateral angle of the eye; the right-sided nasopalpebral reflex was negative. The anterior part of the tongue was sensitive to sweet, salt and bitter, both right and left. General physical, otological and neurological findings were normal apart from the abovementioned manifestations. Laboratory findings were normal, and X-rays of the mastoid bone revealed no abnormality. The tentative diagnosis was acute peripheral facial paralysis of delayed onset after ear surgery.

Assessment of nerve conduction: NC. difference R/L: 1.2 mA. No electromyography was carried out.

On the basis of the slight functional remnant in the orbic. oculi muscle, an expectant attitude was adopted. Antibiotic treatment had been instituted immediately after the operation.

July 26th: unmistakable function in all muscles except for the corr. supercilii muscle. Subjective improvement also. Right nasopalpebral reflex weak positive. *NC. difference* R/L: 0.4 mA. The patient was instructed to support functional restoration by active muscle excercises.

July 29th: all muscles capable of reasonable contraction. Nasopalpebral reflex clearly positive. Eating and drinking no longer impeded. No longer any lacrimation. *NC. difference* R/L: -0.5 mA.

August 3rd: slight remnant of paresis; no complaints; *NC. difference* R/L: 0.3 mA.

One-year follow-up September 5th, 1961. Complete subjective and objective recovery. *NC. difference* R/L: 0.3 mA.

This patient, who developed facial paralysis 7 days after an ear operation (which was subtotal at examination 5 days after the onset), made a full

recovery. The nerve conduction test (NC. difference R/L always within normal limits) is completely in accordance with this.

In this study we have considered the prognosis of facial paralysis and in particular we have made attempts to find data on the basis of which the functional condition of the nerve can be estimated and followed up from the earliest possible moment. A major handicap was found to be the fact that only few patients can be examined shortly after the onset of paralysis. This is a general difficulty for all those whose special interest is the study and treatment of acute facial palsy. Unless every patient with facial paralysis is regarded as an emergency case, to be admitted on the very day of the onset of paralysis (so that careful observation and serial examinations under controlled conditions are possible), it will remain difficult to clarify the aetiology and pathogenesis, the course and the correct mode of treatment of this important affection.

If this study has contributed to a further approach to these problems, and if it has aroused an interest in methods of investigation such as electromyography and assessment of nerve conduction (an electrodiagnostic test of which we have great expectations, especially after further perfection), then we believe that we have attained our goal.

SUMMARY

The correct treatment of patients with acute peripheral facial paralysis of endotemporal origin has so far remained a subject of considerable controversy. This holds true in particular for paralyses known as Bell's palsy and for those of delayed onset after cranial trauma or an ear operation. Recovery is spontaneous and as a rule rapid in about 80% of these patients. It is generally believed that this percentage can be increased (and that by timely and appropriate treatment cures can also be effected in the 20% which have so far shown only incomplete or no recovery), if the individual severity of the facial nerve lesion, and therefore the prognosis, can be established in an early stage after the onset of paralysis.

A discussion of these problems and an explanation of nerve degeneration and nerve regeneration (with the associated synkinesis and contracture formation) is followed by an attempt to establish whether useful prognostic data can be obtained by clinical and electro-diagnostic investigation. The data obtained in 220 cases of peripheral facial paralysis are elaborated for this purpose.

Among the *clinical data,* only the extent of the paralysis (partial or complete), the tone of the facial muscles and the nasopalpebral reflex can yield valuable indications in a number of cases. However, these data provide no firm starting-point for further action before the end of the second week after the onset of paralysis. At that time it is possible to predict a full recovery as highly probable if the paralysis is partial from the onset, muscle tone satisfactory and the nasopalpebral reflex persistent; in cases which show complete paralysis at that time, the chance of full recovery is virtually zero. For the other patients, and at an earlier time, clinical investigation yields no data of value in the individual prognosis.

A discussion of data obtained by *electrical examination* of patients is preceded by a review of various methods of electro-diagnosis. Electromyographic examination and nerve conduction assessment were selected for this study because these methods yield quantitative data which can be compared from examination to examination. An explanation of these testing techniques is followed by a detailed discussion of the findings obtained in normal and paralysed peripheral nerves in general, and those obtained in the facial nerve in particular.

On the basis of *electromyographic* examination it is possible to make an individual assessment of the prognosis of facial nerve paralysis, but not before 10-14 days after the onset of paralysis. Demonstration of fibrillations indicates degeneration of at least a number of nerve fibres; in such cases the possibility of incomplete recovery must be seriously taken into account. If no fibrillations are found even at repeated examination, then complete recovery can be expected.

With the aid of the *nerve conduction* test it is possible — at any given moment — to establish whether the impulse conduction (for electrical impulses with a duration of 0.3 msec.) in the paralysed facial nerve is normal, impaired or lost. Serial determinations facilitate a close follow-up on the paralysis. In this way it is possible to detect the majority (80-90%) of future unfavourable or dubious prognoses within 2 weeks, and a large number (about 50%) even within 1 week of the onset of paralysis.

If surgical exploration of the facial nerve is advocated as soon as there are signs heralding an unfavourable prognosis, on the basis of *clinical findings* such an indication is given in the patients who show complete paralysis two weeks after its onset. On the basis of *electromyographic* findings, a surgical indication can be accepted when fibrillations are found in the presence of complete paralysis or severe paresis. If the patient can be examined in an early stage, and if the course of the paralysis can be followed by serial *nerve conduction* tests (accepting an NC. difference between the affected and the contralateral nerve of 3.5 mA. and more as surgical indication), it is possible to submit a large number of patients with an imminent unfavourable prognosis to surgery within the first week, and the majority of these patients before the end of the second week.

Electrical tests are a supplement to clinical investigation, but the latter constitutes the basis of the therapeutic approach. If surgery is resorted to only when electrical findings correlate with clinical findings (i.e. in complete paralysis or severe paresis), the risk of unnecessary surgery is reduced to a minimum.

The relevant chapter includes some suggestions for perfecting the promising method of nerve conduction assessment. Perfection of diagnosis can only be expected to be effective, however, if an increasing number of patients can be examined in the earliest stages of paralysis, and then followed up. If this is to be attained, it seems necessary to act in accordance with the statement made by COHEN: "Facial paralysis, a medical emergency".

SAMENVATTING

Over de juiste behandeling van patiënten met acute periphere facialisparalyse van endotemporale oorsprong is men het hedentendage nog geenszins eens. Dit geldt vooral voor verlammingen aangeduid als Bell's palsy en voor die welke ontstaan zijn enige tijd na een schedeltrauma of ooroperatie, dus na een vrij interval. Ongeveer 80% dezer patiënten geneest spontaan en in de regel in korte tijd. Men is algemeen van oordeel dat dit percentage verhoogd kan worden — en dat dus ook voor de circa 20% die thans nog onvolkomen of in het geheel niet herstellen, maatregelen ter genezing kunnen worden getroffen — wanneer in een zeer vroeg stadium na het begin van een verlamming voor iedere patiënt afzonderlijk de ernst van het facialisletsel en daarmee de prognose kan worden bepaald.

Na een bespreking van deze problematiek en na een uitweiding over zenuwdegeneratie en -regeneratie (en de daarbij voorkomende synkinesieën en contracturen) wordt nagegaan of bruikbare prognostische gegevens kunnen worden ontleend aan het klinisch en electro-diagnostisch onderzoek. Hiervoor worden de gegevens van 220 patiënten met periphere facialis verlamming bewerkt.

Van de *klinische gegevens* kunnen alleen de ernst van de verlamming (partiëel of totaal), de tonus van de gelaatsmusculatuur en de nasopalpebraalreflex in een aantal gevallen waardevolle aanwijzingen verschaffen. Een redelijk houvast heeft men aan deze gegevens echter niet vóór het einde van de tweede week na het begin van de verlamming. Men kan dan zeggen dat, wanneer de verlamming van het begin af aan partiëel, de spiertonus goed en de nasopalpebraalreflex behouden is gebleven, met aan zekerheid grenzende waarschijnlijkheid een volledige genezing verwacht kan worden; voor patiënten die op dat tijdstip een totale paralyse hebben is de kans op volkomen herstel vrijwel nihil. Voor de overige patiënten

en op een vroeger tijdstip vermag het klinisch onderzoek ten aanzien van de individuele prognose geen aanwijzingen te geven.
De bespreking van de gegevens, verkregen met behulp van *electrisch onderzoek* der patiënten, wordt voorafgegaan door een beschouwing over de verschillende methoden van electro-diagnostiek. Electromyographie en nerve conduction onderzoek zijn voor deze studie gekozen omdat met die methodieken quantitatieve, van onderzoek tot onderzoek vergelijkbare, gegevens verkregen worden. Na een uiteenzetting van de techniek dezer tests worden de bevindingen bij normale en verlamde periphere zenuwen in het algemeen en bij de nervus facialis in het bijzonder uitvoerig besproken.
Aan de hand van *electromyographisch* onderzoek is het mogelijk de prognose van een facialisverlamming te bepalen voor de individuele patiënt, echter niet eerder dan 10-14 dagen na het begin van de verlamming op zijn vroegst. Het vinden van fibrillaties wijst op degeneratie van tenminste een aantal zenuwvezels; men moet dan ernstig rekening houden met een onvolkomen genezing. Worden — ook bij herhaald onderzoek — geen fibrillaties aangetroffen, dan kan men een volledig herstel tegemoet zien.
Met behulp van de *nerve conduction* test kan men op elk gewenst ogenblik nagaan of de impulsgeleiding (voor electrische prikkels met een duur van 0.3 msec.) in de verlamde nervus facialis normaal, bemoeilijkt of opgeheven is. Aan de hand van serie-bepalingen kan men het verloop van een verlamming op de voet volgen. Het is mogelijk op deze wijze van de patiënten, die een ongunstige of dubieuze prognose beschoren is, de meesten (80-90%) binnen twee weken en een groot aantal (circa 50%) reeds in de loop van de eerste week na het begin van de verlamming op te sporen.

Is men voorstander van operatieve exploratie van de nervus facialis zodra tekenen voorhanden zijn dat een ongunstige prognose dreigt, dan komen op grond van het *klinisch* onderzoek voor operatie in aanmerking de patiënten die twee weken na het begin van de verlamming een totale paralyse hebben. Op grond van *electromyographisch* onderzoek is een indicatie tot operatie aanwezig als bij een totale paralyse of een ernstige parese fibrillaties gevonden worden. Is men in de gelegenheid de patiënt reeds in een vroeg stadium te onderzoeken en het verloop van de verlamming aan de hand van serie- *nerve conduction* bepalingen te volgen, dan kan men, uitgaande van een verschil in nerve conduction tussen de verlamde en gezonde nervus facialis van 3.5 mA. en hoger als operatie-

indicatie, van de patiënten voor wie een ongunstige prognose dreigt een aantal reeds in de eerste week en de meesten vóór het einde van de tweede week opereren.
Electrische tests dienen ter aanvulling van het klinisch onderzoek; dit laatste vormt de basis van het therapeutisch handelen. Opereert men naar aanleiding van de uitslag van het electrisch onderzoek alleen dán als die uitslag correleert met de klinische gegevens (dus bij een totale paralyse of een ernstige parese), dan is het risico dat patiënten onnodig geopereerd worden gering.

In het desbetreffende hoofdstuk worden enkele suggesties gedaan ter vervolmaking van het veelbelovende onderzoek der nerve conduction. Verfijning der diagnostiek zal echter slechts vruchten kunnen afwerpen wanneer meer en meer patiënten in het vroegste stadium van de verlamming onderzocht en daarna vervolgd kunnen worden. Om dit te bereiken lijkt de zienswijze van COHEN: "Facial paralysis, a medical emergency" noodzakelijk.

RESUME

Actuellement on n'est pas encore d'accord sur le traitement correct de la paralysie faciale périphérique d'origine endotemporelle. La divergence des opinions regarde surtout la paralysie faciale dite à frigore (Bell's palsy) et la paralysie à la suite d'une opération ou d'un traumatisme du crâne se manifestant après un intervalle libre. Il est certain qu'environ 80% des malades guérissent spontanément et dans assez peu de temps. Pourtant on accepte généralement que ce pourcentage serait augmenté si on pouvait déterminer la sévérité et le pronostic d'une paralysie faciale déjà dans un stade très précoce de la maladie.

Dans cette monographie nous discutons d'abord les problèmes concernant la dégénération et la régénération des nerfs ainsi que les contractures et les syncinésies. Nous nous sommes demandés si l'examen clinique et électrique puissent nous renseigner sur le pronostic de chaque paralysie qui se présente. Pour cela nous avons étudié les histoires de 220 malades atteints d'une paralysie faciale périphérique.
En ce qui concerne les *symptomes cliniques* il y en a trois qui sont de valeur pour le pronostic dans un certain nombre de cas, à savoir l'importance de la paralysie (partielle ou totale), le tonus des muscles de la face et le réflexe nasopalpebral. Cependant ces symptomes n'ont de valeur qu'après quinze jours dès le début de la paralysie. A ce moment-là ont peut distinguer les deux catégories suivantes: 1) Les paralysies d'emblée partielles avec tonus musculaire conservé en présence du réflexe nasopalpebral; pour ces paralysies la restauration complète est presque certaine. 2) Les paralysies totales; pour celles-ci il est prèsque certain que la restauration complète des fonctions n'aura jamais lieu. Dans tous les autres cas ou bien dans une phase plus précoce de la paralysie l'examen clinique ne nous permet pas à tirer des conclusions concernant l'issue de la paralysie.
Après un aperçu sur les différentes méthodes de l'électro-diagnostic nous discutons les données de l'*examen électrique* de nos malades. Pour notre étude nous avons choisi l'électromyographie et l'examen de la conduction

nerveuse ("nerve conduction test") qui permettent une analyse quantitative. Par conséquent on peut comparer les résultats d'une façon objective. Nous décrivons la technique de ces deux tests, ensuite leur valeur pour l'examen des nerfs périphériques normaux et paralysés en général et pour le nerf facial en particulier.
L'examen *électromyographique* nous permet de pronostiquer le cours de la paralysie de chaque malade individuel, mais sûrement pas avant 10 à 14 jours après le début de la paralysie. La présence de fibrillations prouve la dégéneration d'un certain nombre de fibres nerveuses; dans ce cas il faut compter sur une restauration incomplète des fonctions. L'absence de fibrillations au cours des examens consécutifs au contraire fait prévoir la guérison complète.
L'examen de la *conduction nerveuse* nous permet à chaque moment voulu d'établir si la conduction dans le nerf paralysé (pour un stimulus électrique d'une durée de 0.3 msec.) est normale, ralentie ou supprimée. Il est donc possible de suivre de près le cours d'une paralysie par des analyses répétées. Ainsi nous sommes arrivés à reconnaître la plupart (80-90%) des cas défavourables déjà dans les premiers quinze jours et même pour la moitié des cas déjà dans la première semaine de la paralysie.

Pour ceux qui préconisent l'exploration du nerf facial dès la reconnaissance de symptomes défavorables l'indication opératoire se présente 1) si une paralysie totale existe deux semaines après le début de la maladie, 2) si l'électromyographie décèle la présence de fibrillations chez une paralysie totale ou une parésie grave, 3) si au cours des examens consécutifs de la conduction nerveuse se manifeste une différence de 3.5 mA. ou plus entre le nerf paralysé et le nerf normal.
Les tests électriques sont le complément de l'examen clinique qui est lui même à la base de nos décisions pour le traitement nécessaire. Il faut seulement opérer si les résultats des examens électriques sont en rapport avec les données cliniques pour éviter les operations inutiles.

Dans le chapitre correspondant nous présentons quelques idées pour perfectionner l'examen de la conduction nerveuse. D'autre part nous sommes persuadés qu'un raffinement des méthodes n'apportera ses fruits qu'en examinant les malades de plus en plus dans les phases les plus précoces de la paralysie en les suivant ainsi de près. Pour y parvenir il est nécessaire de souscrire à la pensée de COHEN: "Paralysie faciale, cas d'urgence".

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APPENDIX

In the chapter "Assessment of Nerve Conduction" WILCOXON's two sample test and WILCOXON's test for symmetry are studied as statistical methods. A detailed description of these tests is given in:

[1] WABEKE, D., VAN EEDEN, C. (1955). Handleiding voor de toets van WILCOXON. Report S 176 (M 65) of the Statistical Department of the Mathematical Centre (Amsterdam).

[2] BENARD, A., VAN EEDEN, C. (1956). Handleiding voor de symmetrietoets van WILCOXON. Report S 208 (M 76) of the Statistical Department of the Mathematical Centre (Amsterdam).

Here only a review of these studies will be given.

Testing a hypothesis

A test for a hypothesis H_0 is based upon a number of observations $x_1, \ldots, x_n$ of one or more random variables* and is executed by means of a *test statistic* **u**, which is a function of the observations. Supposing H_0 to be true, the probability distribution of **u** can be calculated.
A set Z of possible outcomes of **u** is chosen in such a way that the probability that **u** assumes a value in Z, supposing H_0 to be true, is equal to or smaller than a given number α (the so-called *level of significance*). This set Z is called the *critical region* of the test; the true probability that **u** assumes a value in Z if H_0 is true is called the size of Z. The size of Z

* Random variables will be distinguished from numbers (e.g. from the values they take in an experiment) by printing their symbols in bold type.

is not always equal to α owing to the discrete character of the values **u** can take.
The hypothesis H_0 is rejected if the value of **u** calculated from the observations, lies in Z. The probability that this will happen if H_0 is true is equal to the size of Z and thus smaller than or equal to α.
The result of a test may also be expressed in the so-called *tail probability* k; this is the size of the smallest critical region containing the result. Using a level of significance α, H_0 is rejected if $k \leqq \alpha$.

Wilcoxon's test for symmetry

By means of WILCOXON's test for symmetry the hypothesis H_0 may be tested that the random variables $\mathbf{z}_1, \ldots, \mathbf{z}_m$ are all distributed symmetrically with respect to zero. The test statistic **T** of this test is calculated as follows. The observations which are equal to zero are omitted. Now let $x_1, \ldots, x_{n_1}$ denote the positive observations and let $y_1, \ldots, y_{n_2}$ denote the absolute values of the negative observations. Then if W is the test statistic of WILCOXON's two sample test applied to $x_1, \ldots, x_{n_1}$ as the first sample and $y_1, \ldots, y_{n_2}$ as the second sample then T is defined by

$$T = W - n_1 n_2 + \tfrac{1}{2}(n+1)(n_1 - n_2),$$

where $n = n_1 + n_2$.
Tables of the distribution of **T** under the hypothesis H_0 may be found in [2] for $n \leqq 20$. For $n > 20$ this distribution may be opproximated by a normal distribution with mean

$$\mu = o$$

and variance

$$\sigma_2 = \frac{3n(n+1)^2 + n^3 - (t_1^3 + \ldots + t_h^3)}{12}$$

where h is the number of ties in the non-zero values assumed by $|\mathbf{z}|, \ldots, |\mathbf{z}_m|$ and t_i $(i = 1, \ldots, h)$ is the size of the i-th tie.
The two-sided critical region consists of large values of $|T|$; the one sided critical regions consist of large positive (resp. negative) values of T.

Wilcoxon's two sample test.

By means of WILCOXON's two sample test the hypothesis H_0 may be tested that two samples $x_1, \ldots, x_m$ and $y_1, \ldots, y_n$ are observations of two random variables (**x** and **y**) possessing the same probability distribution.
The statistic **W**, used for this test, is calculated from the observations as follows. We determine the number of observations in the second sample which are larger than the i-th observation x_i of the first sample; let this number be u_i. Then we determine the number of observations in the second sample which are equal to x_i; let this number be v_i $(i = 1, \ldots, m)$.
Then

$$W = 2u_1 + v_1 + \ldots + 2u_m + v_m = \sum_{i=1}^{m} (2u_i + v_i).$$

Tables of the distribution of **W** under the hypothesis H_0 may be found in [1] for m and $n \leqq 10$. For larger values of m and n this distribution may be approximated by a normal distribution with mean

$$\mu = mn$$

and variance

$$\sigma^2 = \frac{mn}{3N(N-1)}\left\{N^3 - (t_1^3 + \ldots + t_h^3)\right\},$$

where $N = m + n$, h is the number of ties in the pooled samples and t_i $(i = 1, \ldots, h)$ is the number of observations in the i-th tie. (A tie is a group of equal observations).
If H_0 is not true **W** will assume large or small values according to **y** being systematically larger or smaller than **x**. Therefore a two-sided critical region consisting of larger values of $|W - \mu|$ is used if one wants to tests H_0 against the alternative hypothesis that **y** is systematically larger or smaller than **x**. If H_0 is tested against the alternative hypothesis that **y** is systematically larger (resp. smaller) than **x** a one-sided critical region is used consisting of large (resp. small) values of W.

STELLINGEN

I Electriseren van verlamde aangezichtsspieren heeft hoofdzakelijk psychologische betekenis.

II Indien bij pasgeborenen verdenking op blindheid bestaat kan electroretinographie hieromtrent zekerheid geven.

(Arch. Ophthal., Chicago 1960, *63*, 282).

III Er is dringend behoefte aan een nauwgezette bestudering van de vraag wat de medicus aan de kankerpatient moet meedelen omtrent de aard en de prognose van diens ziekte.

IV Het bestaan van zogenaamde apocriene secretie is twijfelachtig.

(Zeitschrift für Zellforschung 1961, *53*, 545).

V Voor het bepalen van de juiste plaats van proefexcisie bij botgezwellen is angiographie onmisbaar.

VI De ophopingen van lymphatisch weefsel in de parotis moeten worden beschouwd als echte lymphklieren met een eigen regionair stroomgebied.

(Cancer 1958, *11*, 1156).

VII "Praeventieve" tandextractie voorafgaande aan een radiologische behandeling in de mond- en keelholte dient verworpen te worden.

(Radiology 1953, *61*, 771).

VIII Het ontstaan van een aangezichtsverlamming is een reden tot spoedopname.

IX Wanneer bij een totale parotidectomie de nervus facialis opgeofferd moet worden dient dit zo perifeer mogelijk te gebeuren; indien onmiddellijk herstel van de continuïteit der zenuwtakken niet mogelijk is moeten de proximale en distale zenuwstompen gemarkeerd worden.

X Het doorboren van het neustussenschot bij jonge mannelijke Papoea's is een sexuele convertering.