EXPERIMENTS ON THE FACIAL NERVE

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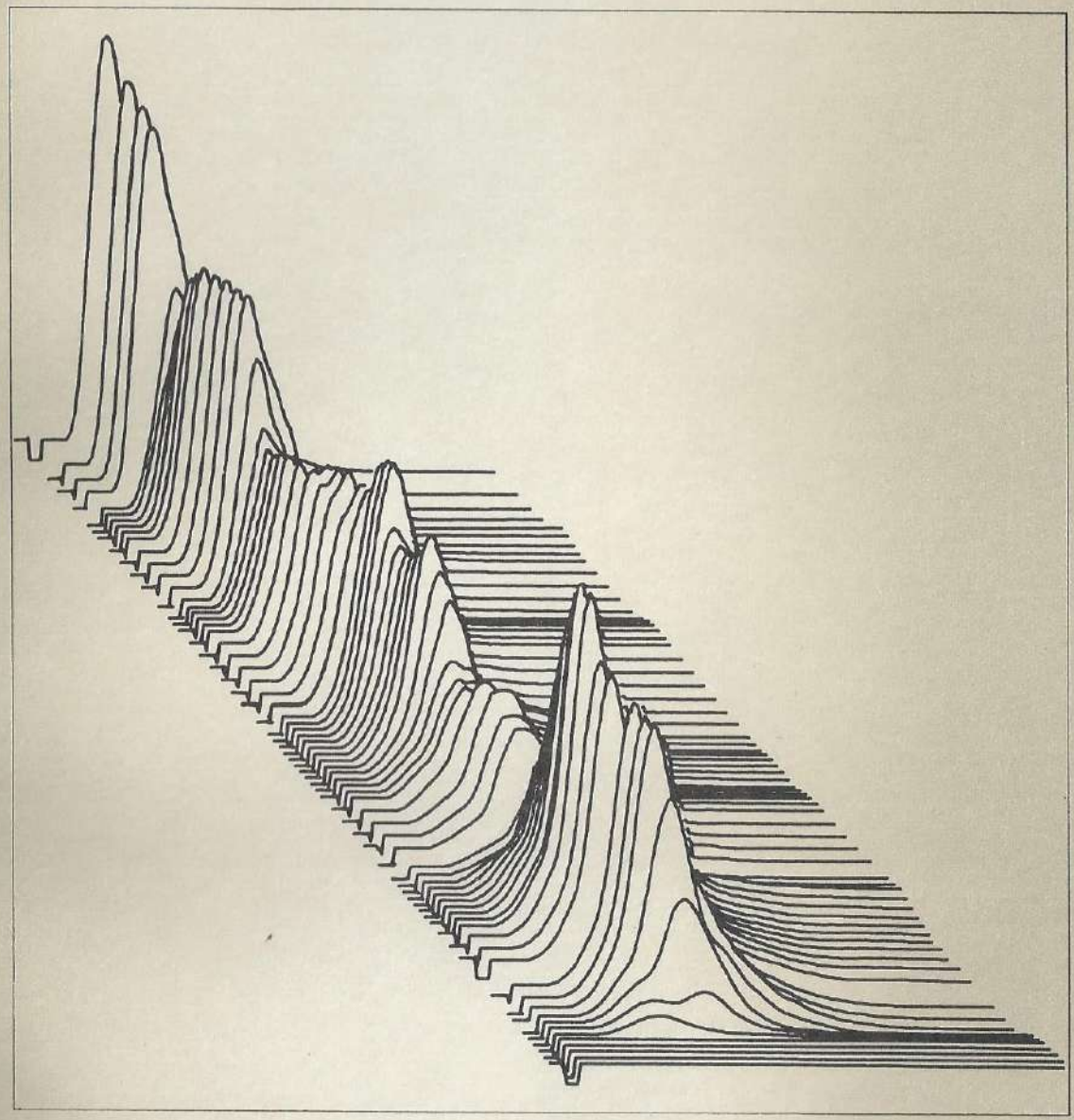

A study of nerve action potentials in the cat

P. P. DEVRIESE — EXPERIMENTS ON THE FACIAL NERVE

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A STUDY OF NERVE ACTION POTENTIALS IN THE CAT

Our present-day concepts (of the nervous system) are still to a large extent based upon assumptions and hypotheses, built upon a modest body of factual knowledge. If further progress is to be made, it is important to be aware of this situation, and to make every effort to distinguish between observations and interpretations. Working hypotheses are important and necessary tools in research, but, as the history of science shows, they have a tendency to become accepted as truths and to hamper instead of promoting progress.

BRODAL, Neurological Anatomy, (1969).

Aan mijn Ouders
Aan Miriam

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PART ONE

CHAPTER I

INTRODUCTION

The present study was undertaken to investigate experimentally the problems related to ischemic paralysis (idiopathic facial paralysis, Bell's palsy) in man.

The variable studied in the experiments was the nerve action potential directly recorded from the cut facial nerve in the cat. In the first Chapter the nerve action potential itself, the facial nerve in the cat, and ischemia and compression of peripheral nerve trunks will first be reviewed.

I – 1. THE NERVE ACTION POTENTIAL

This survey of the single fiber action potential and the compound nerve action potential is based on reviews by HODGKIN (1964), RUCH and PATTON (1965), and WOODBURY (1965).

a. THE ACTION POTENTIAL IN ONE NERVE FIBER

The membrane of excitable cells has certain unique properties. They were described by Hodgkin and Huxley in their study of the squid's giant axon. When the transmembrane charge and voltage of a cell are at their steady state values, the cell is said to be *polarized*. Any increase in the transmembrane potential is called *hyperpolarization*; any decrease is called *depolarization*. Depolarization was found to increase the membrane permeability (conductance) to Na^+. Under normal excitation this increase is transient and falls to its resting value in a matter of a few milliseconds. A survey of the possible molecular mechanisms underlying these properties will not be discussed here, as it bears no relevancy to the subject studied.

HODGKIN and KATZ (1949) proposed that the upstroke or *depolarization phase* of the action potential is brought about by a brief and highly specific increase in the membrane's permeability to Na^+. Repolarization to the resting state would occur as the increased Na^+ – permeability dies out and as the efflux of K^+ exceeds the influx of Na^+. This hypothesis is supported by the fact that the overshoot and rate of increase of the action potential vary with the variations in ion concentrations in approximately the manner expected according to the Nernst equation. The voltage change during *repolarization* hastens the decrease of permeability to Na^+ and, after a delay, to K^+. As a consequence, the permeability to K^+ is still above normal when repolarization is complete, hyperpolarizing the membrane. Thereafter the permeability to K^+ and the membrane voltage fall slowly back to their resting values.

Since during depolarization there is a potential difference between regions of the membrane, a current flows from the active region through the intracellular fluid to an adjacent region and decreases there its membrane potential. The return current flows through the interstitial fluid back to the active region and through the membrane as inward Na^+ current. This *local circuit current flow* acts to reduce membrane charge and voltage in the inactive region. An active region thus stimulates the adjacent inactive regions to above threshold by local circuit current flow and the action potential is conducted away from the stimulated side in both directions at constant speed. Although nerves normally conduct impulses in one direction – sensory fibers towards the central nervous system, motor fibers away from it – all nerves can conduct in *both directions* and the *velocity* at which the impulse propagates is independent of the direction in which it travels.

The myelin sheath of *myelinated nerve fibers* is formed by Schwann cells which cover about 2 mm of axon in the largest fibers (Ø 20 μ). At the gap of approximately 1 μ, between adjacent Schwann cells, the axon membrane is in free communication with the interstitial fluid.

The interruption of the myelin sheath is called the node of Ranvier, and the sheathed portion the internode.

Since the myelin sheath is almost impermeable to the ions constituting the membrane potential, action potentials can only be generated at the nodes. Impulse propagation from node to node is called *saltatory conduction*.

b. The compound action potential

Electrical stimulation of a nerve is usually performed by means of platinum or wire electrodes in direct contact with the nerve. Initiation of an impulse occurs in membrane regions which have been depolarized above threshold. A current flowing between two electrodes in contact with a nerve has two effects, hyperpolarization of membranes in the region of the anode (positive terminal) and depolarization in the region of the cathode. A current which depolarizes to threshold the region of the membrane under the cathode will initiate an impulse. The event which starts the impulse is thus a decrease of the electrical potential across the membrane. The *latency*, the time interval between the shock artifact and the beginning of the action potential, is a linear function of the distance between the stimulating and recording electrodes. A curve relating the strength of a threshold stimulus to its duration is called a *strength-duration curve*. The *rheobase* is the minimal continuous electrical current required to excite a tissue. The *chronaxy* is the minimum time required for excitation of an excitable tissue by a constant electrical current of twice the rheobase.

The *compound action potential* recorded by the active electrode placed on the nerve trunk is composed of the individual action potentials of the constituent axons of the nerve. Nerve fibers are invariably surrounded by an aqueous conducting medium, i.e. the interstitial fluid. This will reduce the

potentials recorded. The volume of the external conducting medium, however, can be limited by lifting the nerve onto electrodes in air or by suspending the nerve in an insulating medium such as mineral oil. In the experiments to be described paraffin and silicon oil were used for this purpose (see Chapter II).

In *bipolar recording* the distance between the recording electrodes is usually less than the length of the depolarized region. Since activity then reaches the distal electrode before repolarization occurs at the proximal electrode, the electrical events interact at each electrode, which may make evaluation difficult.

The action potential can be recorded "*monophasically*" with two electrodes: one on an isolated part of the nerve and another on a part of the nerve permanently depolarized by crushing, burning, cutting or topically applying potassium salts.

The compound action potential recorded monophasically from a nerve trunk after maximal stimulation is usually irregular in contour, displaying two or more elevations in time. At increasing conduction distances each deflection becomes broader and lower in amplitude. Planimetric measurements indicate that the area lying under each deflection remains constant irrespective of the conduction distance (Erlanger and Gasser, 1937). Each deflection of a multiple-hump recording indicates a group of fibers with similar conduction speed. This suggests that within a group as well as from group to group there is a continuous spectrum of conduction speeds. The separation of peaks results not from absolute discontinuities in the velocity spectrum but rather from unequal numerical distribution of fibers representing restricted bands of the spectrum. In a stimulated nerve trunk the conduction velocity and electrical threshold are inversely related, the rapidly conducting axons being more easily excited than the slower ones (Ochs, 1965).

The compound action potential recorded at short conduction distances does not always terminate with the spike potential. A *negative afterpotential* is often grafted to the tail of declining spike. Following the decline of the negative after potential to the baseline, a prolonged *positive afterpotential* occurs. Both afterpotentials are consequences of and dependent on antecedent spike activity; they are of very low amplitude and of long duration relative to the spike. They are also highly labile and depend heavily upon the metabolic state and the previous history of the nerve fiber. During the negative afterpotential, the axons are slightly depolarized and their excitability is elevated (supranormal period); during the positive afterpotential the fibers are slightly hyperpolarized and their excitability is depressed (subnormal period). Afterpotentials are most prominent in small fibers. Their origin is not entirely clear but may reflect metabolic processes associated with recovery. Two mechanisms appear to be responsible for positive afterpotentials: (1) during the post spike period of elevated K^+ permeability, the membrane seeks a potential

level closer to the K^+ equilibrium potential and becomes hyperpolarized; (2) increased active extrusion of Na^+ drives the membrane potential farther from the Na^+ equilibrium potential, in a hyperpolarizing direction.

Axons can be classified into three distinctive types:

Type A: myelinated, somatic, afferent, and efferent fibers. Based on fiber diameter the A fibers can be divided into three groups: I, 12 to 21 μ; II, 6 to 12 μ; and III, 1 to 6 μ. In A fibers (myelinated somatic axons) there is a continuous spectrum of fiber diameters, but the number of fibers in each portion of the diameter spectrum varies in different nerve trunks. Certain bands of the spectrum may lack representation. GASSER and GRUNDFEST (1939) found that the ratio between the conduction rate (in meters per second) and the axon diameter (in μ) within the myelin sheath was 8.7:1. This applies only to A fibers. Type B: myelinated, efferent, praeganglionic fibers in autonomic nerves. Type C: unmyelinated fibers (efferent postganglionic, sympathetic axons and small unmyelinated, afferent axons). The latter type of fibers is sometimes referred to as group IV.

Mammalian A fibers have the following characteristics: fiber diameter: 1–22 μ, conduction speed: 5–120 meter per second, spike duration: 0.4–0.5 msec, absolute refractory period: 0.4–1.0 msec, negative afterpotential: amplitude of 3–5 per cent of spike and duration of 12–20 msec, positive afterpotential: amplitude 0.2% of spike and duration 40–60 msec.

The Greek letters α, β, γ, δ (and sometimes ε) are often used to designate the successive elevations in the monophasically recorded action potential of type A fibers. These elevations result from activity in fibers conducting at different velocities.

An elevation in the compound action potential, however, also reflects the number of fibers involved, provided all nerve fiber action potentials are identically recorded by the electrodes. The Greek letter designation may then also be used as a categorization of fiber diameters. A–α fibers correspond to groups I and II, A–δ to group III. According to PATTON (1965), the deflections in cat nerves originally labelled beta and gamma are largely or wholly artifacts and have no equivalents in the Roman numerical classification.

I – 2. THE FACIAL NERVE IN THE CAT

REIGHARD and JENNINGS (1901) gave a description of the temporal bone and the facial nerve in the cat. The tympanic portion of the temporal bone – the auditory bulla – enveloping the tympanic cavity, is formed by an ectotympanic part surrounding the external auditory meatus, and a larger entotympanic part. Posterior to the fossa for the tensor tympani muscle (the fossa muscularis major of ESCHWEILER, 1899) and separated from it by an oblique bony septum is another fossa enclosed by bone which

contains the stapedial muscle and the facial nerve (the fossa muscularis minor of ESCHWEILER, 1899). A groove may be traced from the caudal border of the fossa for the stapedial muscle to the stylomastoid foramen. The chorda tympani branches off 2 or 3 mm before the emergence of the facial nerve at the stylomastoid foramen. According to VAN KAMPEN (1904, 1905) the third part of the facial canal is formed by the mastoid, the tympanicum, and the entotympanicum. The stylomastoid foramen is situated between these bones and the tympanohyale which originates from Reichert's cartilage.

The facial nerve innervates most of the muscles of the head (REIGHARD and JENNINGS, 1901). On emerging from the stylomastoid foramen it branches off to the digastric muscle, the posterior auricular nerve, and to the inner surface of the cartilaginous external ear. The nerve then curves cranially about the proximal part of the external ear and splits into 2 main branches: a *dorsal ramus* and a *ventral ramus*. The first divides into the temporal and zygomatic branch, the latter, passing across the zygomatic bone to the lateral angle of the eye, sends branches to both eyelids, anastomoses with twigs from the lachrymal branch of the fifth nerve, and passes along the medial side of the eye to the lateral side of the nose. The *ventral ramus* supplies the stylohyoid muscle and the angle of the mouth.

HUBER and HUGHSON (1926) performed an extensive experimental study on the facial musculature in mammals, including a survey of the ramifications of the facial nerve in the cat. The peripheral motor branches can be classified as follows:

Rami auriculares posteriores. Two or three main motor branches leave the foramen stylomastoideum separately. They are accompanied by two small sensory branches: the ramus auricularis internus of the facial nerve and a sensory branch of the nervus vagus, the ramus auricularis nervi vagi. The latter joins the facial branches within the facial canal. Both sensory branches penetrate the ear cartilage at the convex surface shortly after they emerge from the stylomastoid foramen. The rami auriculares posteriores supply the muscles derived from the primitive platysma. Anastomoses occur between these branches and the cutaneous nerves of the cervical plexus.

The main stem of the facial nerve with preauricular branches. Shortly after the stem has emerged from the stylomastoid foramen it breaks up into 5 large branches:

1. the ramus temporo-frontalis
2. the ramus zygomatico-orbitalis
3. the ramus bucco-labialis superior
4. the ramus bucco-labialis inferior and
5. the ramus colli.

Branches to deep facial muscles. In all the experiments to be described, the ramus zygomatico-orbitalis will be used for distal stimulation.

FOLEY and DUBOIS (1943) made a quantitative and qualitative study on the fiber content of the facial nerve in the cat and on its relationship to the ramus auricularis of the vagus nerve and to the cervical plexus. Near the brain stem the three fairly distinct bundles of fibers can be discerned: a dorsal (sensory), ventral (motor), and intermediate (visceral) group.

1. *The motor fibers*

Distal to the geniculate ganglion and on to the stylomastoid foramen the motor element remains fairly discrete; ocassionally it is invaded by small extensions from the sensory group. Although the motor component maintains its individuality throughout the remainder of its course within the facial canal, there is an increasing invasion by auricular vagus and facial sensory fascicles distal to the level of the stapedial muscle. Outside the foramen it divides into a number of fascicles which, in company with auricular and sensory facial fibers, are distributed to the head in peripheral branches of the facial nerve. The axons of the motor component are predominately myelinated, and the fibers range from 1.5 to 13 μ in diameter with the majority being from 3.5 to 5.5 μ. The motor fibers constitute about 70 per cent of the axons in the facial nerve just distal to the geniculate ganglion. The remainder are either visceral motor or sensory in origin. The total number of axons at the level of the stylomastoid foramen varies: the authors report 18, 910 to 28,471 axons in three cats. Immediately distal to the stylomastoid foramen there were from 10,735 to 11,520 myelinated fibers in the trunk of the nerve in two cats; 2,120 to 3,645 of these originated from the ramus auricularis of the vagus nerve and the sensory component of the facial nerve.

2. *The visceral motor fibers*

The visceral fibers, mixed with sensory axons, separate into two sharply defined groups for distribution in the greater superficial petrosal nerve (approx. 70 per cent) and the chorda tympani (approx. 30 per cent). The preganglionic visceral motor fibers may be completely myelinated, largely unmyelinated, or practically any variation between the two arrangements. They are 2.5 μ or less and comprise about 15 per cent of the total axons of the facial nerve.

3. *The sensory fibers*

Thirty-three to 40 per cent of the sensory axons from the facial nerve contribute to the composition of the great superficial petrosal nerve. The remaining sensory fibers run distal in the facial nerve, occupying a position to one side of the motor fibers. Just before the facial nerve leaves the facial canal, it gives off the chorda tympani which removes the remaining visceral motor fibers and the majority of the residue of sensory fibers of the trunk of the nerve. About 50 per cent of the sensory fibers sprout into the chorda tympani while the remaining 12 to 15 per cent continue in the facial nerve distal to the stylomastoid foramen more closely associated with vagal than facial axons. Except those in the greater superficial petrosal nerve, the majority are myelinated, their diameter varying from 1.5 to 6.5 μ. The number of myelinated axons varies in different animals (between 67 and 90 per cent in 3 cats). The sensory fibers comprise about 16 per cent of the facial nerve fibers (2,000 to 2,500 fibers).

4. *The auricular nerve*

The auricular nerve distributes from 6,000 to 10,000 fibres through the trunk of the facial nerve and enters the motor zone at, or slightly proximal to, the origin of the chorda tympani. It then shifts clockwise (right facial nerve), moving towards the sensory component with which it eventually fuses. Fascicles detach themselves from the main body of the nerve and scatter into the somatic and sensory portion. Fascicles of the auricular fibers pass into or in close proximity to all branches of the facial nerve for a short distance after it leaves the stylomastoid foramen. Most of the axons of the auricular nerve collect into a definite nerve which passes to the ear in company with the motor branches of that region.

5. *Peripheral branches*

In one cat about 11,000 motor fibers of 1.5 to 13 μ in size were available in the trunk for distribution into the muscular branches. The posterior auricular and zygomatico-orbital branches (PAPEZ, 1929) received the majority of the large axons. The distribution was as follows: 14 per cent to the anterior auricular, 15 per cent to the zygomatico-orbital, 9 per cent to the superior labial, 14 per cent to the inferior labial, 32 per cent to the posterior auricular and 3 per cent to the several small cervical branches. About 13 per cent was probably distributed to the stapedial, stylohyoid, and digastric muscles. Variation undoubtedly occurs in individual animals.

After degeneration of the somatic motor fibers about 500 small and largely unmyelinated axons remained in the zygomatico-orbital branch. The origin of these fibers has not been determined but they are not believed to arise from cells of the geniculate ganglion.

BRUESCH (1944) studied the distribution of the myelinated afferent fibers in the branches of the facial nerve of geniculate ganglion and auricular nerve origin. The facial afferent component, totaling 1,700 myelinated fibers, 1 or 1.5 to 6 μ in size, distributes 8 per cent of its fibers to the mimetic muscles. The auricular branch of the *vagus* contributes a variable number of myelinated fibers to the facial trunk (about 1,000 to 3,200 fibers). The branch joins the facial nerve immediately distal to the nerve to the stapedial muscle, and, sharing the capsule of the facial nerve, passes through the stylomastoid foramen (VAN BUSKIRK, 1945). Three to five small branches pass from the auricular nerve through the stapedial muscle and join the facial trunk. In a more recent study by BLEVINS (1964)

the latter could not be confirmed. Seven per cent of the fibers (1 or 1.5 to 20 μ in diameter) is distributed to the mimetic muscles.

According to BRODAL (1969) it is apparent from comparative anatomical data as well as from the experimental findings that most of these fibers are distributed to the external ear, partaking in the sensory cutaneous innervation of the concha of the auricle and sometimes of an area behind the ear. This cutaneous sensory branch of the facial nerve is also present in man.

I – 3. ISCHEMIA AND COMPRESSION OF PERIPHERAL NERVE TRUNKS

Many clinical and experimental observations about injuries of peripheral nerves have been published in the past. The literature published before 1967 has been reported by Sunderland (1968). His classification of nerve injuries and related considerations about anatomy and neurolysis will be reviewed here only to the extent that they are relevant to the facial nerve experiments reported.

1. CLASSIFICATION OF NERVE INJURIES

Nerve trunk lesions which produce loss of function can be classified into five degrees of injury of increasing intensity (SUNDERLAND, 1951). The injuries may not be of uniform severity, and produce partial and mixed lesions. A great variety of agents (mechanical, thermal, chemical, ischemic) may cause the five degrees of nerve involvement.

First degree of injury (This is also called loss of conduction or neurapraxia (SEDDON, 1943)).

The axonal continuity is preserved, the disturbance is fully reversible, the conduction block is restricted to the damaged segment and no Wallerian degeneration follows. The excitability of the nerves subjected to mechanical deformation and ischemia is first increased and then declines; they cease to respond to electrical stimulation about thirty minutes after the onset of ischemia (GRUNDFEST, 1936; THOMPSON and KIMBALL, 1936; LEKSELL, 1945; KUGELBERG, 1946; PORTER and WHARTON, 1949; and others). In the rat WEISS and DAVIS (1943) demonstrated a block within ten minutes of the onset of compression in most or all nerve fibers with arterial sleeves and adrenalin around isolated nerve trunks.

Experiments on human nerves reveal that function is rapidly restored following release of pressure which has rendered the limb ischemic for thirty to forty minutes.

A difference in susceptibility to ischemia of sensory fibers at different distances in the arm is shown by LEWIS et al. (1931), distal being less sensitive than proximal. According to SINCLAIR (1948) this can be explained by differences in regional anatomy of the arm. The physiopathological basis of the mildest compressive lesions that produce transient effects

limited to seconds, minutes or perhaps hours is not known. It should be stressed that in experiments with cuff compression the interpretation is complicated by ischemia of all other tissues in the entire limb that is subjected to the experiment. When compression is prolonged, a point is reached at which recovery does not immediately follow release of pressure or is delayed in onset for days or weeks.

It has been shown that conduction is restored before histological changes are totally corrected (DENNY-BROWN and BRENNER, 1944a, b) and that nerve fibers with a grossly modified sheath structure may continue to function efficiently. Moreover, because a nerve block may exist without any detectable modification of morphology (WEISS and DAVIS, 1943), it may be concluded that in prolonged conduction block the disturbance leading to interruption of conduction involves mainly the axon and not its sheath. The conclusion that transient nerve block associated with compression of mammalian nerves without angulation must be effective through the related ischemia (DENNY-BROWN and BRENNER, 1944a) is in general agreement with observations of many authors.

Differences between nerve block due either to pressure only or to uncomplicated ischemia have been reported. Experiments of ALLEN (1938a) would suggest that permanent paralysis arises only from compression. In tourniquet paralysis mechanical pressure plays a greater part, if not the only one, as the cause of paralysis (MOLDAVER, 1954). A nerve block by pressure develops much slower and persists longer than a block by ischemia (BENTLEY and SCHLAPP, 1943b).

It has been suggested (WEISS and DAVIS, 1943) that the centrifugal flow inside the axon provides materials essential for efficient functioning of the axon. This proximo-distal flow may be impeded by compression sufficiently strong to alter the molecular structure of the axoplasm, interrupting conduction but not threatening the survival of the axon. But when the survival demands are no longer satisfied, axonal desintegration and Wallerian degeneration occur.

Persistant constriction sufficient to block conduction but insufficient to threaten axonal continuity may be a factor in those cases in which operative neurolysis is immediately followed by a pattern of recovery which would be impossible via axon regeneration. This type of lesion can be considered as a subdivision of first degree injuries. After a long interval without recovery, exploration and simple neurolysis are followed by the early onset of recovery. The phenomenon is usually explained by the coincidence of the surgical procedure with the onset of spontaneous recovery. This explanation probably accounts for many cases, but in some injuries, associated with prolonged loss of function, fibrous constriction is apparently responsible for maintaining a conduction block which persists until the constriction is relieved. How long conduction can be interrupted in this way, without the physical breakdown of the system, remains unknown.

Second degree injury

Here the axon fails to survive distal to the level of the injury and for a variable, short distance proximal to it. The general arrangement of endoneurium and nerve trunk is preserved; the injury and Wallerian degeneration do not threaten the endoneurial wall. The regenerating axon follows the endoneurial tube it occupied originally and is always directed to the end organ it originally innervated. As a result function is fully restored.

The interval between injury and the onset of recovery of nerve function is influenced by the level of injury, the rate of growth of the axonal tips and duration of denervation of the affected tissues.

The course of recovery differs from that occurring after a first degree injury. The paralysed muscles are reinnervated in the order in which they were originally supplied by the nerve. The sensory recovery will be in strict conformity with the distance which must be grown by the sensory fibers. Function is fully restored only after months as opposed to weeks in the case of first degree injuries.

Third degree injury

In addition to axonal desintegration and Wallerian degeneration, the internal structure of the funiculi is desintegrated. The perineurium shows only minor changes and the general funicular arrangement of the nerve is retained. The continuity of the endoneurial tube, however, is destroyed. In some severe forms of injury the picture is further complicated by intrafunicular hemorrhage, edema, vascular stasis, and ischemia. These complications favor intrafunicular fibrosis which provides the most serious obstacle to regeneration. In some cases the funiculus develops a fusiform swelling.

The retrograde effects are more severe than after a second degree injury, particularly when the nerve is damaged at proximal levels. Some neurons are usually lost. Regeneration is complicated by retrograde disturbances, intrafunicular fibrosis, and misdirection of regenerating axons. The new pattern of reinnervation may differ significantly from the original.

The adverse effects of erroneous cross-shunting may vary according to the fiber composition of the individual funiculi. The consequences in the pattern of normal reinnervation are particularly serious when the fibers are contained in one or two large funiculi (e.g. ulnar nerve, radial nerve) (see Chapter V). As will be shown this is also the case with some parts of the human facial nerve. The chances of erroneous cross-shunting of axons into other endoneurial tubes are increased and the new pattern of innervation may be so distorted that little recovery may result despite the fact that funicular continuity is preserved. There is no external indication of the severity of the intrafunicular damage other than perhaps some light swelling and induration. A funicular arrangement in which the nerve is composed of numerous, small, separated funiculi reduces the effect of these factors. The onset of recovery is delayed for a longer period than after a second degree injury. The course of recovery is the same but recovery in individual structures takes place more slowly and is usually incomplete. Slower recovery of function is due to (1) a longer duration of denervation which may introduce changes in the peripheral tissues; (2) fiber-mixing at the site of injury to such a degree as to prevent full compensation by re-education; and (3) the fact that some muscle fibers may not be reinnervated (as the result of retrograde degeneration or the failure of some regenerating axons to reach the muscle).

Compression, ischemia, and vascular damage introduce intrafunicular complications and aggravate scarring. At levels where the nerve fibers are well intermingled or contained in one or two funiculi, little if any recovery is to be expected. Gross observation may show surprisingly little evidence of the severe disorganisation that has taken place inside the funiculi.

Fourth degree injury

The bundles are so disorganised by the injury that they are no longer sharply demarcated from the epineurium. Continuity of the nerve is preserved; the involved segment is ultimately converted into connective tissue; a neuroma may be formed. The retrograde neuronal effects are more severe than after a third degree injury, and there is a greater reduction in the number of surviving axons. Regenerating axons are free to enter the interfunicular spaces and may terminate blindly. As a result of more severe scarring, few axons reach the periphery and make useful connections. This type of injury requires excision of the involved segment and surgical repair of the nerve.

Fifth degree injury

The continuity of the nerve trunk is lost. The appearance of the severed nerve depends on the time when the injured region is examined, since the reaction developing at the site of injury and the subsequent regeneration modify the picture. Scar tissue as well as the separation of nerve ends constitute a formidable barrier to spontaneous recovery. Neuroma formation may occur on the proximal stump, the distal stump or on both. Recovery after an untreated fifth degree injury is negligible. Even with the most favorable conditions at the time of suture of the nerve ends, a significant loss of axons and distortion of the fiber pattern occurs.

In lesions in continuity, it is possible to meet with every grade of intrafunicular damage, ranging from a first degree of injury to one in which the endoneurial tubes are destroyed. In such mixed lesions the damage falls unevenly across the nerve so that some parts are more severely affected than others. Partial and combined lesions can be explained in terms of the five primary types.

2. ANATOMY OF NERVE TRUNKS IN RELATION TO NERVE INJURY

Differences in size and number of the funiculi, and the amount of epineural tissue surrounding and separating them help to explain why some nerves, or segments of nerves, are more susceptible to mechanical injury. In the first place nerve fibers are more susceptible to compression at places where the nerve trunk is composed of large funiculi with little supporting epineurial tissue. Peripheral nerves composed of a large number of small bundles widely separated by a relatively greater amount of epineurium withstand compression more favorably. In the former case the force falls maximally on the nerve fibers; in the latter the effects of compression are dispersed by the epineurial packing and the funiculi are more easily displaced within the nerve, reducing the effects of the deforming force. Secondly, in the nerve composed of a single funiculus the arteries supplying the nerve are superficially placed; they are less well protected than the more deeply situated vessels of multifuniculated nerves in the connective tissue between the funiculi (DENNY-BROWN and BRENNER, 1944a).

The consequences of a partial lesion are thus particularly serious when the nerve is damaged at a level where the nerve fibers are concentrated in a single funiculus. Rupture of the perineurium leads to more wide-spread changes throughout the funiculus. All or most of the nerve fibers are involved as a result of the associated vascular damage: herniation of the funicular tissue and scar tissue formation. Such damage usually requires total resection and grafting of the peripheral nerves. In this way the ingrowth of connective tissue and the formation of scars are arrested.

3. ISCHEMIA OF PERIPHERAL NERVES

Nerve fibers require an adequate and continued supply of oxygen to function normally (GERARD, 1930; BENTLEY and SCHLAPP, 1943b; PORTER and WHARTON, 1949).

The vasa nervorum normally supply the oxygen but a nerve ischemic by interference with its blood supply, may continue to receive sufficient oxygen by diffusion from adjacent vascularised tissues to survive and to conduct for some time (BENTLEY and SCHLAPP, 1943b). According to BLUNT (1960) this applies, however, only to small nerve trunks in small experimental animals. The arterial supply to peripheral nerves is provided from longitudinally arranged arterioles on the surface of the nerve trunk, an uninterrupted intraneural system of longitudinal arterioles, and regional nutrient arteries reinforcing the longitudinal systems. The anastomoses between these three systems and the overlap in the distribution of the regional arteries indicate that collateral circulatory mechanisms are generally available. If the supply from one or even several nutrient arteries is interrupted, an uninterrupted circulation is ensured in most cases. If extensive occlusion involves the intraneural ramifications, however, the establishment of effective collateral circulations could be impaired or arrested. In this way prolonged local ischemia may result in damage to nervous tissue.

Anatomical studies of the pattern of blood supply provide a basis for physiological studies but cannot furnish conclusive evidence concerning the actual amount of blood perfusing the nerve. Further inquiries concerning its functional significance and its role in various physiological and pathological phenomena must be settled by investigation, preferably of an experimental nature, and not by speculations based solely on the anatomical distribution of the vessels.

Ischemic nerve lesions can be classified in non-traumatic lesions (by arterial embolism or occlusion of the arteriae nervorum) and traumatic lesions (compressing and stretching of the nerve trunk, damage to the arteriae nervorum, damage to the main artery to limb or to one of its major branches, or combined neurovascular injury) (RICHARDS, 1951).

From human pathology it is known that in a *non-traumatic ischemic lesion* by peripheral arterial embolism, the peripheral nerve suffers along with all other tissues of the extremity. If the circulation is restored within a few hours by embolectomy or an effective collateral circulation, the involvement does not proceed beyond a transient conduction block. If more permanent ischemic changes develop, a pattern of injury is followed similar to that after traumatic involvement of the blood supply to the nerve.

In non-traumatic ischemic lesions by narrowing or occlusion of the arteriae nervorum, danger is likely to arise when the vessels are involved in a generalized obliterating condition (e.g. diabetes, arteriosclerosis) or when the blood supply of long segments of the nerve depends on a single vessel. The usefulness of compensatory collateral circulatory mechanisms is then limited.

Of the *traumatic ischemic lesions* only the lesions by compression which indirectly interfere with te blood supply to the nerve trunk will be considered.

Ischemic effects in human peripheral nerves have been studied extensively by interrupting the blood supply to a limb by means of a sphygmomanometer cuff inflated to an pressure greater than the systolic blood pressure. Cuff compression, however, introduces two complications; firstly, all tissues are rendered ischemic; secondly, the encircling pressure required to arrest the circulation in the nerve trunk also deforms it and directly injures nerve fibers.

It is now generally agreed that the initial changes produced by localized pressure are due to the associated ischemia, even if later and more pronounced structural changes are the result of the deformation. It is, therefore, difficult to isolate the effects due to compression deformation from those due to ischemia. In most lesions due to compression, localized ischemia at the site of deformation will be an unevitable complication.

Severe and prolonged compression may so damage the blood vessels

in the compressed segment of nerve that, even when pressure is relieved, the circulation may not be restored to the injured section of the nerve which remains ischemic and undergoes further pathological change. There is no sharp line of separation between what is due to deformation and what is due to ischemia.

The nature, severity, and consequences of nerve trunk ischemia depend on the duration of the ischemia and on the collateral circulation. A wide variety of pathological changes, from first to fourth degree of injury, with the addition of certain features peculiar to ischemic lesions may be present. Due to regional variations in blood supply and differences in collateral circulation, the pathological changes are not usually uniform throughout the nerve and form the basis of mixed lesions.

4. COMPRESSION OF PERIPHERAL NERVES

As has been explained before, there is an ischemic component in most compressive lesions since it is impossible to compress a segment of a nerve trunk without simultaneously affecting its blood supply. The histopathological changes represent a combination of those due to direct pressure and those due to ischemia. The effects of compression depend on the following factors:

1. The rate of application of the deforming force and its duration.
2. Whether the force is localized on one point on the surface of a nerve; whether it is applied obliquely or transversely across the nerve; or whether it is applied over a longer distance of the nerve.
3. The magnitude of the force.
4. The regional anatomy: some nerves have anatomical conditions that predispose them to compression ischemia (e.g. the median nerve in the carpal tunnel).
5. The internal structure of the nerve. A nerve is more vulnerable to compression where its fibers are collected into one single funiculus.
6. Injured nerve fibers are more susceptible to artificially induced ischemia than normal nerve fibers.

The phenomena associated with compression ischemia are essentially the same, regardless of the manner in which the nerve is compressed. The compression can be due to external forces (blunt trauma, crushing, spring clips, tourniquet, bandage) or to forces developing and acting internally (callus, fibrous tissue, swelling of tissues, extravasation of blood). Pressure may result in acute cessation of nervous activity or lead to a gradual and progressive deterioration of function.

Tourniquet lesions may vary in severity from first to third degree involvement (SPEIGEL and LEWIN, 1945).

In these lesions motor fibers seem to be more susceptible to compression (ALLEN, 1938b). Recovery is usually spontaneous and complete after a few months. There is no constant relationship between the length of time

for which a tourniquet is applied and the involvement of the nerve.

The lesions resulting from compression ischemia may be grouped into three main categories, though mixed varieties are common. The first category consists of transient conduction block. The cessation of nerve function may last for seconds, minutes, or hours and is followed by a complete and rapid recovery on release of pressure. The various functions return in reverse order to that in which they failed.

In the second category there is prolonged conduction block with delayed (for days or weeks) but complete recovery on release of pressure. The nerve trunk becomes narrowed at the site of constriction, or flattened if compressed against an unyielding surface. Axons are interrupted within their endoneurial sheaths and undergo Wallerian degeneration (second degree of injury). The circulation in the vessels is slowed by compression of the interneural vessels. The veins are the first to suffer. Obstruction results in hyperemia and edema of the affected segment of the nerve trunk and adds to the pressure deforming the nerve fibers. The ischemia is responsible for an increase in the connective tissue which is converted into fibrous tissue by fibroblasts multiplying in the protein exudate. More resistant fibers may still be conducting normally but in most of the surviving thinned fibers conduction velocity is reduced (THOMAS and FULLERTON, 1963). If the compression is only arrested but not relieved, the lesion enters a chronic phase with increasing fibrosis. The nerve becomes swollen and hyperemic proximal to the lesion as a result of the obstruction of vascular flow in the longitudinal intraneural vessels and of endoneurial fluid, the accumulation of edematous exudate, and an increase in connective tissue. Distal to the site of compression the nerve trunk regains more normal dimensions.

In the third category Wallerian degeneration followed by regeneration occurs. The regeneration and recovery depend on the extent to which the pressure and ischemia have disorganized the structure of the nerve trunk. If the endoneurial sheath is preserved, regeneration results in complete but delayed return of the original pattern of innervation (second degree of nerve injury).

Destruction of the endoneurium with disorganization of the intrafunicular tissues and, finally, disorganization of the internal architecture of the nerve trunk represent third and fourth degree nerve injuries. Regeneration is so seriously affected by extensive fibrosis and marked collagenization that recovery is incomplete or negligible.

5. SURGICAL NEUROLYSIS

Neurolysis involves freeing the nerve trunk from constrictive adhesions and scar tissue which obstruct the growth of regenerating axons or block conduction in the nerve fibers.

In external neurolysis the entire nerve trunk is separated from the scar tissue by dissection; in *internal neurolysis* release of the individual funiculi

from interfunicular tissue is attempted. Internal neurolysis can be performed in two ways. The thickened epineurium can be incised longitudinally in order to relieve compression on the contained funiculi. This procedure can be extended by dissecting the perifunicular scar tissue from around individual funiculi. Great care must then be taken to avoid damage of the very fine interfunicular communications and also to the interfunicular vascular network, since both hemorrhage and ischemia could induce further scarring.

Another method consists of interfunicular injections of warm saline solution. It is uncertain whether this method is useful in breaking down constricting scar tissue.

Internal neurolysis is of no value in third degree injuries: the intrafunicular scarring causes destruction of some axons and blocks the regeneration of others. Under these conditions it may aggravate the lesion by destroying surviving fibers. Opening of the perineurium is only justified in cases of involvement with the leprosy bacillus where the inflammatory process is confined within the resistant perineurium and the intrafunicular pressure gradually increases to a point where the nerve fibers contained in the funiculi are compressed. The symptoms are often alleviated by incising the sheath of the entire nerve and the perineurium of the individual bundles.

Many of the dramatic recoveries claimed for neurolysis are due to the mistaken belief that the elapsed time for the return of spontaneous recovery is inevitably short, and that the delayed onset is evidence of a mechanical block.

Credit for the neurolysis can be claimed if recovery follows so quickly after the operation that it cannot be explained by the growth of axons to the periphery.

These cases may be complicated first degree injuries which show no recovery from a single conduction block until the agent responsible for the conduction block has been removed, or second degree injuries in which the axons have already regenerated to the periphery but where the conduction is still blocked.

Credit can also be claimed when spontaneous recovery is long overdue at the time of neurolysis and a further delay then appears which is consistent with the growth of axons to the periphery following their release from constricting scar tissue.

When neurolysis is followed by orderly reinnervation of the periphery occurring too early to be accounted for the regeneration of axons released from constriction, the neurolysis must be regarded as having been premature and coincidental to spontaneous regeneration. When, however, the normally proceeding recovery is suddenly arrested or reversed, the nerve should be freed from constricting scar tissue without delay.

In simple first degree injury there is no place for neurolysis. In complicated first degree injury removal of the constriction is followed by

immediate and full recovery. In second degree injuries occasionally internal neurolysis will be indicated. In third degree injuries the considerable scarring inside the funiculi is inaccessible. Internal neurolysis is impractible under these circumstances. In fourth and fifth degree injuries where the nerve is repaired by suture or grafting, the efficacy of neurolysis following an unsatisfactory recovery will depend on the state of the suture line and the surrounding tissues.

In general, the time required for full restoration of nerve function is measured in months after a first degree injury and usually extends into the second year after second degree damage.

1 – 4. DEFINITION OF THE PROBLEMS

This study of the facial nerve in the cat and of the pathology of the facial nerve in man will deal with the following problems:

1. Is it possible to analyse the nervous activity of the facial nerve under stable experimental conditions during a number of hours?
2. What are the characteristics of its normal compound action potential?
3. What is the influence of a surgical decompression procedure on the function of the nerve?
4. What is the influence of ischemia on the nerve?
5. What is the influence of intraneural pressure on the function of the nerve?
6. What are the possible implications of the experimental results for the treatment of ischemic paralysis in man?

PART TWO

CHAPTER II

THE FACIAL NERVE ACTION POTENTIAL

II – 1. INTRODUCTION

Although recording of nerve action potentials of peripheral nerves is a routine procedure in neurophysiology, it was necessary to develop an experimental setup for in situ recording in the middle ear of the cat permitting also microsurgery on the temporal bone. The setup was designed to permit stable recording for very long periods. The compound action potential recorded from the nerve will be discussed in detail.

Some of the data in Chapters II and III have been previously published (DEVRIESE, 1972).

II – 2. MATERIALS AND METHODS

1. ANIMALS AND OPERATIONS

Mature cats weighing between 1.6 and 3.2 kg were used. The animals were anesthetized by injecting 25 mg sodium pentobarbital per kg body weight (Nembutal® 30 mg/ml) intraperitoneally. The left femoral vein was cannulated with a small-bore catheter (Intracath® No 1617) in order to administer saline solution and additional anesthetic throughout the experiment. A tracheotomy was performed to prevent aspiration of saliva, blood, and irrigating fluid during surgery on the middle ear. Without this precaution, removal of the tensor tympani muscle and enlarging of the middle ear cavities may result in aspiration through the Eustachian tube.

In order to maintain a good general condition in the cat, an electrically heated cushion was placed on the operating table. In addition an infrared bulb was suspended at about 50 cm above the animal's head; by this procedure excessive cooling during nerve dissection and preparation of the electrodes was avoided. The body temperature was checked rectally. The temperature was kept between 37 and 39° centigrade.

Operative technique

The animal was fixed in a headholder with lateral supports to acquire absolute immobilization necessary for stable recording during hours. The best position for presentation of the experimental field was obtained by turning the headholder in three angles relative to the operating table (Fig. II – 1).

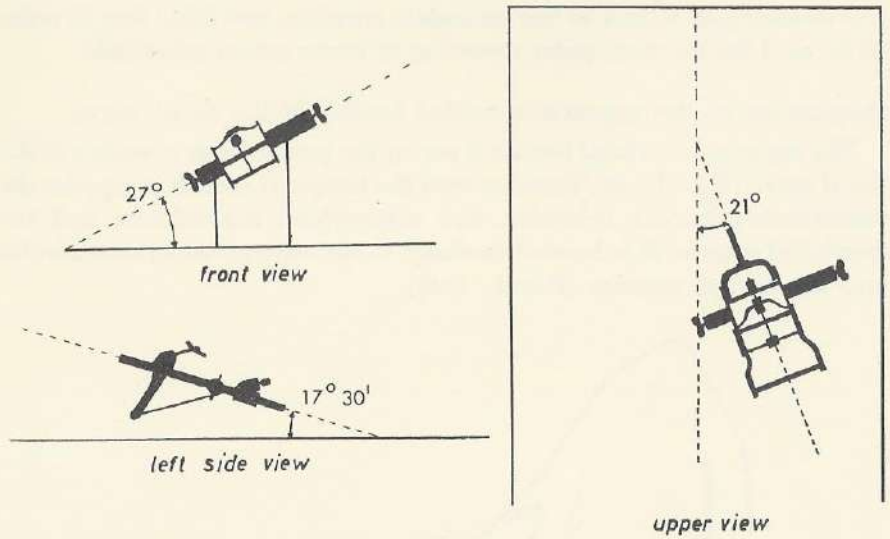

Fig. II–1. Positioning of the modified headholder in relation to the operating table.

The skin incision was initiated posteriorly and superiorly to the right auricle and extended anteriorly to the right external canthus. After identification of its inferior limit the temporal muscle was loosened from the temporal bone and the zygomatic arch. Then skin and muscle were retracted and fixed upwards by means of nylon ligatures and hemostatic clamps across the surgical field. Amputation of the auricle was performed and the cartilage of the external auditory canal fixed sideways; its superior part was removed. The attic was opened with a small cutting burr. The tympanic sulcus laterally to the head of the malleus was destroyed in order to remove the malleus, incus, and tensor tympani muscle. The facial canal could now be seen in its intratympanic course. This permitted safe removal of the bone lateral to the canal. The enlargement of the middle ear cavity was necessary in order to facilitate the placement of the recording electrode. Finally, careful homostasis was performed, and the cavity was filled with Locke's* solution to prevent exsiccation.

Preparation of the facial nerve in the middle ear.

The bony facial canal was thinned with diamond burrs under frequent irrigations with Locke's solution (at 38° C). The intratympanic part of the nerve was exposed up to the geniculate ganglion to provide a piece of nerve as long as possible. When the canal was opened, the bone around the nerve was further removed with dental excavators. The facial nerve

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* Composition of Locke's solution: NaCl 9 g, KCl 0.42 g, $CaCl_2 \cdot 0H_2O$ 0.24 g, $NaHCO_3$ 0.2 g, aqua dest. ad 1l. Before use 1 g dextrose is added (EYZAGUIRRE, 1960).

was severed just lateral to the geniculate ganglion and lifted free in order to be used for the monopolar recording of nerve action potentials.

Preparation of the zygomatico-orbital branch of the facial nerve.

The zygomatico-orbital branch is one of the preauricular branches of the facial nerve (Fig. II – 2). Together with the temporal branch it supplies the preauricular, frontal, orbicular, and supraorbital musculature and the nasolabial muscle. It is located medially to the anterior superior auricular and the frontal muscles (PAPEZ, 1929).

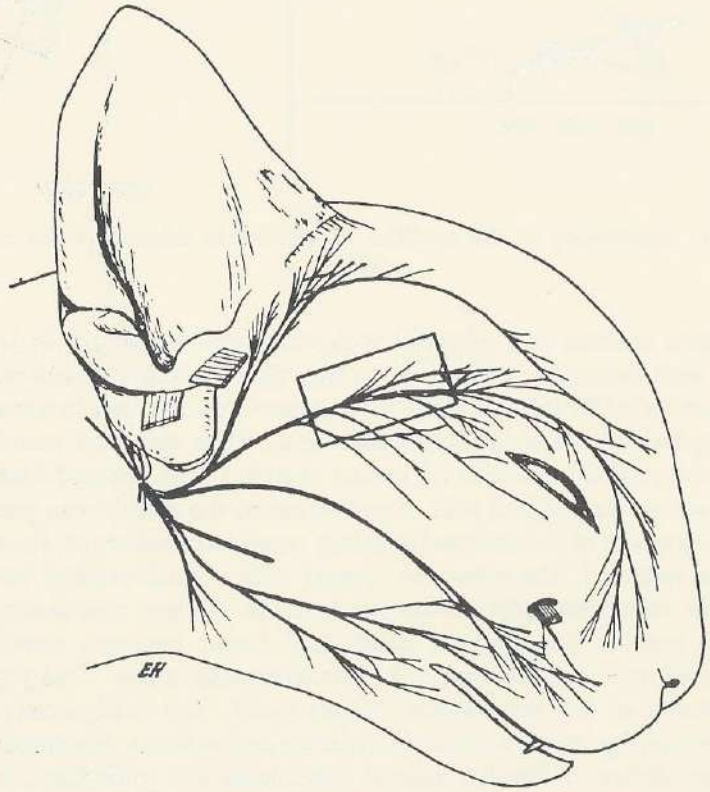


Fig. II–2. Part of the zygomatico-orbital branch of the facial nerve used for electrical stimulation. (Modified from HUBER, E. and W. HUGHSON, 1926).

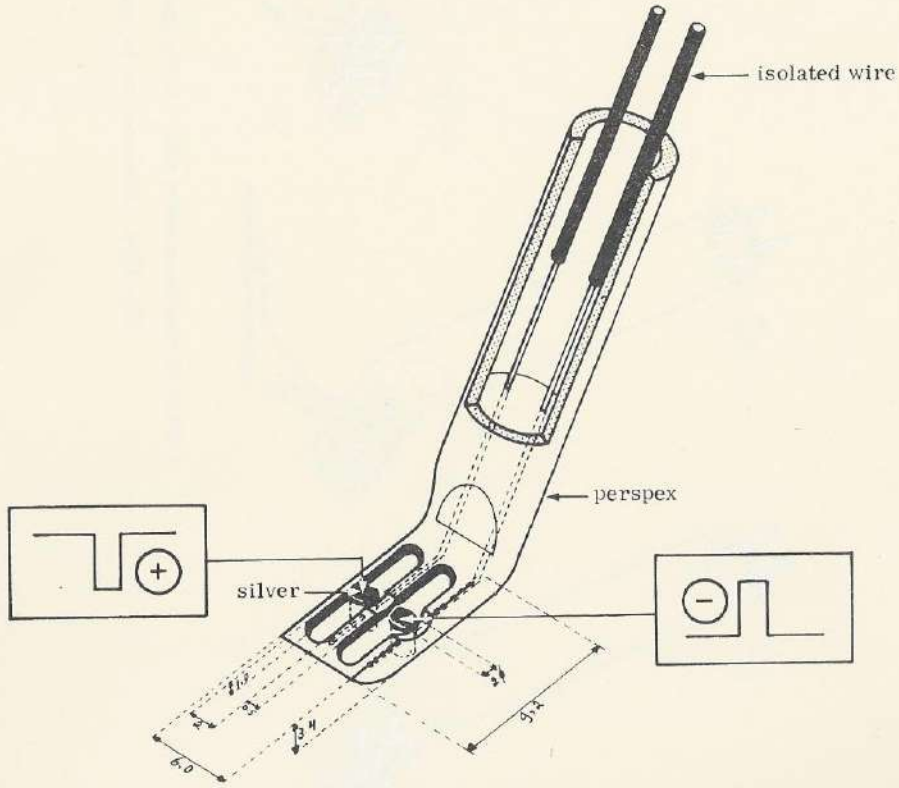
Very carefully, to prevent stretching, the nerve branch was loosened from the surrounding tissue and cut at about 0.5 cm from the eye. The connective tissue was removed as far as possible without damaging the perineurium. The nerve branch was freed in the direction of the ear until a preauricular artery and vein were crossed. More proximal dissection was hazardous because of the anatomical relationship of this branch with the parotid gland. The prepared part of the nerve was used for bipolar stimulation. The length of the nerve between stimulating and recording electrodes (conduction distance) was about 5 cm (± 0.5 cm). Its length inside the temporal bone is about 6 mm. The freed end in the middle ear was about 3 mm and the extra temporal part about 4.2 cm.

Description and placement of the electrodes

Stimulating electrode.

This electrode consisted of two pieces of silver incorporated in a block of perspex. The silver contacts were separated by a small bridge (diameter 0.5 mm) and laid on the bottom of two excavations designed in the perspex to prevent sliding of the agar-moulds (see later) around the nerve (Fig. II – 3). The stimulating electrode fixed on a three axial micromanipulator (NARISHIGE) was moved into a position where the zygomatico-orbital branch could be placed on the electrode without stretching the nerve (Fig. II – 4).

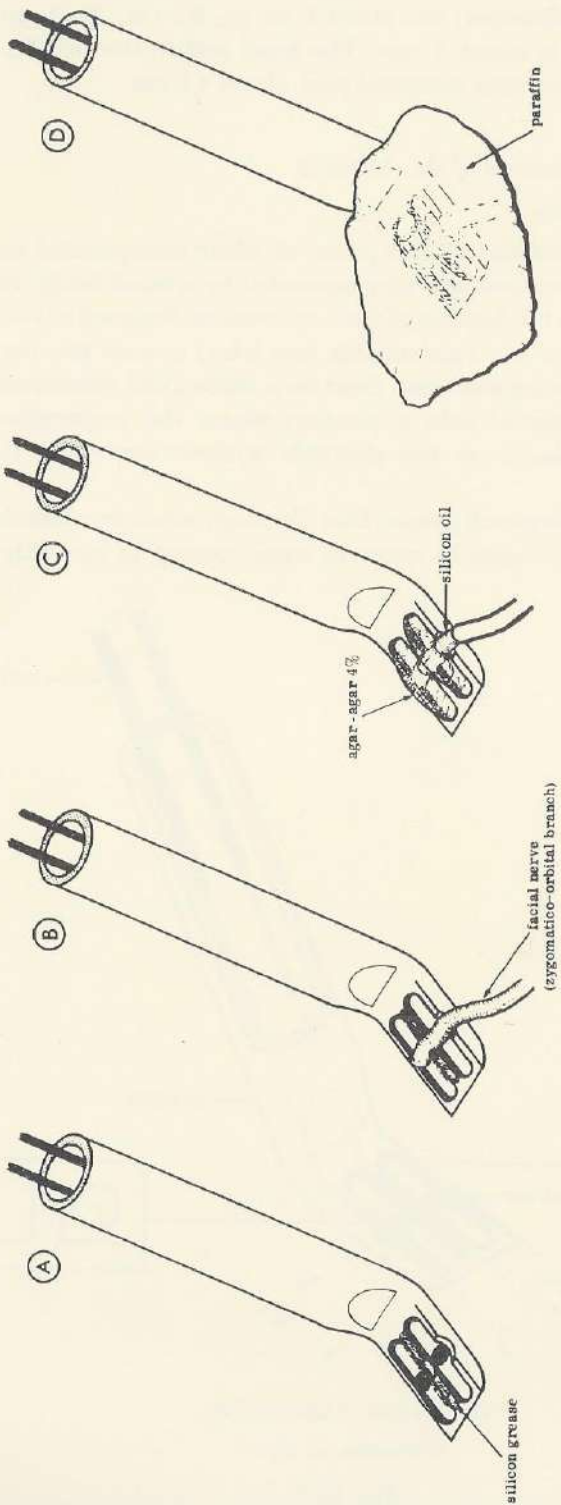
a. Silicon grease (stopcock grease, Dow Corning), was spread on the bridge between the two contacts in order to avoid oozing of electrolytic fluid



STIMULATING ELECTRODE

(distances in mm)

Fig. II–3.



STEPS IN PREPARATION OF NERVE BRANCH ON THE STIMULATING ELECTRODE

Fig. II–4. Explanation in text.

between the two electrodes during the experiment. Otherwise impedance changes would have endangered the stability of stimulation.

b. The nerve was placed on the electrode in such a way that contact with both pieces of silver could be made. Any trauma to the nerve (angulation, crushing, stretching) was carefully avoided.

c. Drops of a 4 per cent solution of agar in physiological saline were moulded around the nerve at the points of contact. After dissolving at 80° C the solution was cooled down to about 40° C. The viscous agar solution was distributed with a syringe and a short polythene cannula perpendicularly over the nerve at the level of the silver electrodes. The agar solution congealed quickly below 40° C and fixed itself around the nerve in the excavations on both sides of the perspex bridge. Should some of the agar cross the bridge – making electrical contact between the two electrodes – it could easily be removed peacemeal after congelation. The agar drops provided electrical contact with the circumference of the nerve at the stimulating points. Therefore the stimulus was delivered more uniformly to the nerve and the stability of the experimental setup enhanced. A few drops of silicon oil (Silicone defoamer Type S5109 Radiometer, Copenhagen) along the freed part of the nerve, not in contact with the stimulating electrode, prevented capillary migration of electrolytes along the nerve.

d. Finally, the electrode and the prepared nerve were completely covered with paraffin at 39° C (melting point of the paraffin 49–50° C; congelation point 39–40° C). This method proved effective for electrical, thermal, and, especially, mechanical stabilization during the experiments.

Recording electrode.

In our experimental setup, it was not possible to record monophasic action potentials in the way described in Chapter I. Therefore an alternative method was used which made possible the recording of monophasic action potentials. The physical and physiological conditions, however, are not so well defined.

The electrode (Fig. II – 5) consisted of a glass tube filled with Locke's solution. A silver wire, 1 mm in diameter, made the connection with the amplifier. The opening where the wire perforated the glass was sealed with a contact adhesive ("Snelfix®" manufactured by Cetabever, Beverwijk, Holland). The system was closed on one side with a rubber tube, clamped with a hemostat, and lowered by a micromanipulator into the middle ear which contained the same solution. Air bubbles in the electrode (preventing electrical contact) were evacuated by pressing the rubber tube while the tip of the cannula was immersed in the fluid inside the middle ear. Releasing the rubber tube produced an underpressure which, together with moving the electrode towards the nerve, introduced about 3 millimeters of the nerve into the glass tube.

The remaining liquid was removed by suction. A few drops of silicon oil assured further electrical isolation around nerve and cannula. The

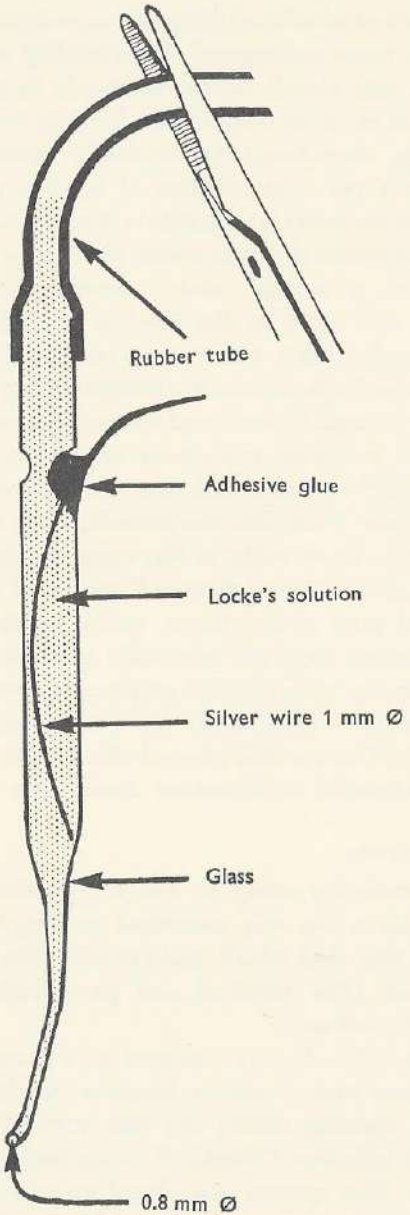


Fig. II–5. Recording electrode.

final step in this preparation was fixation of both electrode and nerve with paraffin in the same way as on the stimulating side.

This electrode recorded the action potential against an indifferent electrode in the immediate neighborhood which consisted of a pointed silver wire (diameter 1 mm) placed in the subcutaneous tissue posterior to the ear.

The piece of nerve inside the electrode included the severed end of the

nerve and a few millimeters of intact nerve. The recorded action potential is called monophasic since the nervous activity was recorded from one site of the nerve (the glass electrode) while the reference electrode (the silver wire) was assumed not to be influenced by the nerve action potential. The recorded action potential is the summation of the potential fluctuations at the last intact nodes of Ranvier inside the recording electrode.

2. Recording procedures

Stimulation

A constant current stimulus was delivered to the nerve by means of an electric configuration as described in Figure II – 6. A wave form generator which determined the stimulus frequency started the stimulus sequence; it triggered the recording and processing instruments and also a second wave form generator. The output of the second wave form generator was fed simultaneously into two pulse-generators which had different amplitude settings. By means of switch S1 both were connected to a stimulus isolation unit. The stimulus isolation was necessary to prevent ground coupling between stimulating and recording systems. The stimulating electrodes were connected to the stimulus isolation unit in series with a 1 kΩ resistor, the potential difference over it being used as a measure of the stimulating current, and a 10 kΩ resistor to achieve an approximate constant current stimulation. In every experiment the distal electrode was positive against the proximal one.

The stimulus parameters were determined as follows:

1. *Frequency*

The frequency of stimulation was based on the results of a series of preliminary experiments (see results). It was fixed at 10 per second which permitted recording of 500 action potentials in 50 seconds.

2. *Duration*

The duration of the stimulus was calculated from a strength-duration curve at the beginning of each experiment; it was fixed at twice the chronaxy. It is realized that the definitions of rheobase and chronaxy apply only in approximation for nonhomogenous nerves.

3. *Amplitude*

Three different amplitudes were determined as follows: a *submaximal* stimulus producing 50 to 80 per cent of the maximal amplitude of the action potential, a *maximal* stimulus giving the maximal amplitude and a *supramaximal* stimulus of at least 6 times the rheobase. The stimulus could be made visible on the oscilloscope by means of switch S2.

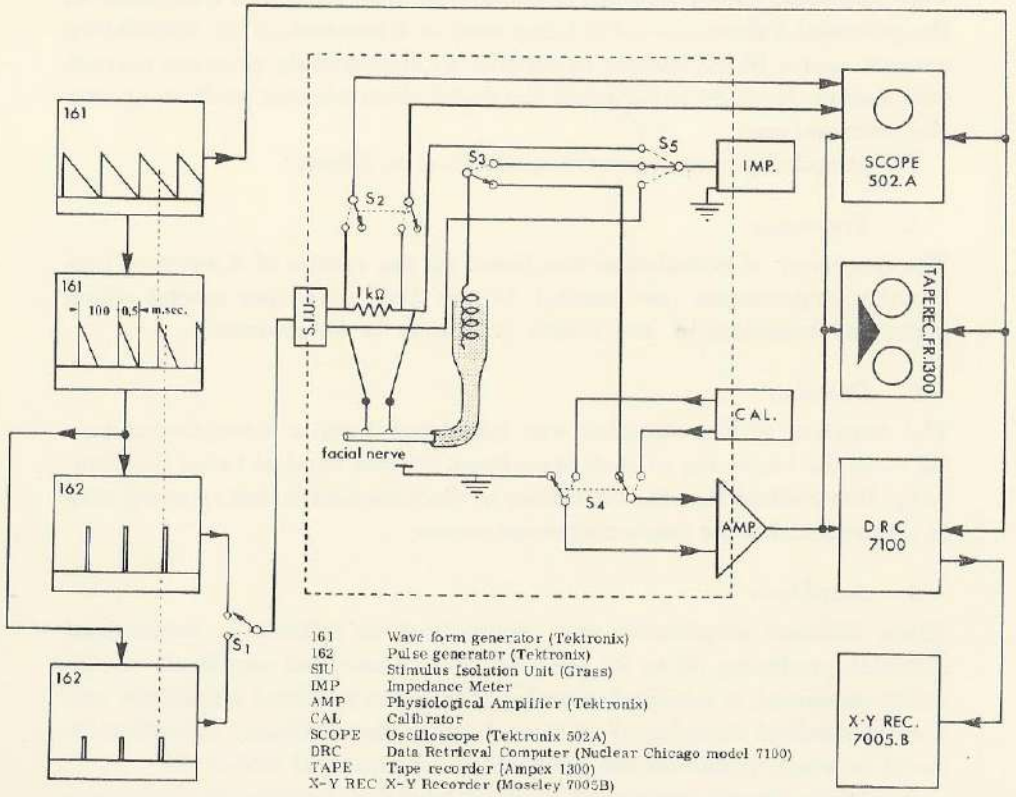
It is of the utmost importance to stress here that antidromic stimulation of the motor fibers of the facial nerve was used. This considerably simplified the experimental setup. By doing so, movements of facial muscles pro-

voked by stimulation of other motor branches were avoided which would have resulted in movements of the electrodes. In this way artificial ventilation could also be circumvented.

Recording

The monophasic action potential in the sectioned facial nerve was recorded by the previously described glass electrode placed inside the middle ear (Fig. II – 5). The indifferent electrode was placed in the subcutaneous tissue posterior to the ear. Its best location – with the smallest stimulus artifact – was found by observation on the oscilloscope. Baseline deflections between artifact and action potential as well as those in the tail of the compound action potential were often almost completely compensated by this procedure.

The action potential was amplified 1,000 times and fed into the oscilloscope for monitoring, the tape recorder for recording, and the special purpose computer for immediate processing. Calibration of amplification was accomplished by means of switch S4, while switch S3 and S5 were necessary to measure the impedance of the electrodes (Fig. II – 6).



BLOCK DIAGRAM OF THE EXPERIMENT

Fig. II–6.

The experiments were performed inside an electrically shielded room; the animal, the shielding of the electrodes, the micromanipulators, the headholder, and the amplifier were all connected to a common ground to avoid ground loops.

Processing

Processing during the experiments.

The amplified nerve action potential together with the trigger pulse of the first wave form generator were recorded on two channels of a tape recorder (tape-speed 30 inches per second) for processing at a later date. An additional oscilloscope was used as a check on the recording. The action potential was also fed into an averager (Data Retrieval Computer, Nuclear Chicago 7100).

Five hundred consecutive action potentials were used for the analysis of nerve function. The first 3.75 or 7.5 msec following the trigger pulse were averaged. The averaged action potential was then displayed by an X-Y recorder (Moseley, 7005 B). Calibrations were processed in the same way.

Off-line processing.

After completion of the various series of experiments the collected data were fed into a digital computer (PDP-9 Digital Equipment Corporation, Maynard Massachusetts) for calculation and display of the results. More details are given in the Appendix. The different steps in processing were:

1. Analog-to-digital conversion and data reduction.

Each observation consisting of 500 consecutive action potentials and the calibrations (200 or 500 microvolts) recorded on magnetic tape were converted and averaged, and the averaged curve was stored on magnetic data tapes (Dectape) for further processing.

2. Processing of the stored data.

Six parameters were calculated for every averaged action potential (Fig. II – 7):

(1) The maximum amplitude of the nerve action potential in microvolts.

(2) The maximum amplitude of the derivative (with respect to time) of the action potential in microvolts per millisecond. This parameter was calculated in order to obtain information about the fastest conducting fibers.

(3) The "surface" of the action potential here, defined as the square root of the sum of the squared values, was expressed in microvolts. It was calculated in this way to include the overshoot occurring sometimes in the tail of large action potentials.

As demonstrated by ERLANGER and GASSER (1937), the surface of the recording covered by a compound action potential that is monophasically

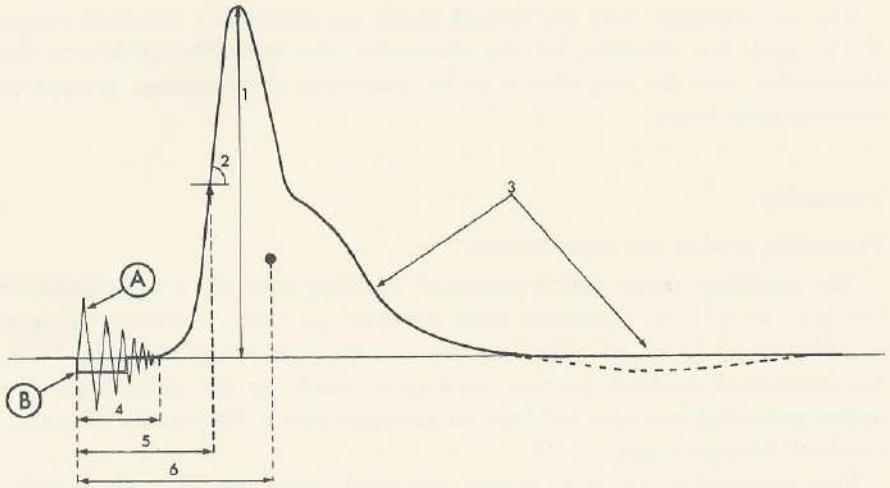


Fig. II–7. Parameters of the action potential and modification of the stimulus artifact.

1. Maximum amplitude (microvolts).
2. Maximum amplitude of the derivative of the action potential (microvolt per millisecond).
3. "Surface" (square root of the sum of the squared values in microvolts).
4. Latency time from onset of stimulation to onset of action potential (microseconds).
5. Latency time from onset of stimulation to maximum amplitude of the derivative of the action potential (microseconds).
6. Latency time from onset of stimulation to gravity point of the surface of the action potential above the baseline (microseconds).

A. Original artifact – B. Modified artifact.

recorded from a nerve trunk remains constant irrespective of the conduction distance.

(4) The latency time from the onset of the stimulus artifact to the onset of the action potential in microseconds. This latency is related to the conduction velocity of the action potential and to the conduction distance.

(5) The latency time from the onset of the stimulus artifact to the moment of the maximum amplitude of the time derivative of the action potential in microseconds. This is assumed to be related to the conduction velocity of the fastest fibers of the stimulated nerve branch.

(6) The latency time from the onset of the stimulus artifact to the center of gravity of the "surface" of the action potential above the baseline. This parameter was studied to detect changes in conduction velocity of the nervous activity as a whole.

The measures as defined here were chosen in preference to others mainly because of their simple computability. Other measures would most probably have lead to identical conclusions.

3. Display of the parameters.

The six parameters were displayed on a storage oscilloscope (Tektronix 611). The calibrations of 200 or 500 microvolts were used as references for the amplitudes. Corrections were made for calibrations differing more than 5 per cent.

4. Calculation of the parameters.

First all amplitudes were taken in reference to the calibrations. Then these data were used for statistical analysis.

5. Three-dimensional display of the experiments on a storage oscilloscope and plotter (Complot, Houston Instruments). The first curves appear in the right or left upper corner and the last ones in the left or right lower corner (horizontal axis in milliseconds, vertical axis in microvolts and oblique axis in hours). Since the orginal artifact disturbed the display of the action potentials, it was necessary to modify the stimulus artifact for this purpose. A uniform artifact was programmed for every curve, the duration being the same as the actual stimulus duration.

3. Histological techniques

Histological examination was performed in seven cats; two animals were sacrificed for study of the normal facial nerve and five were studied after the performance of a pressure experiment on the nerve (see Chapter V). At the end of the experiments the fixation process was initiated by dripping Susa fixative (Romeis, 1968) with a pipette in the stylomastoid foramen, with the facial nerve and the needle inside it, for about 15 minutes. In the meantime the head of the animal was removed in order to trim down the temporal bone with rotary saw blades to suitable dimensions for histological processing. The fixation was continued for 4 hours in the same fixative solution. The blocks were then dehydrated in an alcohol series, embedded in Rallwax 1 (Lamb, London) and decalcified in 5 per cent nitric acid in a saturated solution of picric acid (70 per cent ethanol). Sections, parallel to the base of the skull, were cut at 6 μ in a cranial-caudal direction and stained with hematoxylin-phloxin and Mallory-Azan.

II – 3. RESULTS

Frequency of stimulation

In a series of preliminary experiments the frequency of stimulation was varied, since too frequent stimulation of the nerve in order to record a series of action potentials could possibly decrease the excitability of the individual axons. Therefore, continuous stimulation up to one hour with a frequency of 1 per 10 seconds and of 1, 5, 10, 20 and of 50 per second, followed by a period of rest, was performed. Frequent stimulation of the nerve (e.g. 20 or 50 times per second) resulted in a decrease in amplitude of the action potential; the latency time seemed not affected. After a period of rest the amplitude of the action potential recovered. Finally, a stimulus frequency of 10 per second was chosen for the experiments. It permitted recording of 500 action potentials every five minutes without overt decrease in amplitude (Fig. II – 8).

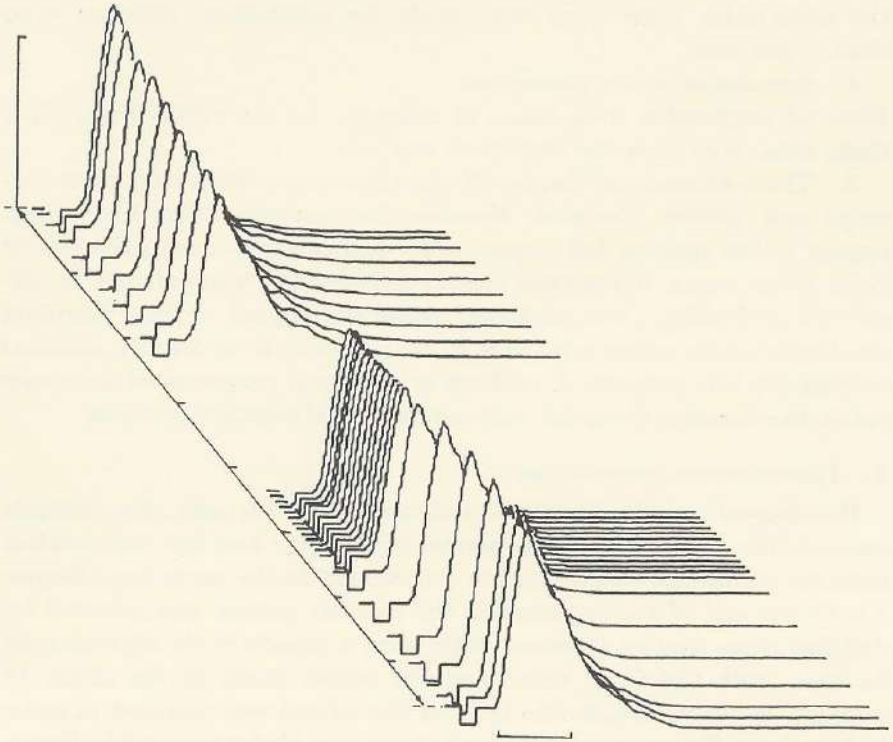


Fig. II–8. Control experiment. In this and all similar Figures, the first curve of the experiment is displayed in the left or right upper part of the Figure, the last curve in the right or left lower part. Each curve represents 500 averaged action potentials. – Calibrations: 250 μV (vertical bar) and 1 millisecond (horizontal bar). The time (oblique axis) is indicated in hours. Note the uniform stimulus artifact and the recording of action potentials every five minutes (top of the lower part of the Figure). Experiment 54-SD (stimulus duration): 210 μsec-SI (stimulus intensity): 400 μA-MA (maximal amplitude in the experiment): 284 μV-RT (total recording time of the experiment): 7 h. 35 min.

Contact between nerve and electrodes

Throughout the experiments the impedance of the respective electrodes was checked by means of turning on switches S3 and S5 (Fig. II – 6). Provided there was a good contact with the nerve the impedance between the stimulating electrodes was 0.9 to 3.2 kΩ. Between the indifferent and the recording electrode it was 17 to 51 kΩ. In case of a strong increase of impedances, the contact with the nerve had to be restored.

Stimulus strength

In Fig. II – 9 nerve action potentials provoked by increasing suprathreshold stimulations are represented. The stimulation strength of the lower curve was 70 microamperes, the second curve 90 microamperes, the third 100 microamperes and the upper curve 300 microamperes (Stimulus duration: 200 microseconds; stimulus frequency: 10 per second). The rheobase of this nerve branch was 32.5 microamperes. It can be seen that – for increasing stimulus strengths – the action potential becomes higher, broader, and faster. At 100 microamperes an elevation in the descending part of the action potential appears; at supramaximal stimulation (300 microamperes), which results in a maximal action potential, the elevation is most distinct. In some cats even more elevations appeared by increasing the stimulus intensity.

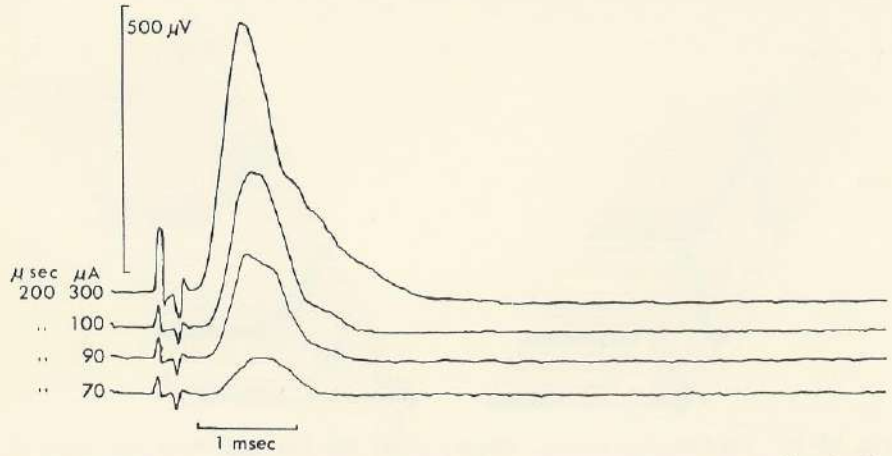


Fig. II–9. Nerve action potentials provoked by increasing suprathreshold stimulations. The stimulus intensity and duration are indicated on the left side of the curve. The stimulus frequency was 10 per second in all cases. The rheobase was 32,5 microamperes.

Stability of the setup

The stability of the setup was judged in seven control experiments at random, interspersed among the different series of experiments and by observation of action potentials after reversible nerve blocks. In Figure II–10 a control experiment is represented. General anesthesia was started about three hours before the first recording in the upper part of the Figure. The last recording was done nine and a half hours later. In Figure V–10 the action potential after 4 periods of pressure block is similar to that recorded six and a half hours before.

The maximal nerve action potential

For description of the maximal nerve action potential and as a basis for further observations, the "normal" values were calculated in 36 cats. Since the maximal amplitude seemed to be most easily influenced by the condition of the animal, injury to the nerve, etc,. it was used to select the experiments for calculation of these normal values. Experiments were rejected when stability of the action potential was not obtained after preparation of the nerve on the electrodes or when the nerve potential was

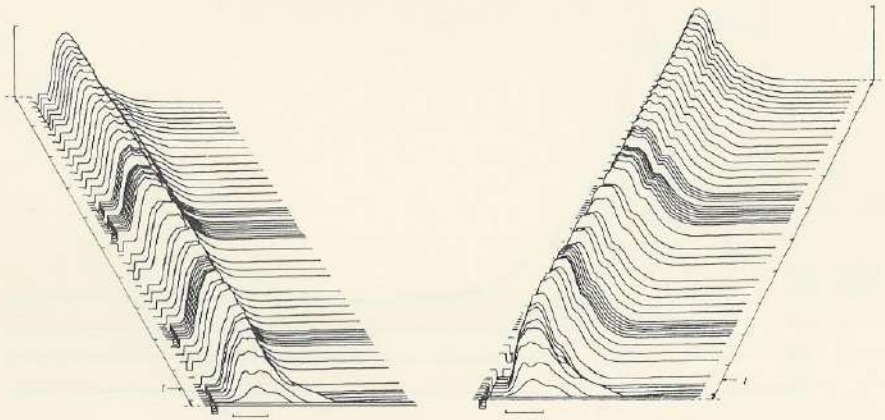


Fig. II–10. Control experiment. Display from the left and from the right to expose the ascending and descending parts of the action potential. General anesthesia started about 3 hours before the first recording (in the upper part of the Figure). The last recording in the living animal was done nine and a half hours later. I (ischemia) indicates the arrest of the circulation.
Exp. 89 – SD: 150 μsec – SI: 800 μA – MA: 205 μV – RT: 10 h. 12 min.

presumed to be too low because of trauma to the nerve. In Table II–I six parameters are represented; they are expressed in medians and ranges. In Figure II–11 histograms of latencies and amplitudes are given.

The correlation between the latencies and the amplitudes of the action potential were calculated and are represented in Table II–2. The amplitudes were highly correlated to each other, as were the latencies. There proved to be no linear relationship between the latencies on the one hand and the amplitudes on the other hand.

II – 4. DISCUSSION

1. Method

The setup as described in paragraph 2 of this chapter proved effective to study facial nerve action potentials in vivo. In our opinion the preparation of the nerve with an agar-solution, the isolation with silicon-oil and

Table II–1. Latency times and amplitudes of maximal nerve action potential by stimulation of the zygomatico-orbital branch of the facial nerve in 36 cats.

LATENCY	Median	Range
to onset of action potential	425 μsec	300– 625 μsec
to maximal slope of action potential	687 μsec	512–1112 μsec
to gravity point	1242 μsec	922–1764 μsec
AMPLITUDE		
of action potential	325 μV	168– 952 μV
of "surface"	151 μV	77– 429 μV
of time-derivative of action potential	61 μV/msec	24– 204 μV/msec

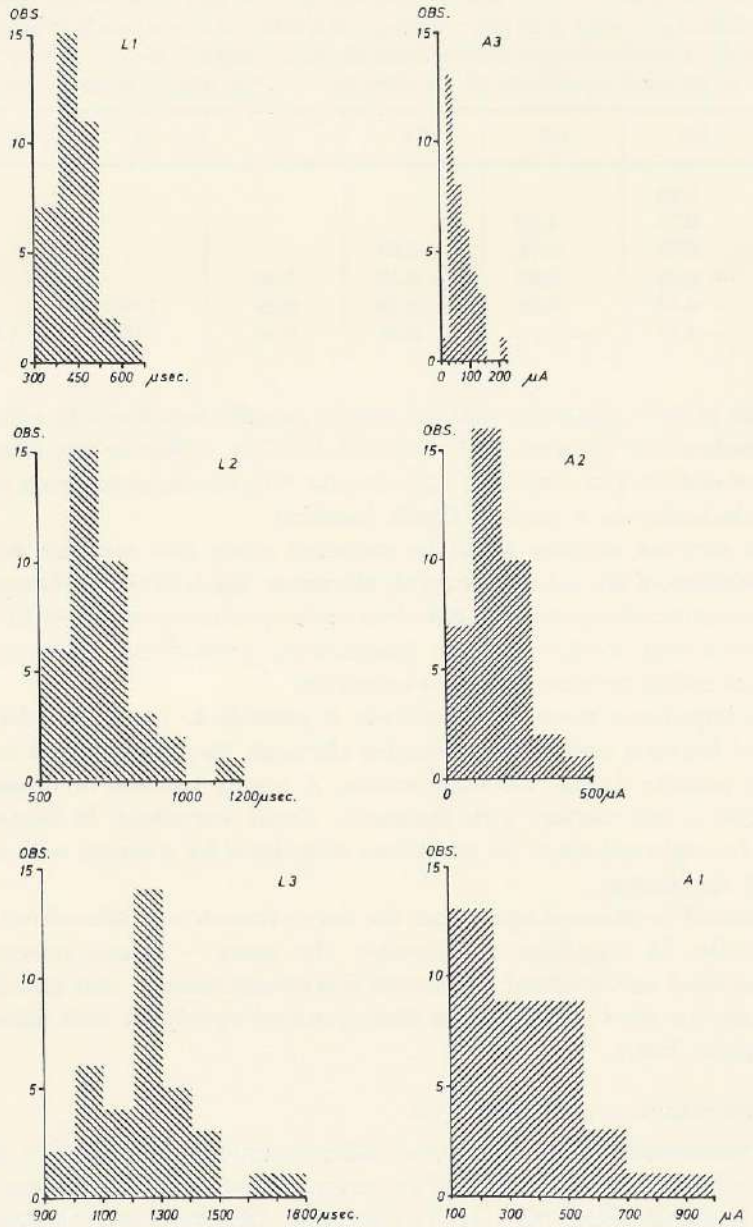


Fig. II–11. Distribution of latencies and amplitudes of the facial nerve action potential in 36 cats. Latency to the onset of the action potential (L_1), to the maximum of the derivative (L_2), and to the center of gravity (L_3). Amplitude of the maximum of the action potential (A_1), of the "surface" (A_2), and of the time-derivative (A_3).

TABLE II–2. Correlation matrix of latencies and amplitudes of the action potential of the zygomatico-orbital branch of the facial nerve in 36 cats.

Latency to onset of nerve action potential (L1), to maximum of the derivative of the action potential (L2) and to center of gravity of the action potential (L3); amplitude of maximal nerve action potential (A1), "surface" of the action potential (A2) and maximal amplitude of the derivative of the action potential (A3).

	L1	L2	L3	A1	A2	A3
L1	1.00					
L2	0.73	1.00				
L3	0.72	0.74	1.00			
A1	− 0.03	0.06	− 0.13	1.00		
A2	− 0.07	0.00	− 0.13	0.99	1.00	
A3	− 0.11	− 0.16	− 0.31	0.94	0.93	1.00

fixation of both electrodes and nerve with paraffin considerably enhanced the mechanical, thermal, and electrical stability of the setup. (Stability was not obtained in a series of experiments with the facial nerve on copper wire electrodes in a pool of liquid paraffin).

The nervous activity could be recorded every five minutes without deterioration of the action potential. Moreover, the technique of averaging five hundred action potentials per observation produced curves undisturbed by interfering electromagnetic phenomena, even those provoked by hospital calling systems based on induction.

The impedance measurements made it possible to check the electrical contact between nerve and electrodes although they were buried in congealed paraffin during the experiments. A strong increase of impedance indicated a lost contact with the nerve. Small variations in impedance could be neglected since the nerve was stimulated by a source with a high output impedance.

It should be stressed again that the nerve branch was stimulated antidromically. It simplified considerably the setup – muscle movements were avoided and artificial ventilation was circumvented – and should not influence the results, since nerve fibers conduct equally in both directions (HODGKIN, 1964).

2. THE NERVE ACTION POTENTIAL

As mentioned before, the nerve action potential was recorded monophasically from the distal part of the cut facial nerve in the middle ear by a glass electrode filled with Locke's solution. The reference electrode (a silver wire) was localized at the relatively inactive subcutaneous tissue posterior to the ear. Activity at the glass electrodes was recorded as a negative going variation of the steady demarcation potential and represented by an upward deflection on the oscilloscope.

According to PAPEZ (1929) and FOLEY and DUBOIS (1943) about 1,700 motor fibers are available in the zygomatico-orbital branch of the cat.

The motor fibers are 1.5 tot 13 μ in size, the majority being from 3.5 to 5.5 μ.

The myelinated afferent fibers totaling about 150 (1.5 to 6 μ in size) for *all* the mimetic muscles (BRUESCH, 1944) most probably do not contribute significantly in our setup.

The recorded action potentials can thus be considered as the result of stimulation of A fibers of the motor efferent type only. The stimulation strength was not strong enough and the time base used for the scope display was not long enough to observe possible B or C group fibers.

Shape of the action potential

The conduction distance between the electrodes was short; approximately 5 cm. Consequently, the various elevations in the action potential are grouped in a short deflection (ERLANGER and GASSER, 1937). In most experiments one elevation was observed followed by a smaller one in its descending phase (Fig. II–9). The second elevation is due to a group of smaller, more slowly conducting fibers in the A group which have a higher stimulation threshold. The second elevation is not present in the lowest recording. This was also checked by the method developed by ERLANGER and GASSER (1937) based on the difference of refractory period in different nerve fibers. The nerve is therefore stimulated by two stimuli of different intensities, the first one sufficient to produce the first elevation; the second one produces the first and the second elevation. At progressively shorter intervals of stimulation the first elevation of the second action potential disappears, because of the refractory period, while the second remains unaltered.

It was not possible, however, to differentiate in every experiment between the fiber groups. The conduction distance was too short to separate groups of fibers of different velocity, neither was it possible to record the action potentials in the middle ear by two electrodes from two places of the nerve. Moreover, variations between the individual nerves are likely.

The appearance of more elevations in the action potentials in some cats, by increasing the stimulus intensity, could be explained by the suprathreshold stimulation of neighboring motor fibers. The delay was caused by the longer conduction distance.

Amplitude of the action potential

Theoretically, the compound action potential is formed by the sum of the spikes of the individual axons. During the experiments the amplitudes of the action potential varied strongly, while the latencies did not. These variations in the amplitudes can be attributed to several factors. At the *site of the stimulation* the number of nerve fibers present in the nerve branch, the damage to the nerve during preparation and the number of fibers on

the stimulating electrodes varied. Variations in the *conduction distance* may also have caused differences in amplitudes. The reduction with respect to the theoretically expected amplitude at the *site of recording* must be ascribed to shunting between the cut nerve fibers.

It can be concluded that the setup itself may have strongly influenced the amplitudes and therefore was not well suited to determine the total nerve response. During the experiments on the nerve the amplitudes also proved to be the parameters the most influenced by interference with the nerve. The theoretical nerve response could be better calculated from the fiber composition of the nerve branch. The aim of the experiments, however, was to study influences upon the nervous activity in a stabilized setup.

Conduction velocity

The conduction velocity of the action potential was calculated from the conduction distance (approx. 5 cm) and the latency time. In 36 cats the median value to the onset of the action potential was 425 microseconds, to the maximal ascending slope of the curve 687 microseconds and to the gravity point 1,242 microseconds (Table II–1). All latencies were calculated from the onset of the stimulus artifact. Accordingly, the conduction velocity of the fastest fiber initiating the action potential was 117 meters per second; the fastest group of fibers was 73 meters per second; a complete volley travelled at 40 meters per second. According to CHAMBERS et al. (1970) the conduction velocity of the fastest fibers should correspond to fibers of about 12.5 μ (alpha fibers) in diameter.

Calculated in this way, the fiber diameter falls in the high values given by PAPEZ (1929) and FOLEY and DUBOIS (1943). The latter authors stressed that their values should not be considered standard for all cats and that variations undoubtedly occur in individual animals. In fact the composition of the zygomatico-orbital branch and the fiber size reported are based on measurements in six cats. On the other hand the results of our calculations could be influenced by an overestimation in the conduction distance in our setup. It is difficult to measure the nerve length exactly in the retromandibular fossa and in the parotid gland. Although dispersion exists within the groups of nerve fibers, it was assumed that the dispersion factor in this type of fibers, over this short conduction distance, was too small to induce considerable errors in the measurements of the conduction velocities. During the first recordings of an experiment a positive (downwards) deflection of the baseline often followed the action potential. This was only the case for large action potentials and it could reach up to five per cent of the amplitude of the spike. This deflection came too early in the recordings (at about 2 milliseconds) to be attributed to a positive after-potential. When the action potential itself decreased during the experiment the positive deflection disappeared.

The high correlation between the amplitudes (Table II–2) and between

the latencies suggest that the parameters of the normal action potential can be represented by one amplitude and one latency.

The absence of correlation between amplitude and latency, for instance, may be explained by individual variations from cat to cat in the number of fibers in the zygomatico-orbital branch stimulated during the experiments.

CHAPTER III

DECOMPRESSION OF THE FACIAL NERVE

III – 1. INTRODUCTION AND LITERATURE

Exposure of the facial nerve is nowadays advocated as treatment of facial nerve paralysis in such conditions as Bell's palsy or ischemic paralysis, temporal bone fractures or complicated otitis media. In these cases the bone around the nerve is removed and the nerve sheath slit open; the whole procedure is called "decompression of the facial nerve".

In the clinical literature evidence is given that a decompression can be performed without overt damage to the nerve. This evidence, however, is indirect, since it is based on clinical observations of patients operated upon because of a paralysis of the nerve, or on electromyographical studies.

In this chapter a series of experimental decompressions under direct inspection of the nerve action potential will be described.

1. INTRATEMPORAL DECOMPRESSION IN MAN

The history of surgery of the facial nerve in all its aspects has been extensively reviewed by MIEHLKE (1960). The present chapter is limited to the description of various approaches and techniques of decompression of the facial nerve in the temporal bone.

The earliest approaches are characterized by a *complete mastoidectomy*. The extensive study of BALLANCE and DUEL (1932) on decompression in combination with nerve grafting revived the interest in surgical treatment of facial nerve paralysis. Before them the most important article about the approach to the facial nerve was written by NEY. In 1922 he described a technique to open the facial canal with chisels. The entire mastoid tip and the posterior meatal wall are removed. The lower portion of the nerve is then freed by gently lifting the nerve on a hook and carefully dividing its periostal attachment to the canal. The nerve sheath has to be opened because "most operative lesions in peripheral nerves are not satisfactorily overcome unless the nerve sheath is opened, decompressing the nerve bundles". It is grasped on each side with mosquito forceps and opened with blunt iris scissors. In order to protect the nerve from immediate contact with the bone, a flap of temporal fascia or pericranium is passed under the nerve. Finally, a portion of temporal muscle denuded from its fascia is turned over the nerve in order to protect it from compression.

In 1945 TICKLE reported on a series of 300 decompressions and nerve grafts.

A method to expose the facial nerve in the stylomastoid foramen and in the inferior end of the canal within the mastoid cavity has been described by LATHROP (1948). The digastric ridge and the stylomastoid foramen are identified after removal of the cellular structure in the mastoid tip and exposure of the plate of the sigmoid sinus with an electrically driven drill. According to GUILFORD (1970) a cutting burr in a high-speed air drill is less traumatic to the nerve than a conventional drill.

SULLIVAN (1952) using the same approach incises the connective sheath along the side of the nerve, in order to prevent damage to the longitudinal arterial anastomoses.

KETTEL (1959) first performs a complete mastoidectomy for two reasons: in the first place curretage of mastoid cells later in life would endanger the exposed nerve, and secondly the nerve can be approached more safely after mastoidectomy. He prefers to decompress the facial nerve from the stylomastoid foramen up to the lateral semicircular canal after removal of the tip of the mastoid process. The canal is opened from below upwards by inserting a fine curette just under the edge of the covering plate of bone. In case of Bell's palsy the nerve sheath is split with an iridectomy knife from below upwards; in cases of infection of the ear this is not done in order to avoid intraneural infection. The denuded nerve is finally covered with a piece of amniotic membrane.

KETTEL and JONGKEES (1963) ascribe the progress of endotemporal surgery of the facial canal to the introduction of the operating microscope, better illumination and the electrical drill. They favour a high-speed electrical drill for precise drilling and frequent irrigations in order to avoid thermal lesions to the nerve. They warn against trauma to the vessels of the nerve by dislodging it from the bony canal. The nerve should be decompressed in both directions until a normal looking nerve appears. MIEHLKE (1960) is of the same opinion.

SHAMBAUGH (1967) recommends the use of diamond burrs parallel to the course of the nerve and continuous or intermittent irrigation with Ringer's solution or Tis-U-Sol®, in order to remove bone dust and prevent overheating.

Others approach the nerve *without performing a complete mastoidectomy* (MEURMAN, 1958). According to MIEHLKE (1960) the nerve sheath should be slit starting distal to the stylomastoid foramen and as far proximally as the nerve is red and edematous. The nerve is identified in the retromandibular fossa 6–8 mm medially from the end of the tympanomastoid suture.

The third landmark in intratemporal facial nerve surgery is represented by the *approach to the labyrinthine segment* of the nerve. In 1954 CLERC and BATISSE grafted the petrous portion of the facial nerve through the middle cranial fossa.

HOUSE (1961, 1963 a, b) revived the interest in the approach to the internal auditory canal and stressed the usefulness of diamond burrs in order to avoid mechanical damage to the nerve and of continuous irrigation to prevent thermal damage. In cases where preservation of hearing is not important (e.g. temporal bone fractures through the cochlea) a postauricu-

lar translabyrinthine approach can be used to expose all portions of the facial nerve from the brain stem to the stylomastoid foramen.

Finally, total exposure of the facial nerve is possible by a combination of the various approaches (HOUSE, 1963a; PULEC, 1966; and FISCH, 1969, 1970). The meatal and labyrinthine segment of the nerve is exposed through a middle cranial fossa or through a translabyrinthine approach in patients who have lost a functional ear, and the tympanic and the mastoid segment of the nerve are exposed through a mastoidectomy and a posterior tympanotomy.

From this review it can be concluded that microsurgical instruments and application of tympanoplastic and neurosurgical principles make it possible, step by step, to gain access to the entire facial nerve in the temporal bone without damaging either the internal or the middle ear. The most appropriate access must be individually chosen in every patient.

It does not seem very important whether one approaches the nerve via a retroauricular or an endaural incision. Also it is not of great importance whether one starts the opening of the facial canal from the stylomastoid foramen upwards or from the posterior end of the lateral semicircular canal downwards.

On the other hand it seems to be very important to avoid damage to the nerve by either heat or through handling of the vascular system of the nerve. Therefore drilling on one and the same place for longer than some seconds should be avoided; irrigation with fluids is advisible. The greatest care in opening the bone of the canal as well as the sheath of the nerve (cupula-technique, delicate cutting of the sheath) is required.

It also seems essential to cover the nerve after the slitting of the sheath lest connective tissue grows into the nerve. Amniotic membrane is a good covering substance (JANSSEN, 1963).

2. DECOMPRESSION IN ANIMALS

Only a few studies have been made to study the possible consequences of a decompression operation on the facial nerve under non-pathological conditions. According to MCGOVERN and HANSEL (1961), in a preliminary study in dogs, "the decompression operation alone did not produce a detectable paralysis (diminishing of the eye reflex), although undoubtedly a varying amount of trauma was incurred to the nerve". The decompression and opening of the nerve sheath of the facial nerve was performed chiefly in the region of the stylomastoid foramen. The nerve was uncovered in the lower vertical segment, and the tight fibrous foramen area was opened widely from above downwards. Because of the dense connective tissue sheath surrounding the nerve at the foramen, attempts to decompress the nerve by dissecting from below resulted in destruction of the nerve. It was not possible in this study to detect by clinical observation the trauma to the nerve caused by the decompression procedure. It should be noted though

that the decompression and opening of the nerve sheath were limited to the lower vertical segment.

BOYLE (1967) studied the effect of decompression in one monkey (Macacus Cynomolgus) in order to evaluate possible neural damage; clinically there was no evidence of weakness of facial musculature. Histological examination (Luxol fast blue) three weeks after decompression revealed a very small area of demyelinization and some increase in vascularity of the nerve trunk in the decompressed area.

The author concluded that even during a carefully performed facial nerve decompression, some degree of neural damage may occur, even though it is not reflected clinically.

BINNS (1967) decompressed the facial nerve in the cat. The facial movements were observed clinically and by electromyography. The nerve was then decompressed from below to the end of the incus and stimulated directly on the exposed nerve as far proximal as possible. Further tests were made at intervals during twenty-eight days. Four weeks after decompression histological sections from the nerve (Luxol fast blue) just distal to the point of decompression and from a distal branch to the eye did not show any sign of degeneration. It was concluded that in cats facial nerve decompression caused no detectable harm measurable by clinical tests, electromyography, conduction time, and histological picture, except in one cat.

However, these experiments cannot be called conclusive: in the first place preoperatively and postoperatively different stimulus parameters were used in the same cat on different places; secondly, a light paresis observed clinically could not be confirmed at an early stage by electromyography. Moreover, it is not clear from the description whether or not the nerve sheath was opened.

Using the same method, in another series of experiments, BINNS (1968) tried to determine how much manipulation the facial nerve in the cat can withstand. The nerve was exposed from the geniculate ganglion to the stylomastoid foramen, then the nerve sheath was opened and the nerve was lifted out of its bed and replaced; all the bone around the nerve was removed and the nerve was left lying freely across the middle ear and mastoid; finally, the nerve sheath of the freely lying nerve was opened.

It was concluded that, if the nerve is intact, recovery will probably ensue, even though the facial nerve may have been grossly displaced from its normal site inside the facial canal.

III – 2. METHODS

Using the same method as described in Chapter II-2 we tried to expose the facial nerve in its mastoid portion as is done in cases of facial nerve paralysis in the human (e.g. Bell's palsy, complicated otitis media, etc.). The bone around the nerve has to be drilled away and the nerve sheath has to be opened. Under the present circumstances the procedure strictly

cannot be called a "decompression", since there is no reason to believe that the intraneural pressure was increased in the experimental setup. Nevertheless we will continue to use the term, since the same operative procedure was employed.

Two stimuli were given: a maximal or supramaximal stimulus in order to be sure that all the relevant nerve fibers were stimulated and a submaximal one in order to observe possible slight changes in the nerve action potential (see Chapter II.).

Before starting the decompression itself, we waited until the nerve action potential was stabilized. The paraffin that covered the operative field was then partly removed in order to gain access to the mastoid process; the stylomastoid foramen was identified for better orientation. The bone was then removed with cutting burrs. Pressure on the burr was carefully avoided and the area was frequently irrigated with Locke's solution at 38° C. In the vicinity of the nerve diamond burrs instead of cutting burrs were used. The drilling was continued until the nerve became visible through the bony canal wall, which fragmented as a result of the drilling. The overhanging bony ridges – the outer half of the facial canal – were removed with dental excavators from the stylomastoid foramen up to the point where the nerve enters the middle ear. In the cat this represents a distance of about 5 mm consisting of a vertical part of about 3 mm (from the stylomastoid foramen to the stapedial muscle) and a more horizontal part of about 2 mm along the stapedial muscle. At the junction of these two parts the nerve crosses a groove between the ectotympanic and the entotympanic part of the bulla. (The last minimal bridge of bone was not removed in order not to dislodge the recording electrode).

Eventually, the sheath of the nerve was opened with a very sharp cataract knife from the stylomastoid foramen upwards to the middle ear. Opening of the nerve sheath was started inferiorly towards the recording electrode in order to prevent movement of the nerve in the electrode. Cutting the nerve fibers was carefully avoided. At the foramen the nerve sheath was very rigid and solidly attached to the bony canal. When the nerve sheath was opened the nerve bulged out slightly.

III – 3. RESULTS

Twelve successful experiments were performed. Both the maximal and submaximal nerve action potential (see Chapter II) were recorded in seven cats, the maximal action potential alone in five cats; there were 19 observations during the 12 experiments.

The most sensitive parameter appeared to be the amplitude of the action potential and was, therefore, used to classify the results.

The removal of paraffin to reach the mastoid process was always followed by a temporary change of the amplitude of the action potential.

The first recording after removal of the bone around the facial nerve showed in almost any case (17 out of 19 observations) a distinct decrease in amplitude. In five experiments where the nerve function recovered after opening of the nerve sheath the decrease in amplitude lasted until the nerve sheath was opened 60 to 90 minutes after drilling. In the other seven experiments the decrease was irreversible until the end of the experiment.

Measurement of the action potential after opening of the nerve sheath revealed an increase in amplitude in 5 cats (7 observations) and a decrease or no change in 7 cats (12 observations).

The decompression was considered damaging when the amplitude did not reach 95 per cent of its initial value at the end of the experiment (Fig. III–1).

In 5 cats the action potential deteriorated more than 20 per cent in amplitude during the experiment. In only one of these cases this could be attributed to the deteriorating general condition of the animal.

In 2 cats (3 observations) the action potential decreased less than 20 per cent during decompression of the nerve.

Decompression was considered successful in 5 out of 12 cats (7 obser-

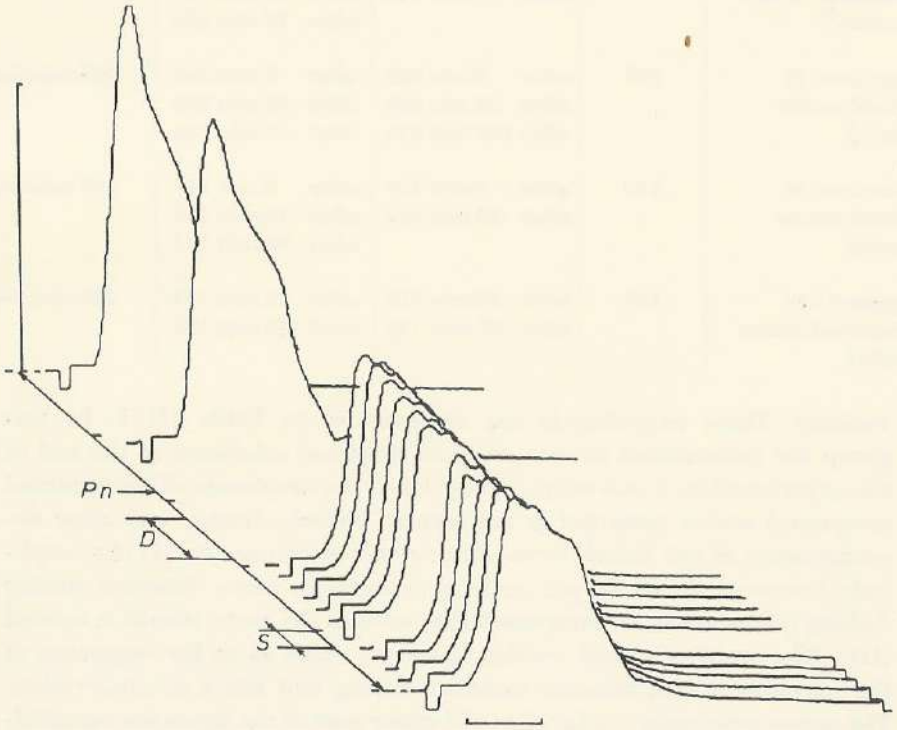
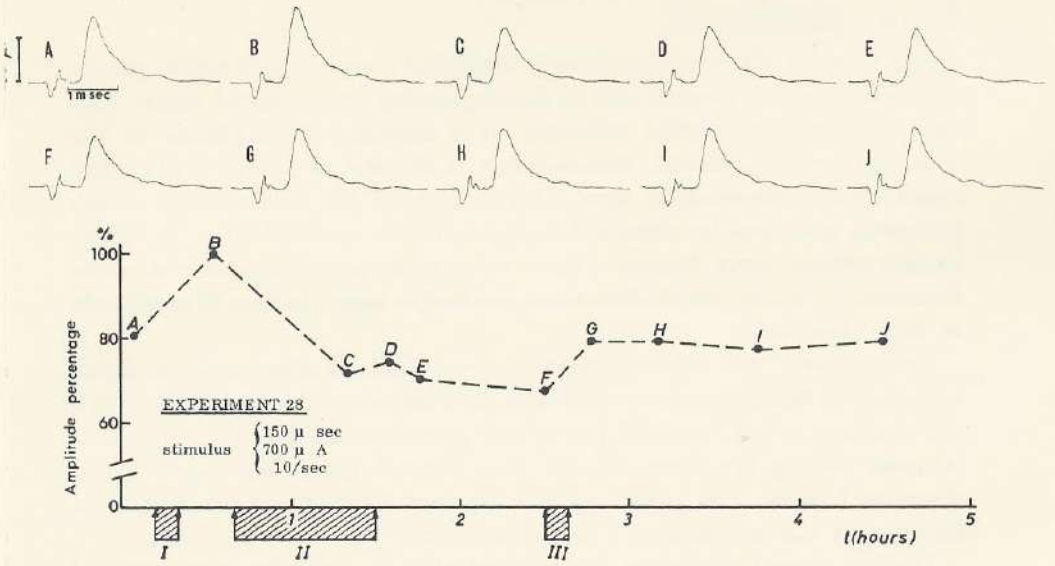


Fig. III–1. Decompression of the facial nerve. The drilling away of bone around the facial nerve provoked a decrease in nerve function. Pn indicates the removal of paraffin, D the drilling away of bone around the nerve, and S slitting of the nerve sheath. The second curve is smaller than the first one, due to stabilization of the setup.
Exp. 24 – SD: 140 μsec – SI: 300 μA – MA: 315 μV – RT: 4 h. 55 min.

TABLE III–1. Amplitude of nerve action potential (in μV) before, during, and after decompression of the facial nerve.

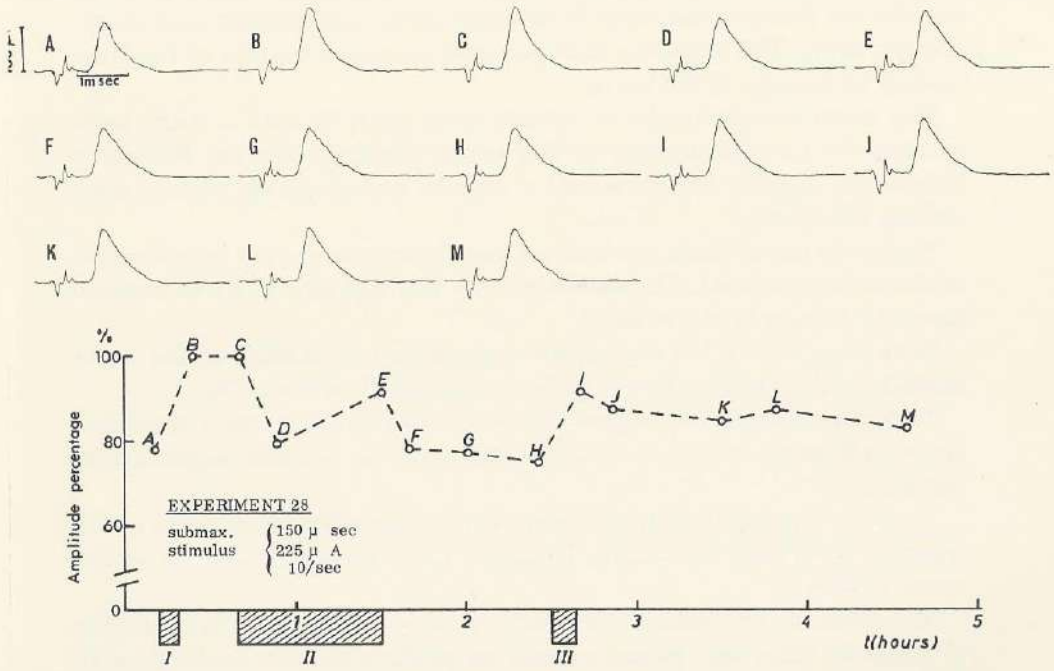
	Before decompression	After drilling	After opening of the nerve sheath	Elapsed time between first and last recording
Experiment 21 maximal action potential	148	after 7 min 148 after 28 min 150	after 4 min 162 after 24 min 168 after 80 min 148	168 minutes
Experiment 22 maximal action potential	463	after 5 min 437 after 21 min 458	after 1 min 463 after 60 min 447 after 75 min 453	385 minutes
Experiment 26 maximal action potential	240	after 5 min 213 after 35 min 219 after 55 min 218	after 6 min 231 after 28 min 214 after 131 min 246	281 minutes
Experiment 26 submaximal action potential	135	after 10 min 141 after 60 min 146	after 1 min 160 after 43 min 145 after 73 min 160	273 minutes
Experiment 27 maximal action potential	270	after 5 min 248 after 25 min 256 after 105 min 224	after 7 min 241 after 42 min 239 after 97 min 246	258 minutes
Experiment 28 maximal action potential	149	after 5 min 137 after 60 min 124	after 6 min 147 after 75 min 144 after 90 min 147	265 minutes
Experiment 28 submaximal action potential	119	after 10 min 119 after 55 min 114	after 1 min 139 after 115 min 126	265 minutes

vations). These experiments are summarized in Table III–1. In this group the submaximal action potential remained unaltered at the end of the experiment in 2 cats only. In Fig. III–2 the amplitude of the maximal compound action potential is represented before, during, and after decompression of the facial nerve. After removal of paraffin (I) the amplitude increased about 20 per cent. A decrease in nerve function during drilling (II) seems to be compensated as soon as the nerve sheath is opened (III). The amplitude then reaches the same value as at the beginning of the experiment and remains constant during two hours of observation. The action potentials displayed in the upper part of the figure are essentially the same throughout the experiment except for small temporary fluctuations in amplitude. Neither latency changes nor variations in the shape of the action potential are seen. In Fig. III–3 the amplitude and the action potentials provoked by submaximal stimulation during a decompression are represented.



I Partial removal of paraffin – II Drilling away of bone – III Slitting of the nerve sheath.

. III–2. Decompression of the facial nerve under observation of the maximal nerve action potential. e amplitude of the action potential provoked by maximal stimulation of the nerve is represented on vertical axis in percentage of the maximal amplitude (185 μV = 100 per cent). The action potentials referred to in points A, B, C, D, etc. are displayed in the upper part of the Figure.



I Partial removal of paraffin – II Drilling away of bone – III Slitting of the nerve sheath.

. III–3. Decompression of the facial nerve under observation of the submaximal action potential (152 μV = 100 per cent).

III – 4. DISCUSSION.

Our method of averaging strongly amplified evoked nerve action potentials required the utmost care in decompression of the facial nerve. Any damage was immediately reflected in a decrease of amplitude of the maximal action potential. Subsequently it did not reach its initial value again during observation time. Observation of the submaximal action potential, produced by stimulation of part of the nerve fibers of the zygomatico-orbital nerve branch was an even more sensitive check of nerve function, since very small alterations resulted in large changes in amplitude in both directions.

Drilling away the bone around the nerve seems to affect nerve function more and is therefore more dangerous than slitting the nerve sheath. In 2 observations out of 19, drillings did not provoke a decrease in amplitude, whereas slitting the nerve sheath was followed by a decrease in nerve function 7 times. The latter had no influence 5 times and resulted in an increase in nerve function 7 times (see Table III–1).

Drilling should, therefore, be performed very carefully with minimal pressure on the bone, preferably with a high-speed drill which cuts more easily. The use of diamond burrs for fragmentation of the bone is preferred to that of cutting burrs in the neighborhood of the nerve.

Moreover, a Locke's solution or equivalent physiological fluid at body temperature should be used for irrigation. Physiological saline is to be avoided for this purpose, since it damages living cells (KERTH and SHAMBAUGH, 1964). The irrigating fluid prevents excessive heating of the bone leading to damage of the nerve.

The nerve sheath should be opened very carefully with a sharp knife in order to prevent damage to the nerve fibers. In the cat the nerve bulged out slightly and presented a smooth and gleaming surface after slitting the sheath.

Neglect of one of these precautions was always reflected in deterioration of the action potential. The results indicate the high sensitivity of nervous tissue to damage however slight.

More than half of the decompressions resulted in damage to the nerve insofar as the amplitude of the action potential reflects this.

It seems reasonable to suggest that in decompression in man, the facial nerve will more often be damaged than in the present experimental procedure.

It can be concluded that it is possible to uncover the facial nerve in the mastoid portion of the temporal bone of the cat without damage to the nerve function.

Experimental evidence justifies surgery on the facial nerve in a patient; if correctly done this procedure can be performed on a healthy nerve without damage to its function.

CHAPTER IV

ISCHEMIA OF THE FACIAL NERVE.

IV – 1. INTRODUCTION.

The oxygen supply to peripheral nerves and their paralysis by pressure has been extensively discussed in the literature. A short review is given in Chapter I.

The acute experiments reported gave the opportunity to study ischemia of the nerve at the end of control experiments (Chapter II), decompressions (Chapter III), and pressure experiments (Chapter IV).

In this Chapter the behaviour of the facial nerve action potential under such circumstances will be described. The deterioration of the action potential by ischemia will be compared to that by pressure on the nerve fibers in Chapter V.

IV – 2. METHODS

Ischemia of the nerve was obtained by arrest of the blood circulation by a lethal dose of Nembutal® or by a saturated solution of potassium chloride (3 mol) intravenously. Since injection of Nembutal was followed by several minutes of irregular heart action the method with KCl, which caused immediate cardiac arrest, was adopted. The action potentials and calibrations were recorded just before ischemia. If necessary, the stimulus strength was adjusted. Recordings of the influence of the developing ischemia on the nerve were made at intervals of 5 minutes until all activity had disappeared.

IV – 3. RESULTS.

Ischemia by injecting potassium chloride was studied in 12 cats, 2 of the control group, 1 at the end of a decompression operation of the nerve and 9 after a reversible pressure block of the nerve (Chapter V). The nerve block by pressure was judged reversible when the nerve action potential recovered in amplitude and configuration after release of pressure. Experiments during which the amplitude of the action potential fluctuated were also included. In Fig. IV–1 the maximal amplitude of the action potential is plotted against the duration of ischemia.

The ischemia in experiments 31 and 89 followed a control experiment, and in experiment 26 a successful decompression of the facial nerve. The mean value of the amplitudes before ischemia was 234 μV. The other nine curves represent ischemia after infusion compression of the nerve (Chapter

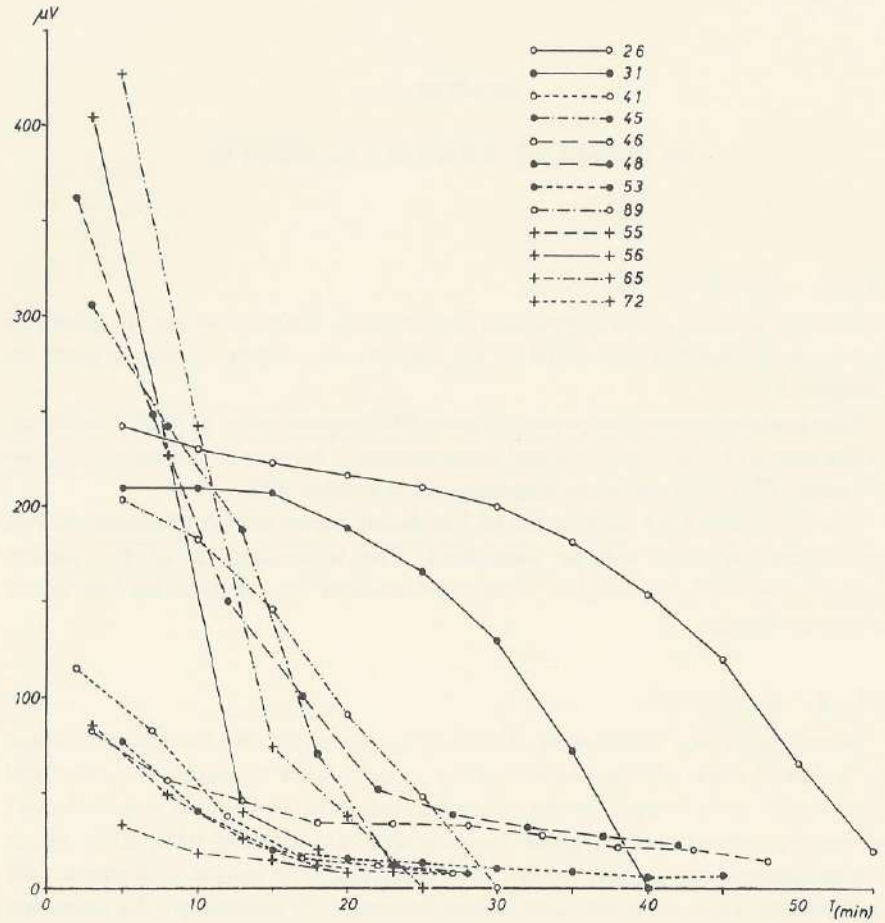


Fig. IV–1. Influence of ischemia on the maximal nerve action potential in 12 cats. The maximal amplitude of the action potential is plotted against the duration of ischemia. Insert: the number of the experiment.

V); their mean amplitude before ischemia was 239 microvolts. It appears from this Figure that the process of ischemia in experiments 26, 31 and 89 differed distinctly from the other nine curves.

In the former case the nerve resisted the process of ischemia for a relatively long time; the action potential decreased to 50 per cent after 17 to 45 minutes; then the breakdown rapidly followed. In the latter case the action potential decreased rapidly after onset of ischemia, within 15 minutes to 50 per cent; it, thereafter, went down more slowly. In Figure IV–2 a few examples of the influence of ischemia on the nerve action potential are given.

Abnormal susceptibility of damaged sensory and motor fibers to ischemia after sphygmomanometer cuff compression has also been demonstrated (Gilliat and Wilson, 1954; Fullerton, 1963).

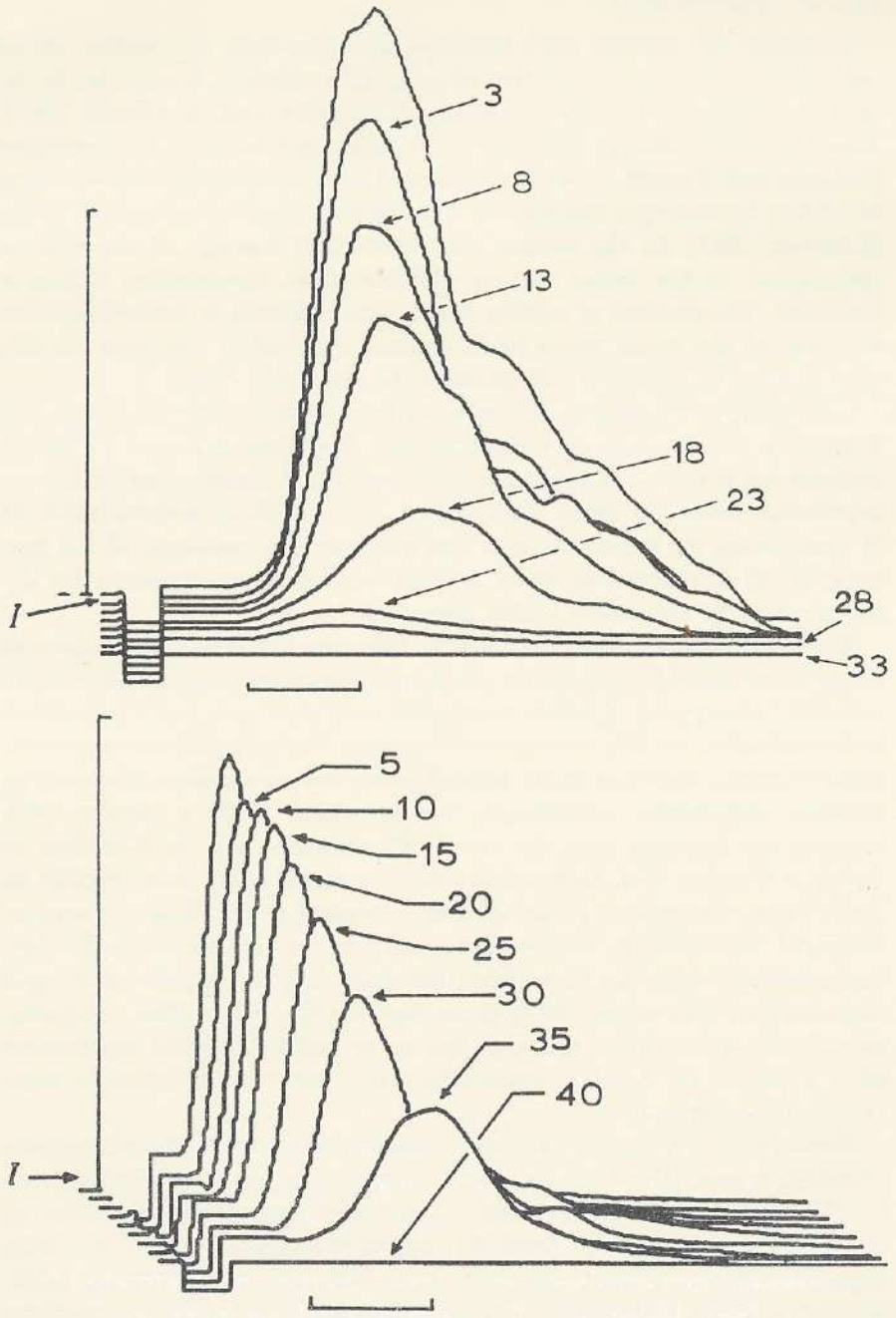


Fig. IV–2. A few examples of decrease in nerve action potentials under influence of ischemia after a reversible infusion compression (upper curves) and at the end of a control experiment (lower curves). The figures indicate the duration of ischemia in minutes. Calibrations: 250 μV (vertical bar) and 1 millisecond (horizontal bar). Exp. 45 (upper curves) – SD: 300 μsec – SI: 700 μA – MA: 370 μV – RT: 36 min. Exp. 31 (lower curves) – SD: 360 μsec – SI: 500 μA – MA: 224 μV – RT: 40 min.

IV – 4. DISCUSSION

It should be stressed that ischemia was provoked by cardiac arrest induced by intravenous injection of potassium chloride. It can also be induced by rapid intravenous injection of air (Fox and Kenmore, 1967). This method is clearly different from nerve ischemia by a tourniquet (Gilliat and Wilson, 1954; Fullerton, 1963; and others) or substitution of oxygen by other gas mixtures in a chamber containing an isolated nerve (Lehman, 1937). In the former case mechanical damage of nerve fibers may occur, in the latter oxygen diffusion from surrounding tissues is excluded. The method of cardiac arrest was preferred in order to achieve ischemia of the facial nerve in its normal anatomical situation. It also circumvents the influence of pressure on the nerve fibers.

The mechanisms underlying ischemia have been reviewed by Fox and Kenmore (1967). Anoxemia, hypercapnia, hyperkalemia, and metabolic acidosis are involved in ischemia. Lactic acid and carbon dioxide have a depressant effect on nerve stimulation. The resulting accumulation of H^+ ions raises the surface tension and increases the resistance of the proteolipid cell membrane to water soluble ions which are essential for the propagation of the nerve action potential.

The results shown clearly indicate a difference between the influence of ischemia on intact nerves and on nerves which were previously subjected to infusion compression. The mean amplitude of the nerve action potential before ischemia was the same for both groups. The only difference between the two groups was that in the second group the nerves were subjected to infusion compression beforehand, during which Locke's solution with heparin was injected into the nerve. This method will be described in detail in Chapter V–2. Ischemia by cardiac arrest acted more rapidly in the infusion compression group than in the control group. This indicates an increased vulnerability to ischemia, and can be explained by the fact that edema between the nerve fibers (see page 64 Plate 6) prevents oxygen diffusion from the surrounding tissue towards the nerve. The conclusion can also be drawn that, although the nerve action potential regenerates after a period of infusion compression, the nerve is nevertheless more vulnerable to ischemia.

Edema of the nerve is considered an important factor in the pathogenesis of ischemic paralysis of the facial nerve in man (Chapter V–1). Consequently, this experimental evidence emphasizes that surgical decompression of the nerve in these cases should be undertaken with extreme care with regard to the blood circulation of the nerve (Kettel and Jongkees, 1963; Miehlke, 1960). Disturbance of the arterial supply to an edematous nerve may be more hazardous than to a normal one.

CHAPTER V

INFUSION COMPRESSION OF THE FACIAL NERVE

V – 1. INTRODUCTION AND LITERATURE

The experiments on the intratemporal portion of the facial nerve reported in the literature will first be reviewed. Infusion compression under various conditions is described and discussed. Finally it is compared to compression and ischemia of peripheral nerves.

Many attempts have been made to damage the facial nerve in such a way as to imitate an intratemporal facial nerve paralysis and to study the effect of a decompression of the nerve. The experiments are reviewed and discussed in three main groups, according to the mechanism of injury:

a. MECHANICAL LESIONS

In the rat Sullivan and Smith (1950) placed a suture about the main trunk of the nerve just below the stylomastoid foramen. The vascular supply of the nerve was studied by intravenous injection of trypan blue. The nerve function was judged by observation of the resulting paresis or paralysis of the facial musculature, and by observing the reaction to application of faradic current to the exposed nerve. According to the authors pressure directly by stricture or indirectly by edema production extending proximally from the stylomastoid foramen can cut off the venous return with subsequent tissue damage. They hypothesize that early reestablishment of normal circulation by decompression will hasten recovery.

McGovern and Hansel (1961) performed the same operation in dogs. The eyelid reflex was used as an index of reinnervation. The facial nerve tended to recover sooner if the nerve was decompressed and neurolysis was performed in the region of the stylomastoid foramen. In 6 incompleted experiments, however, three dogs showed a faster recovery on the side not decompressed.

The authors concluded that the histological findings in dogs were similar to published reports of nerves obtained from cases of Bell's palsy and from experimental work on facial paralysis. There was no macroscopic swelling when the nerve was reexposed for biopsy. The nerves were removed two, four, eight and ten weeks postoperatively.

It should be noted that the nerve was studied outside the bony canal and that the decompression was limited to the stylomastoid foramen in these experiments.

McGovern et al. (1963) decompressed the facial canal in its lower

segment in dogs after injury to the nerve trunk by complete transsection near its exit from the stylomastoid foramen and resuture with 0000 silk. Decompression of the nerve inside the bony canal included a dissection that freed the nerve from the dense fibrous tissue around it at its entrance into the foramen. The impression gained on inspection that the decompressed nerve was better preserved and showed hardly any serious nerve involvement was confirmed by microscopical examination. The faster recovery on the decompressed side is thought to have been accomplished by releaving the constricting effect of the rigid canal, limiting the factor of secondary compression. According to these authors the decompression concept can possibly be applied to traumatic lesions of the extratemporal part of the nerve. If nerve repair is then combined with lower segment decompression the paralysis following traumatic edema may recover with greater speed and with greater eventual effect.

Rosenberg and Alford (1966) compressed the auriculopalpebral branch of the facial nerve in 40 dogs by a methyl methacrylate clamp tightened by means of two stainless steel screws. The amount of partial pressure was gauged by the presence or absence of blood in one of the small venules coursing through the epineural sheath visible through the clamp.

A stimulating electrode (insulated stainless steel wires) was implanted into the nerve with a 25-gauge needle 1 cm distal to the clamp. Eyelid-motion and nerve excitability thresholds were studied postoperatively. Decompression was performed by releasing the screws, removing the clamp and incising the perineural sheath.

In animals with paralysis due to partial pressure and where nerve excitability remained normal, there was prompt and complete return of function whether or not decompression of the nerve was performed. This was interpreted as a state of neurapraxia.

Five animals with complete palsy and with loss of nerve excitability showed incomplete return or no return of function without decompression. The authors concluded that denervation must have occurred. Eventually one animal in this group recovered completely.

Regardless of the amount of pressure, the animals decompressed at the earliest stage showed the best results. In all dogs with reduced excitability and complete paralysis by partial or maximum pressure, decompression within one week resulted in prompt return of function without synkinesis. Animals with complete palsy decompressed after 2 or 3 weeks had a prolonged complete recovery of function and some had synkinesis. The authors concluded that decompression of the facial nerve has definite merits as a means of improving the prognosis or preventing further degeneration.

Partial or maximum pressure was thus applied from outside the nerve. However, it is not clear whether application of the clamp itself or implantation of the electrode did not damage the nerve to a certain extent, thus accounting for part of the described results.

Binns (1967) studied the facial nerve in the cat by clinical observation and by electromyography.

In 12 cats the nerve was exposed in its most exterior curvature and lifted out of its bed with a hook. The nerve was then replaced. Ten of these nerves degenerated. When decompression followed immediately or 48 hours later normal movement was maintained and no degeneration occurred (18 cats). Decompression 72 hours after mobilization did not prevent degeneration (6 cats). It was concluded that exposure of the nerve at its most exterior curvature did no harm but that excessive stretching or manipulation should be avoided.

Factors other than pressure (e.g. stretching), which are difficult to control, were probably involved in this experiment.

Boyle (1966) studied facial nerve regeneration in monkeys. It was concluded that spontaneous recovery of function can occur after injury of moderate degree but it is precluded in the case of extensive resection of the facial nerve. Spontaneous recovery, when it occurs, is due to a random regeneration of axonal elements of the facial nerve and not to vicarious functions from the trigeminal nerve. In six monkeys ligation with catgut and crushing in two with a clamp was performed just anterior to the stylomastoid foramen. Recovery, when it occurred, was accompanied by synkinesis.

In rabbits the facial nerves were dissected bilaterally near the stylomastoid foramen (Moszynski, 1968). The nerves were then crushed bilaterally by a nylon loop against a glass rod held under them. One side was then decompressed, the other side remained unopened.

In the first week after injury similar changes were seen on the decompressed and on the non-decompressed side: signs of early degeneration in the distal part of the nerve and swelling of the nerve proximal to the stylomastoid foramen (5 rabbits).

Three weeks after the operation – after recovery from the paralysis – the swelling of the proximal portion was less extensive on the decompressed side in 12 out of 17 rabbits. The recovery in this group, judged by clinical observation and by EMG, was faster on the decompressed side in 10 animals, equal on both sides in 5, and slower on the decompressed side in 2.

The author concluded that liberation of the injured facial nerve from the osseous canal accelerates its recovery. The extent of the decompression, however, is not mentioned, and neither is it stated whether the nerve sheath was opened or not. The nerve was crushed outside the temporal bone.

Boyle (1967) used ultrasound irradiation to the labyrinth in monkeys (Macacus cynomolgus). An ultrasonic generator probe was placed on the horizontal semicircular canal after a simple mastoidectomy. Decompressive surgery in 12 animals after the heat-induced vascular lesion had no significant effect on the rate and degree of recovery of function, regardless when it was done.

The author stresses that the decompression procedure was done in the presence of a mild type of neural lesion that was known to be reversible. The lesion consisted primarily of vascular dilatation with intraneural hemorrhage and extravasation of red blood cells. Myelin degeneration followed but no axonal breakdown (as judged from sections stained with Bodian's protargol stain, Luxol fast blue myelin stain and hematoxylin-eosin). Gradual resolution of the hemorrhagic lesion was followed by clinical recovery of the normal function. It is concluded that a vascular lesion can cause facial palsy and that the severity of the paralysis is probably related to the duration of ischemia in the nerve.

b. LESIONS INDUCED BY COLD, HEAT OR CHEMICAL SUBSTANCES

SULLIVAN and SMITH (1950) tried to produce a dysregulation in the circulation of the facial nerve by applying cold to the shaven facial skin of rats. The nerve function was judged by observation of the paralysis and by observing the reactions to the application of faradic currents to the skin. Cold produced intravascular edema. The authors hypothesize that early reestablishment of normal circulation by decompression at the stylomastoid foramen will quicken recovery in Bell's palsy.

COASSOLO (1952, 1953a, 1953b) induced paralysis of the facial nerve in rabbits, sensitized by horse serum, by application of ice on the extratemporal part of the nerve. Histological examination revealed edema with degeneration of nerve fibers. According to the author nerve excitability measurements by galvanic current showed an analogy between his experimental paralysis and Bell's palsy. This paralysis "a frigore" is attributed to an allergic mechanism. If procaine (0.015 g/kg of a 1% solution) was given intravenously 5 minutes prior to refrigeration, neither paralysis nor macroscopic lesions appeared. The author advocates early administration of procain in order to inhibit the release of histamine.

A paralysis obtained by freezing the exposed facial nerve trunk, with or without protein shock, was judged to be too transient to give meaningful results in dogs (MCGOVERN and HANSEL, 1961).

In other experiments, MCGOVERN et al. (1966) tried to produce an ischemic facial paralysis in dogs. Injection into the stylomastoid artery of potent peripheral vasoconstrictors (epinephrine 1:1000, phenylephrine 1:1000 and angiotensin amide) did not produce facial paralysis. Additional interference with the blood supply of the nerve by mobilizing and wrapping the nerve in tantalium foil in order to isolate the vertical section of the nerve in the facial canal, proved ineffective.

These three chemically different vasoconstrictors were then injected into the facial canal. In one series the nerve was exposed at the stylomastoid foramen and the bony facial canal around the foramen was removed sufficiently to insert a 25-gauge needle into the canal between its wall and the nerve; in another series the nerve was exposed at the stylomastoid foramen, the fibrous collar was removed and the needle was passed upward into the canal inside the nerve sheath. The lid reflex was used as indicator of the degree of paralysis which seemed to be in proportion to the amount of vasoconstrictor injected into the Fallopian canal. Injection of 0.1 cc of phenylephrine inside the nerve sheath peripherally from the foramen on one side and proximally to the foramen on the other side resulted in a distinct paralysis in the latter instance.

Microscopic examination of 7 pairs of nerves 2 to 8 weeks after surgery revealed no abnormalities. The paralysis was interpreted as an ischemic neurapraxia or a conductive block, caused by a transient obstruction of the vasa nervorum.

As the bone of the facial canal was removed all around the stylomastoid foramen and in some cases also the fibrous collar around the nerve, the transient paralysis in these experiments could be ascribed to the insertion of the needle together with the injection of saline.

In another series of experiments BOYLE (1966) correlated clinical nerve excitability and histological findings in graded facial nerve injuries with and without interruption of the anatomical continuity of the nerve. In 5 monkeys the facial nerve inside the parotid gland just anterior to the stylomastoid foramen was injected with hot water (85 to 90°C) and in one with 70 per cent ethyl alcohol. All the animals showed immediate facial paralysis. Three of the 5 animals recovered complete function in 24 to 48 hours. Nerve excitability was lost in the other three animals.

Histological examination confirmed axonal continuity in all instances with evidence of demyelinization in those nerves with a greater degree of damage.

The conclusion was reached that the preservation of nerve excitability for 72 hours after injury (including mechanical lesions) is an excellent prognostic sign while loss of nerve excitability after 48 to 72 hours does not preclude return of function. Return of clinical function follows the return of nerve excitability.

c. LESIONS BY COMPRESSION INSIDE THE NERVE

The work of JAIN and SHARMA (1964) is of importance because their modified infusion compression method is used in our pressure experiments. The authors devised a method to produce intrafascicular edema and compression inside the facial nerve. In the rabbit infusion under pressure of normal saline solution into the facial nerve inside the Fallopian canal produced complete facial paralysis. A similar compression of the nerve outside the canal was relatively ineffective. In their opinion application of ice, ligatures or clips around the nerve are methods difficult to standardize; no uniform pressure can be applied on all the fibers inside the nerve. The functional state of the nerve was observed under light pentobarbital anesthesia (eyelid reflex) 1 or 2 hours after the operation when the rabbit regained full consciousness (loss of spontaneous and reflex movements of whiskers and loss of tone and movements of the voluntary musculature of the ear).

In three dogs saline was injected into the facial canal by McGovern et al. (1966), producing a transient paralysis in one dog. Introduction of saline inside the nerve sheath in 2 dogs seemed to confirm the theory of Jain and Sharma. In a later series of experiments, injection of saline into the facial canal on one side as a control produced only a transient paralysis except when injected under prolonged pressure or in excessive amounts.

V – 2. METHODS

The method we used is a modification of the infusion compression method of Jain and Sharma (1964). Difficulties in fixation and orientation during these experiments were attributed to variations in anatomy of the skull in the individual cats. In order to minimize these differences the last series of experiments was performed in cats weighing between 2.3 and 2.7 kg.

1. INSTRUMENTS

Many attempts with conventional hypodermic needles failed: the nerve function decreased to an unacceptable degree, probably because of transsecting too many nerve fibers with the sharp edges of the needle or leakage or clogging of the needle, which made exertion of pressure in the nerve impossible. Finally we used a specially modified 25-gauge hypodermic needle (Fig. V–1). The tip of the needle was occluded by silver and polished in such a way that it was located more centrally than in the conventional needle. Small holes of 0.3 mm were drilled in the lateral wall of the needle as near as possible to the tip. Leakage occurred rarely if we used the needle with the openings near the tip of the needle. Obstruction was also prevented by the lateral position of the holes. The needle was curved at an angle of 41 degrees with a fine wire inside the needle. Finally the remaining sharp edges of the needle were removed.

The needle was connected to a polythene cannula No 4 (bore 2.0 mm, wall 0.5 mm) and fixed on a perspex holder. A three-axial micromanipulator (Brinkmann RP IV–L), fixed at about 10 cm on the right side of the head of the animal was used for fixation of the needleholder by means of a ball-and-socket joint. The polythene cannula was connected by a three-way stopcock to a mercury manometer or to open air (Fig. V–1). The mercury manometer was placed at the level of the nerve.

In some experiments the blood pressure of the cat was also measured. Therefore the femoral artery was cannulated with a catheter (Intracath 1617) which was moved into the abdominal aorta. The blood pressure was measured by an electromanometer (Elema Schönander Company, EMT 35) connected to a polygraph (Elema Schönander Company, Mingograph EM 34). The electromanometer was placed at the level of the heart of the animal. Before each measurement the patency of the cannula was checked by injection of a saline solution with heparin (1/1000).

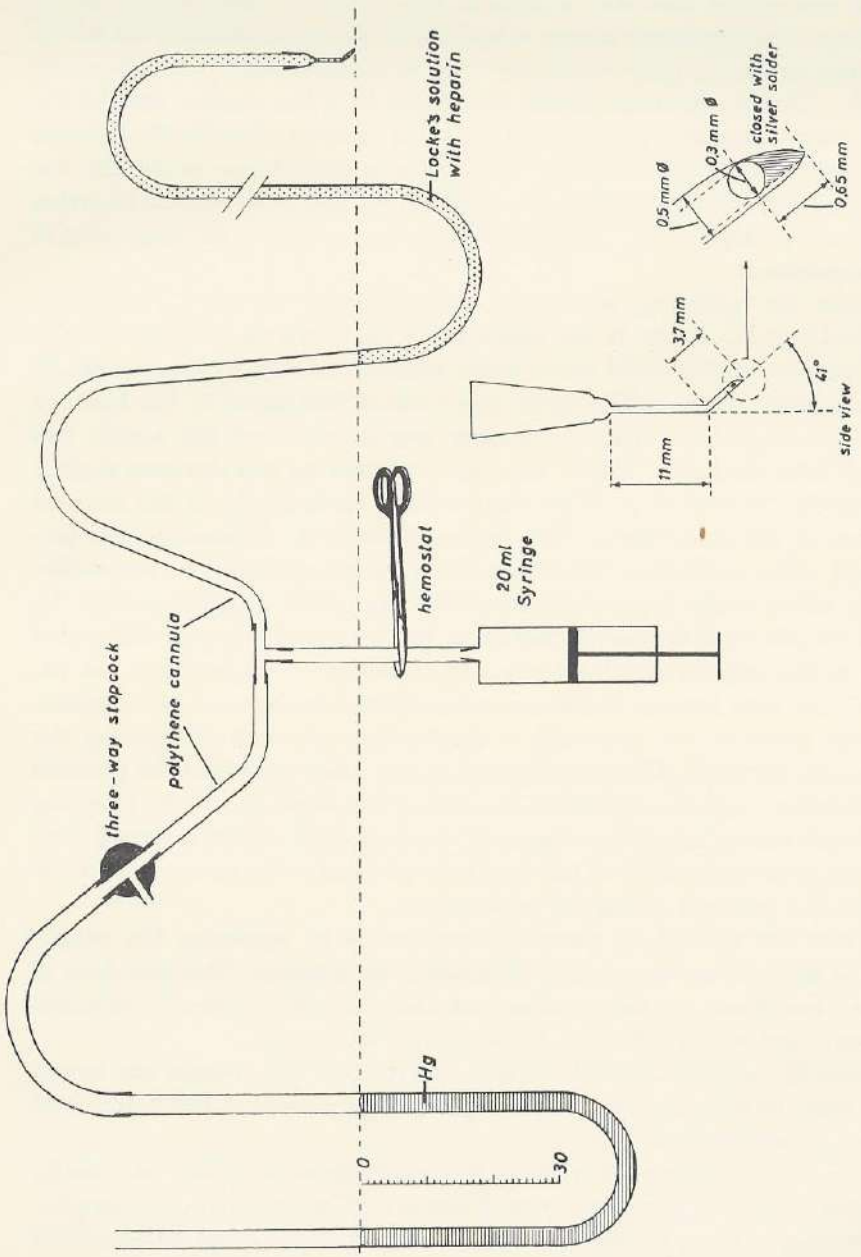


Fig. V–1. Pressure system for infusion compression of the facial nerve. In the right lower corner a detailed description of the hypodermic needle.

2. METHOD

Before insertion of the needle, the pressure-system was filled with Locke's solution containing heparin (0.6/1000). A sufficient amount of liquid was stored into the cannula of the system in order to be able to continue the experiment in case of leakage of the system outside the nerve. Pressure inside the nerve could never be maintained when air entered the needle. After stabilization of the action potential the paraffin around the stylomastoid foramen was removed partly in order to visualize the foramen together with the nerve. It was of the utmost importance to identify the nerve in the foramen clearly in order to achieve a successful insertion (orientation, depth, etc). This identification was done at the beginning of the experiment.

Under the operating microscope (magnification 10 ×) the needle was inserted into the nerve. It was necessary to do it in a curved way because the branches of the facial nerve curve laterally as soon as they leave the stylomastoid foramen. The nerve was entered just distal to the foramen in its posterior half. The epineurium was pierced and the needle was moved into the nerve slowly. The tip of the needle was directed slightly posteriorly; finally its position was 2–3 mm horizontally in the mastoid portion of the facial nerve. This extremely delicate manoeuver was performed under continuous inspection of the action potential on the oscilloscope, which made it possible to position the needle more favorably. An assistant attached the needle holder to the clamp on the ball-and-socket joint of the micromanipulator when the needle had reached the best position. The fluid level in the system was adjusted to the level of the needle and the pressure was equalized to atmospheric pressure by opening the three-way stopcock. After stabilization of the action potential the pressure in the nerve could be augmented by closing the stopcock and by injecting fluid with the syringe after closure of the hemostat. Again the fluid level was adjusted to the level of the needle. If necessary, the pressure could be adjusted or released during the experiment.

It was also possible to provoke compression by squeezing the rubber tube of the system, temporarily increasing the pressure. This was done in those cases where the nerve action potential did not decrease by external pressure and the needle was supposed to be clogged.

Generally, a small amount of liquid (about 0.15 ml) entered the nerve. This could be evaluated by measuring the displacement of the liquid level in the pressure system.

In 22 cats an attempt to influence the maximal nerve action potential by infusion compression did not succeed because of leakage either of the pressure system or along the needle (5 cats), clogging of the needle, or because of heavy damage to the facial nerve by inserting the needle into the nerve (17 cats).

In suprasystolic infusion compression, the pressure inside the compression system was kept above the systolic blood pressure: between 150 and 320 mm of mercury. The intra-arterial blood pressure values in 12 cats were between 90–120/130–160 mm of mercury. The experiment was considered successful if the following criteria were fulfilled:

1. An amplitude of the action potential of at least about 100 μV after insertion of the needle.
2. A decrease of the action potential of at least 70 per cent within the first 30 minutes of infusion compression.
3. A residual action potential (after a period of pressure) of no more than 20 per cent.

V – 3. RESULTS

1. SUPRASYSTOLIC INFUSION COMPRESSION

Electrophysiology

In 29 cats the nerve action potential decreased after injecting fluid into the nerve under suprasystolic pressure. In 12 of these 29 experiments the effect appeared slowly, or fluctuated. Therefore, only 17 experiments satisfied the criteria for suprasystolic compression.

From these experiments, 4 animals were also used for histological study, in 2 suprasystolic pressure was applied more than once and in 3 the nerve was submitted to various pressures, combined with suprasystolic pressure.

In Figure V–2 a three-dimensional display is given of the nerve action potentials during the experiment. The nerve was compressed for a period of 105 minutes. The infusion compression was followed by an over-all decrease of the nerve action potential. It did not disappear completely; 15 per cent of its amplitude remained visible.

As soon as the pressure was relieved, the action potential reappeared. The return of the action potential, however, was distinctly different from its disappearance, the right part of the curve appearing faster than the left part. Finally, the action potential became about the same as before although small differences in recovery time of certain fiber groups were still evident. The experiment was finished by recording action potentials after circulatory arrest (ischemia). A detail of an experiment of 58 minutes of compression followed by recovery is given in Figure V–3. Another short period of suprasystolic compression (42 minutes) is represented in Figure V–12. In experiment 48 (Fig. V–4) infusion compression was continued for 180 minutes. Although the amplitude of the action potential recovered after release of pressure, the action potential was delayed and altered in shape. The center of gravity became delayed from 1450 to 2100 μsec in relation to the stimulus artifact.

In 2 experiments, number 60, (Fig. V–5) and number 62 (Fig. V–6) the infusion compression lasted 210 minutes and in experiment 67 (Fig. V–7) 245 minutes. In these three experiments the amplitude of the nerve action potential remained negligible after release of pressure over the rest of the observation time.

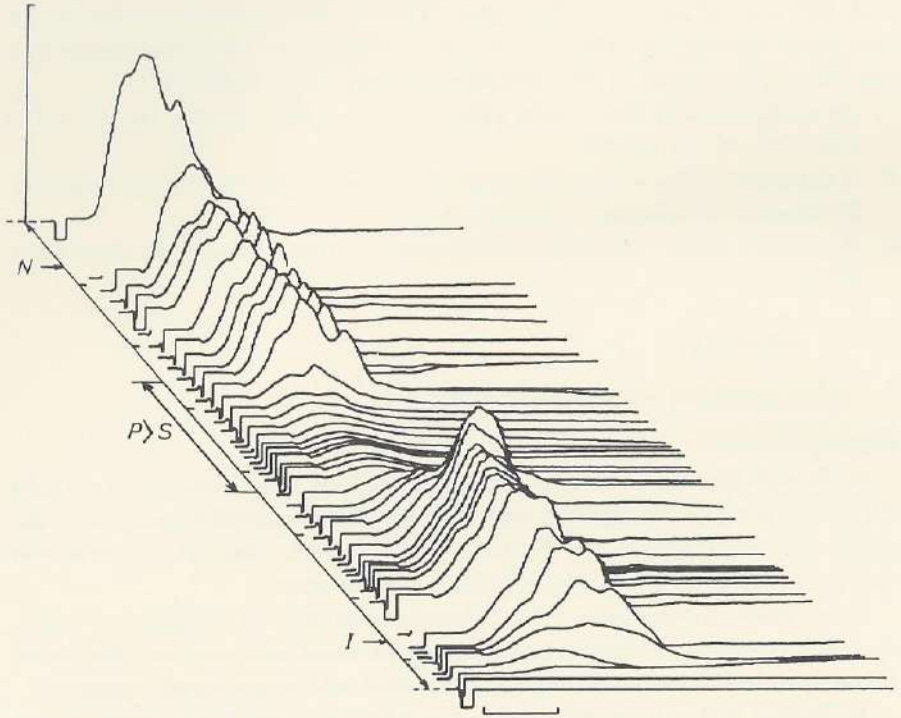


Fig. V–2. Three-dimensional display of nerve action potentials during 105 minutes of suprasystolic infusion compression. Each curve represents 500 averaged action potentials. The first curve recorded during the experiment is in the upper part of the Figure, the last one in the lower part. N indicates the insertion of the needle; P > S suprasystolic compression; I ischemia.
Exp. 41 – SD: 150 μsec – SI: 300 μA – MA: 189 μV – RT: 7 h. 27 min.

In Table V–1 and Fig. V–8 a survey is given of 9 experiments in which suprasystolic infusion compression was performed. The amplitude of the action potential, in per cent of the amplitude before infusion compression, is plotted against the time from the onset of compression. After release of pressure the amplitudes regained their original values in all the experiments except in experiment 60, 62 and 67 where the nerve was compressed for more than 180 minutes. There is a distinct difference in effect of release after short and long times of compression.

Histology

In 4 cats the facial nerve, with the needle inside it, was prepared for histological investigations. The fixation process was initiated in vivo with the pressure still on the nerve and postfixed for 4 hours in "Susa" mixture, as described in Chapter II.

Fixation was begun after about one hour of suprasystolic pressure in 2 cats and after 4 hours in two others. In the longest of these experiments

the fixation was started 10 to 11 hours after section of the nerve. In Plates 1 and 2 (see page 64) two transverse sections through the vertical part of the normal facial nerve are presented before compression.

In Plate 3, a general view of the facial nerve is presented after slightly over one hour of infusion compression. The microphotograph is to be compared with Plate 2 of the normal nerve with the same magnification. In the compressed nerve lighter areas represent dilatations of the endoneural

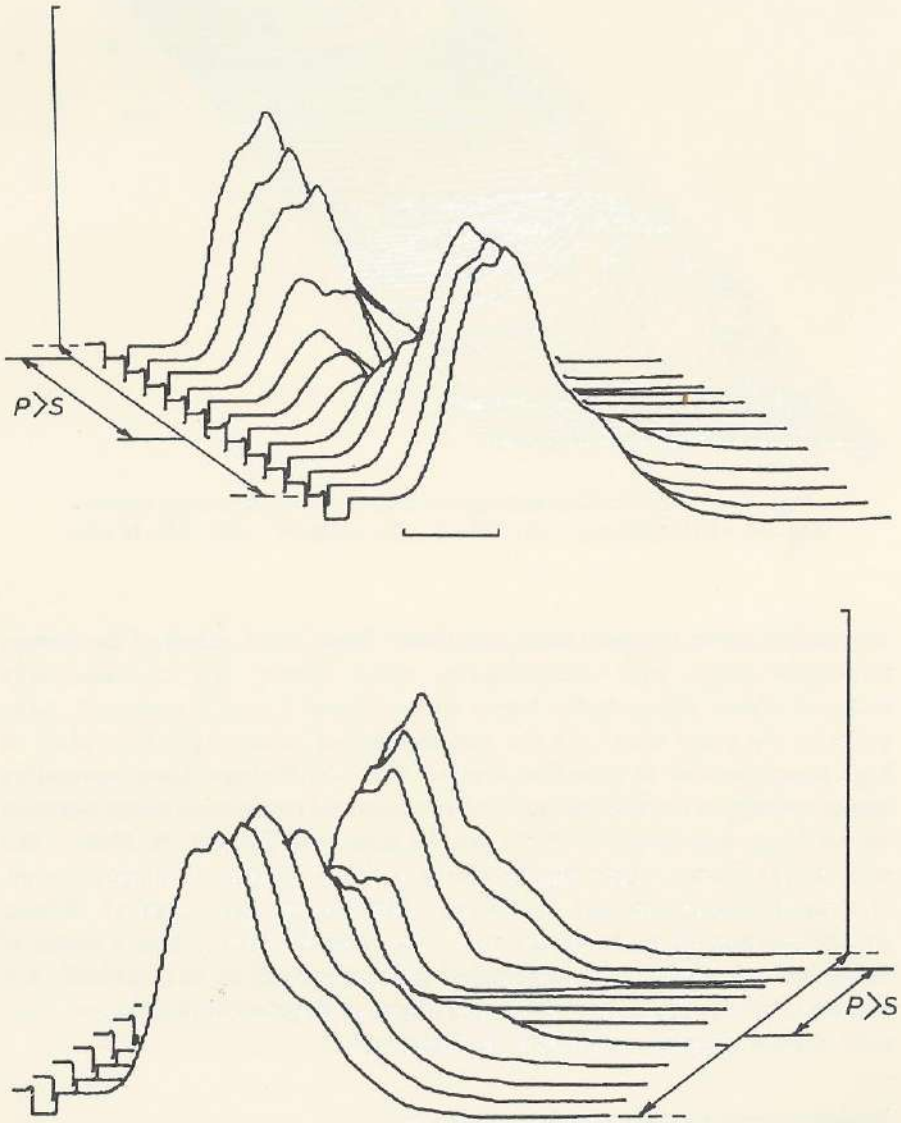


Fig. V–3. Fifty-eight minutes of infusion compression. Displays are given from left side and from right side for better evaluation of the changes in action potentials.
Exp. 38 – SD: 240 μsec – SI: 450 μA – MA: 170 μV – RT: 1 h. 50 min.

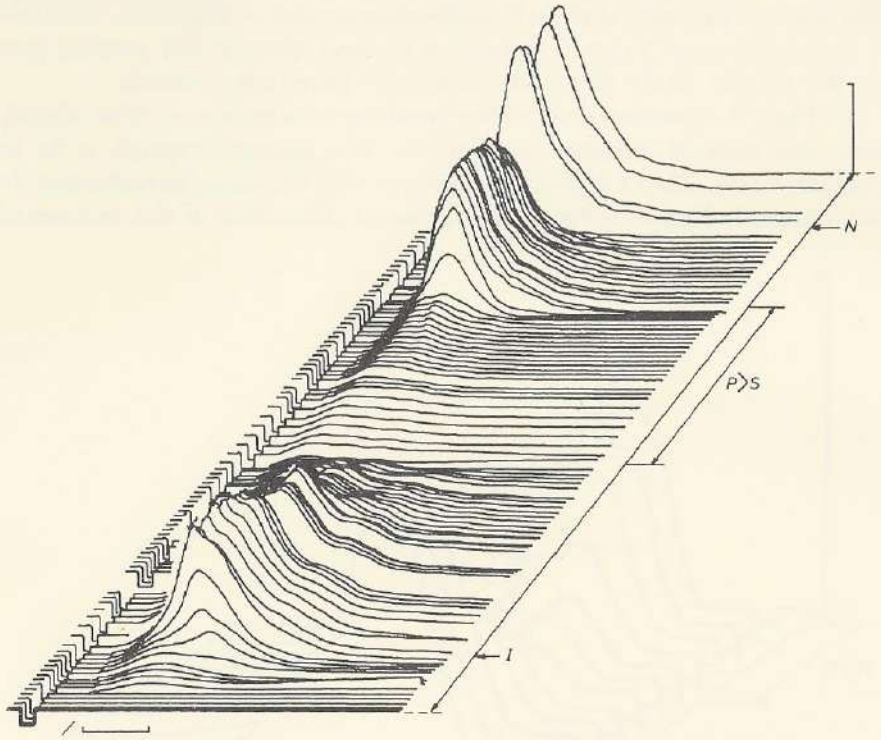


Fig. V–4. One hundred and eighty minutes of infusion compression.
Exp. 48 – SD: 200 μsec – SI: 900 μA – MA: 468 μV – RT: 9 h. 42 min.

connective tissue between the nerve fibers. Plate 4 is a detail of the former microphotograph and demonstrates more clearly the experimentally induced edema between the nerve fibers. Plates 5 and 6 represent other areas in the same nerve. In the last mentioned picture, photographed at high magnification in a section treated with the Mallory-Azan connective tissue technique the dilatations of the endoneural connective tissue between nerve fibers and vascular stasis can be observed. Finally, in Plate 7 the site in the nerve where the needle had been located is demonstrated. Around the insertion site the nerve fibers are packed together. Edema and disruption of parts of the nerve can also be seen. After 4 hours of infusion compression, the histological picture proved to be essentially the same except for differences in degree. Signs of degeneration of nerve fibers and myelin sheaths could not be observed.

Repeated suprasystolic infusion compression

In 2 cats the compression of the nerve was repeated during the same experiment.

In experiment 40 (Fig. V–9), after 98 minutes of infusion compression,

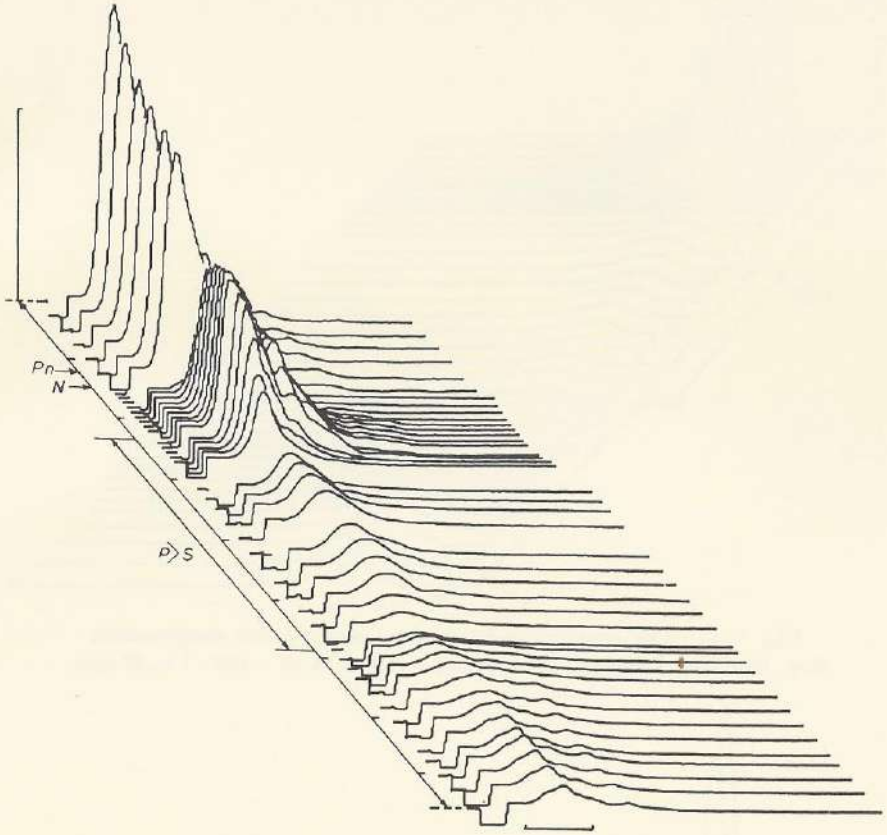


Fig. V–5. Two hundred and ten minutes of infusion compression.
Exp. 60 – SD: 280 μsec – SI: 600 μA – MA: 374 μV – RT: 8 h. 25 min.

TABLE V–1. Suprasystolic infusion compression of the facial nerve for more than 40 minutes, in 9 cats.

Experiment	Action potential after insertion of the needle (percentage of initial value)	Amplitude before applying pressure (μV)	Duration of pressure (minutes)
72	44	96	42
38	64	170	58
55	32	100	76
41	50	108	105
36	50	219	122
48	90	373	180
62	67	604	210
60	70	190	210
67	100	516	245

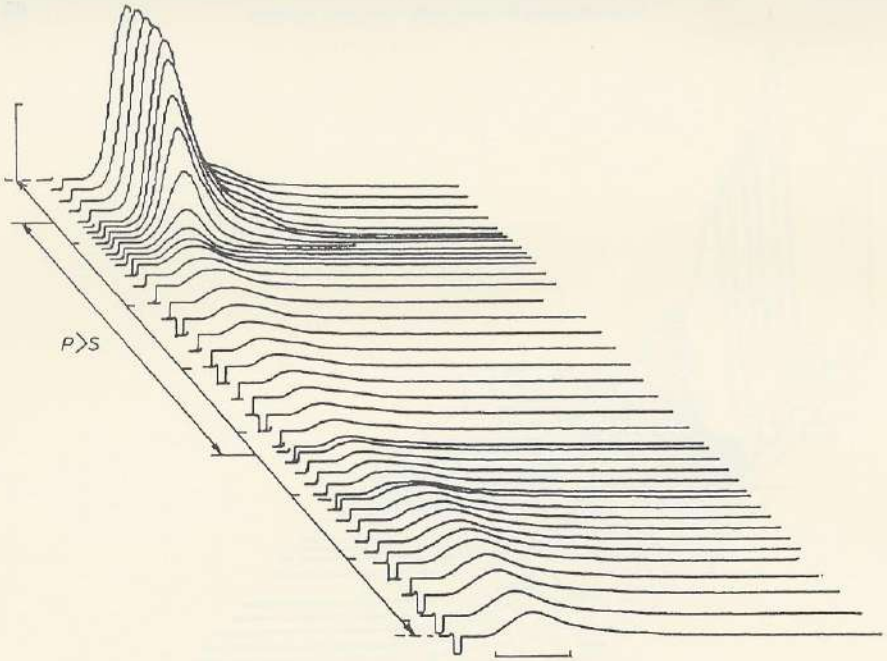
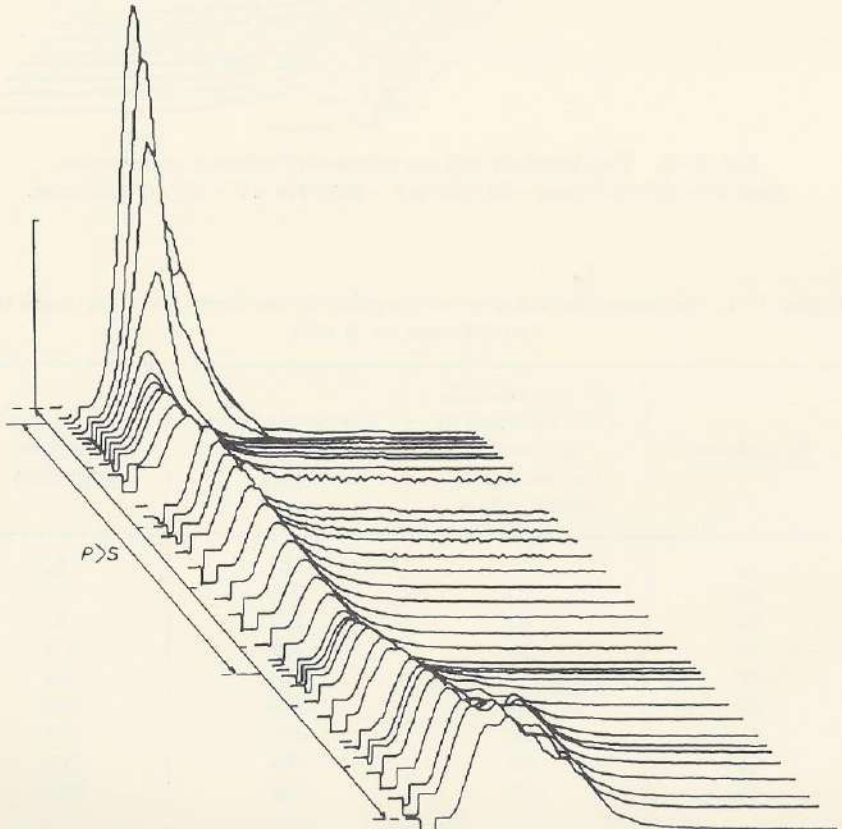


Fig. V–6. Two hundred and ten minutes of infusion compression.
Exp. 62 – SD: 120 μsec – SI: 300 μA – MA: 616 μV – RT: 7 h. 10 min.



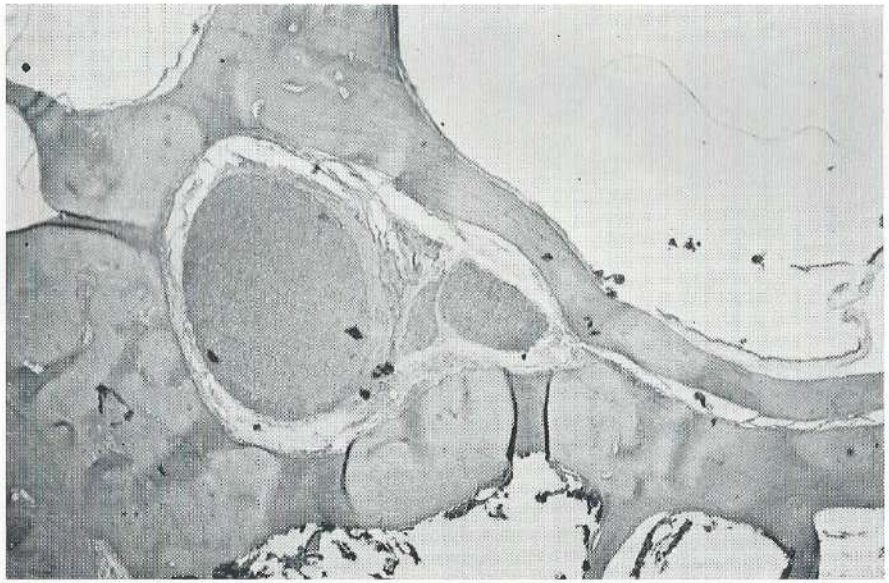


Plate 1. General view of the normal facial nerve of the cat with chorda tympani in the vertical part in a section parallel to the base of the skull. Hem-Phlox, 30×.

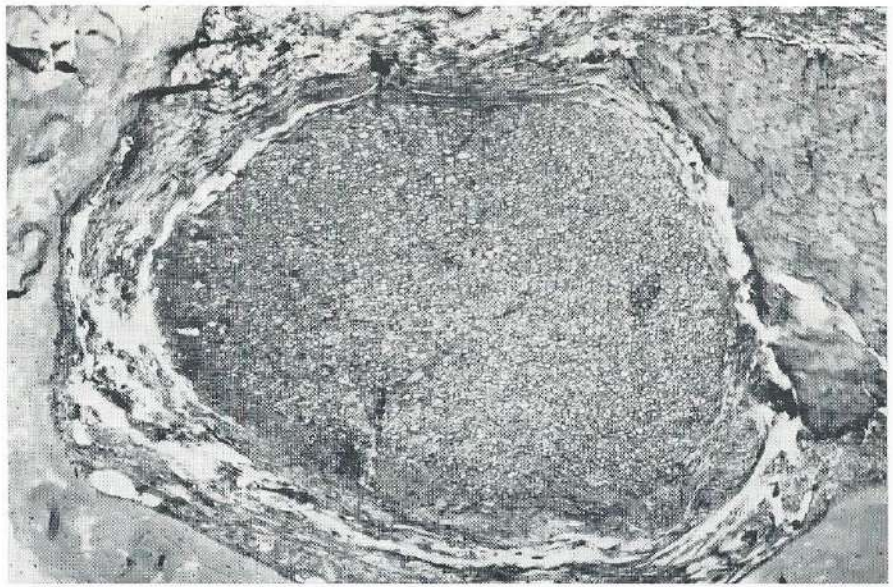


Plate 2. Transverse section of the normal facial nerve of the cat in its descending part. Hem-Phlox, 100×.

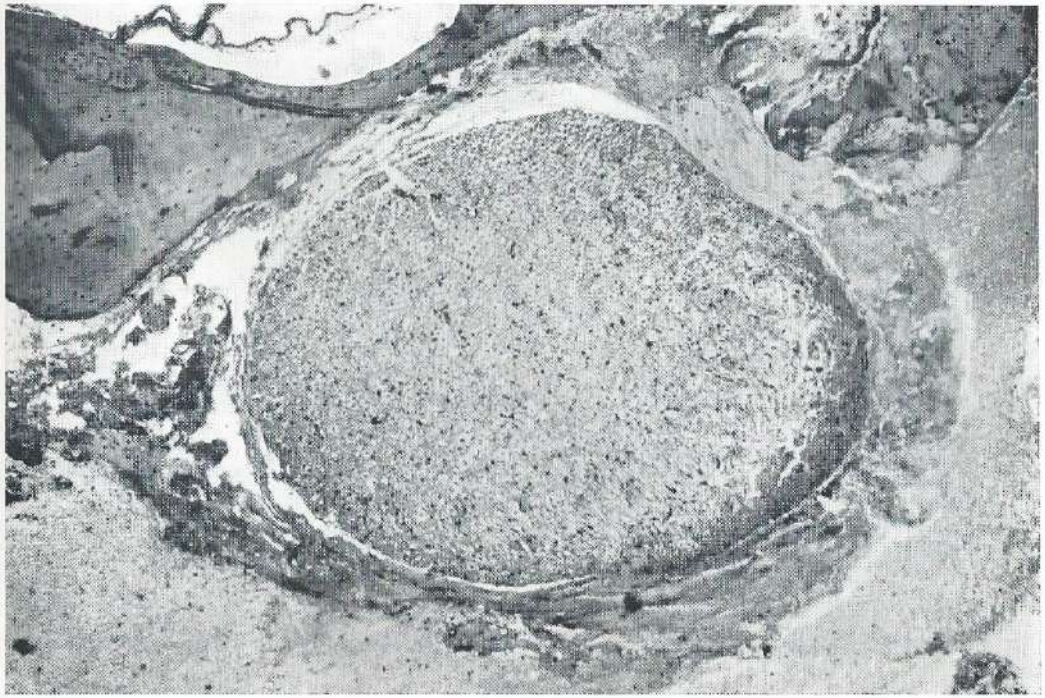


Plate 3. Transverse section of the facial nerve of cat number 79, submitted to compression for one hour and fixed in situ. Hem-Phlox, 100 ×.

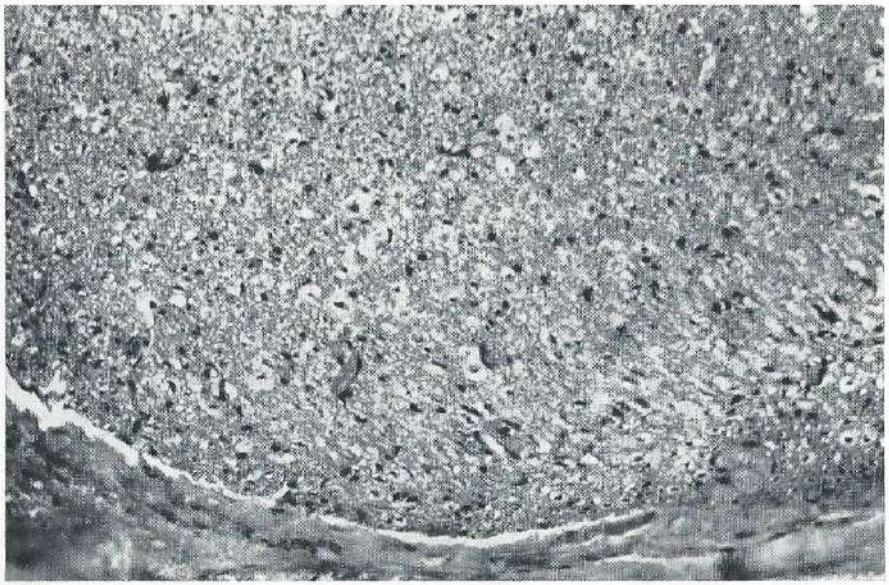


Plate 4. Detail from Plate 3; showing the more condensed part of the infused nerve, with small blood vessels filled with erythrocytes. Hem-Phlox, 180×.

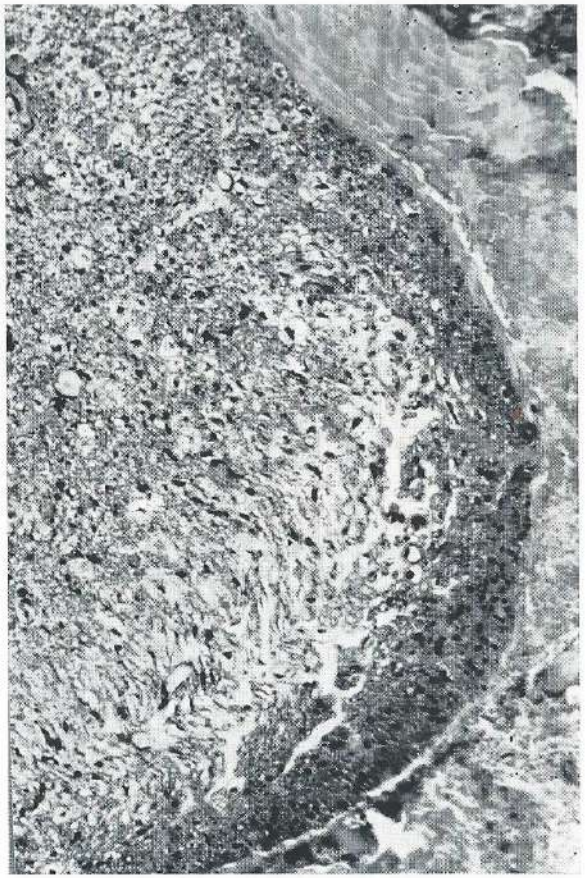


Plate 5. Detail from Plate 3 (cat number 79); part of the infused nerve, showing local dilatation of the endoneural spaces between the nerve fibers, with close packing beneath the perineural sheath. Hem-Phlox, 180 ×.

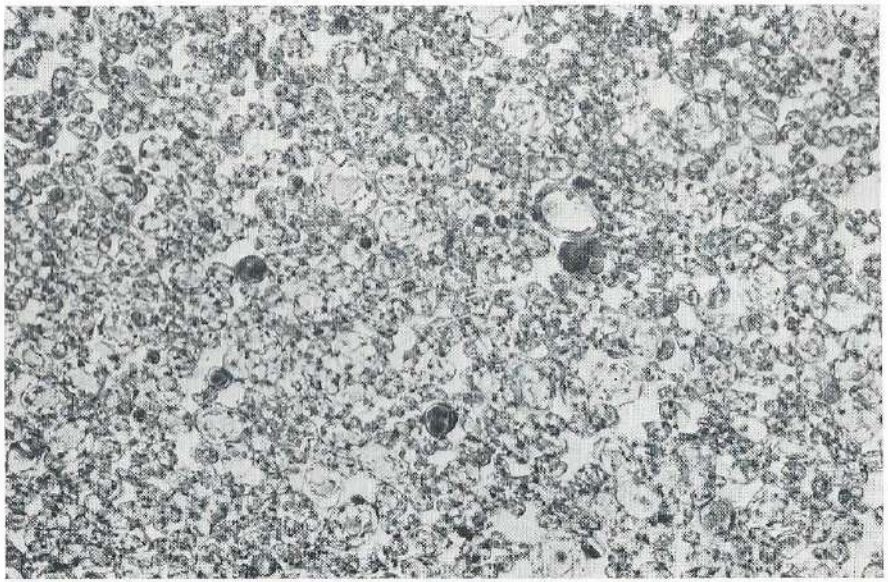


Plate 6. Higher magnification from a part of the nerve from Plate 3; showing dilatation of the endoneural space. Note the filling of small venules and capillaries with a dilatated arteriole. Mallory-Azan, 500×.

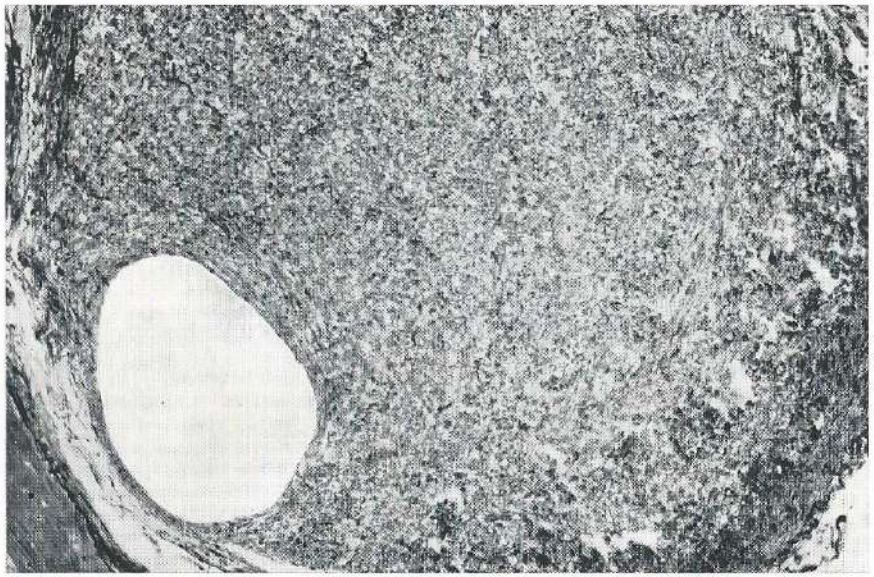


Plate 7. Transverse section of the facial nerve of cat number 84 submitted to infusion compression for one hour, showing the site where the needle was localized with the surrounding compression of the nervous tissue. Mallory-Azan, 180×.

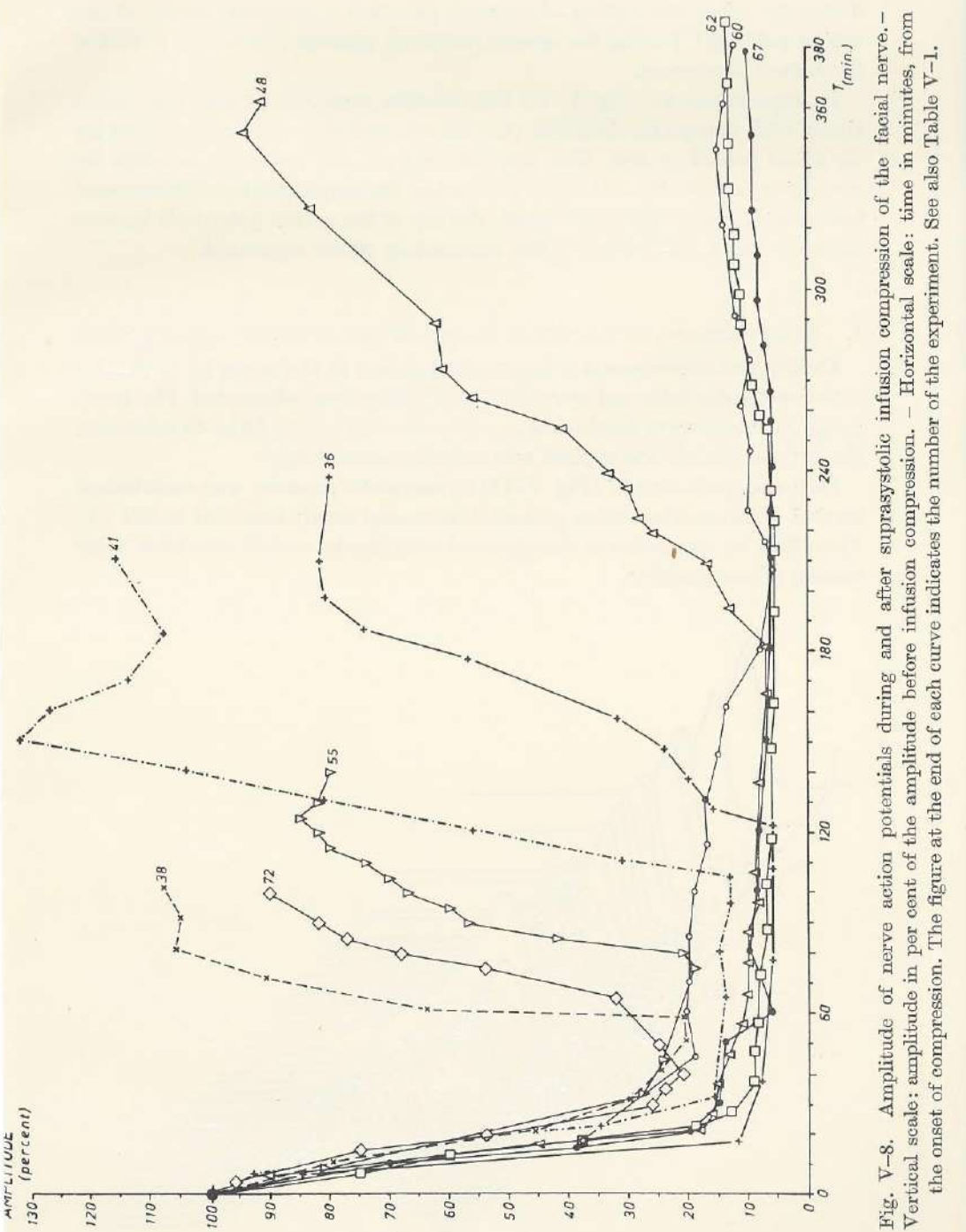


Fig. V–8. Amplitude of nerve action potentials during and after suprasystolic infusion compression of the facial nerve. – Vertical scale: amplitude in per cent of the amplitude before infusion compression. – Horizontal scale: time in minutes, from the onset of compression. The figure at the end of each curve indicates the number of the experiment. See also Table V–1.

pressure was released for 4 minutes. Observation of the action potential 3 minutes after restoration of pressure revealed a complete return of the action potential. During the second period of pressure the action potential fluctuated somewhat.

In experiment 52 (Fig. V–10) the infusion compression was repeated 4 times with increasing duration (10, 20, 40, and 80 minutes), followed by an equal period of rest. The last curves were the result of ischemia by circulatory arrest. It can be seen that, while the amplitude lightly decreased towards the end of the experiment, the top of the action potentials became broader and a deflection in the descending phase appeared.

2. INFRASYSTOLIC, INFRADIASTOLIC, AND MIXED INFUSION COMPRESSIONS

During two experiments infusion compression of the nerve by a pressure between the diastolic and systolic blood pressure was attempted. The blood pressure values were checked at regular intervals (every 20 to 40 minutes); the pressure inside the system was adjusted accordingly.

During experiment 87 (Fig. V–11) infrasystolic pressure was maintained for 243 minutes. The action potential decreased slowly from 294 to 221 μV. The effect of compression disappeared completely within one hour after release of compression.

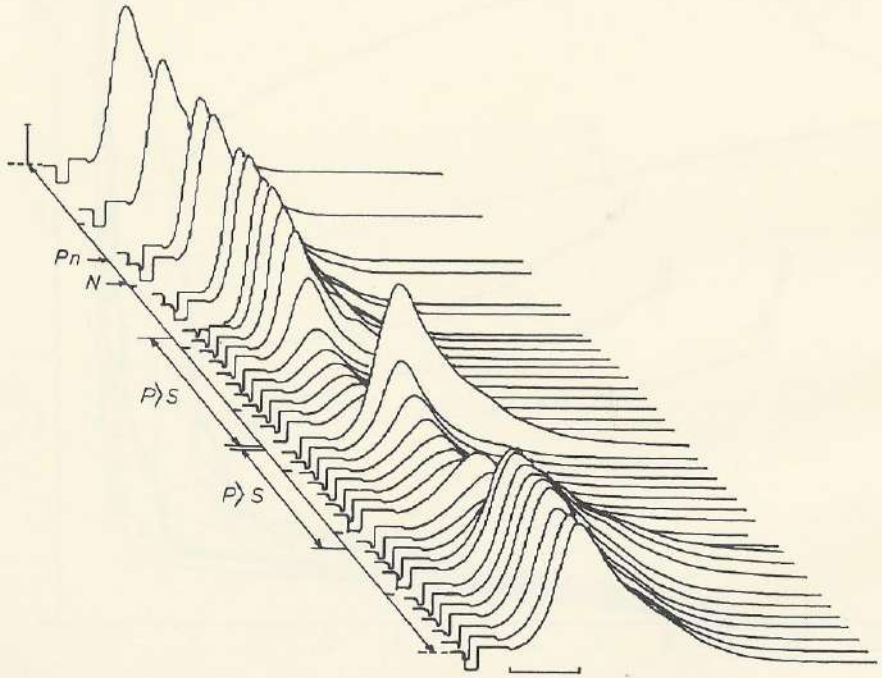


Fig. V–9. Repeated suprasystolic infusion compression.
Exp. 40 – SD: 180 μsec – SI: 400 μA – MA: 987 μV – RT: 7 h. 48 min.

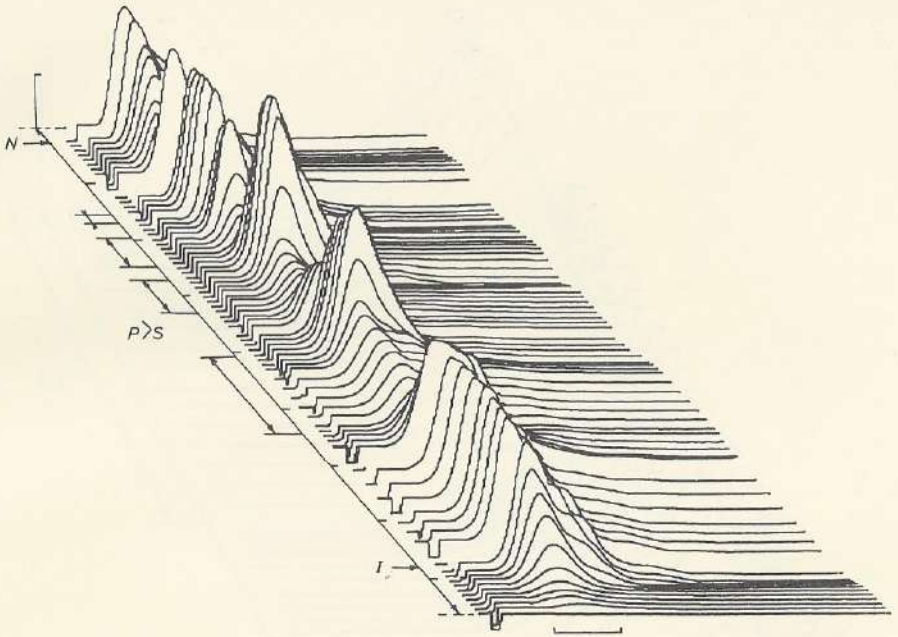


Fig. V–10. Ten, twenty, forty, and eighty minutes of infusion compression followed by an equal period of rest.
Exp. 52 – SD: 150 μsec – SI: 500 μA – MA: 809 μV – RT: 8 h. 35 min.

Experiment 72 is displayed in Fig. V–12. Fig. V–13 gives more details about the experiment. In the upper part of the Figure the blood pressure values are given; in the middle part the latencies to the onset (L_1), to the maximum of the derivative (L_2), and to the center of gravity (L_3) of the maximal action potential; in the lower part the amplitudes of the maximal action potential (A_1), of the "surface" (A_2) and of the derivative (A_3). The time is expressed in minutes after insertion of the needle into the nerve; ischemia was started 500 minutes later.

Infradiastolic compression of 70 mm of mercury during 50 minutes apparently did not influence the action potential. Infrasystolic infusion compression of 125 mm of mercury (between diastolic and systolic blood pressure) was then attempted for 3 hours. After 75 minutes, however, the systolic blood pressure decreased and the infusion compression exceeded the systolic blood pressure. The amplitudes reacted immediately. This situation lasted for about 55 minutes. The blood pressure then increased again and the effect of infusion compression decreased. After 3 hours of compression the amplitude decreased from 91 to 54 μV. After release of pressure the amplitudes decreased abruptly (A_1, A_2), one latency parameter increased (L_3), one decreased (L_2) and the last one was not influenced (L_1). Thereafter a return to precompression level was seen.

Finally, experiment 68 (Fig. V–14) is described because of combined infrasystolic pressure followed by 4 hours of suprasystolic pressure.

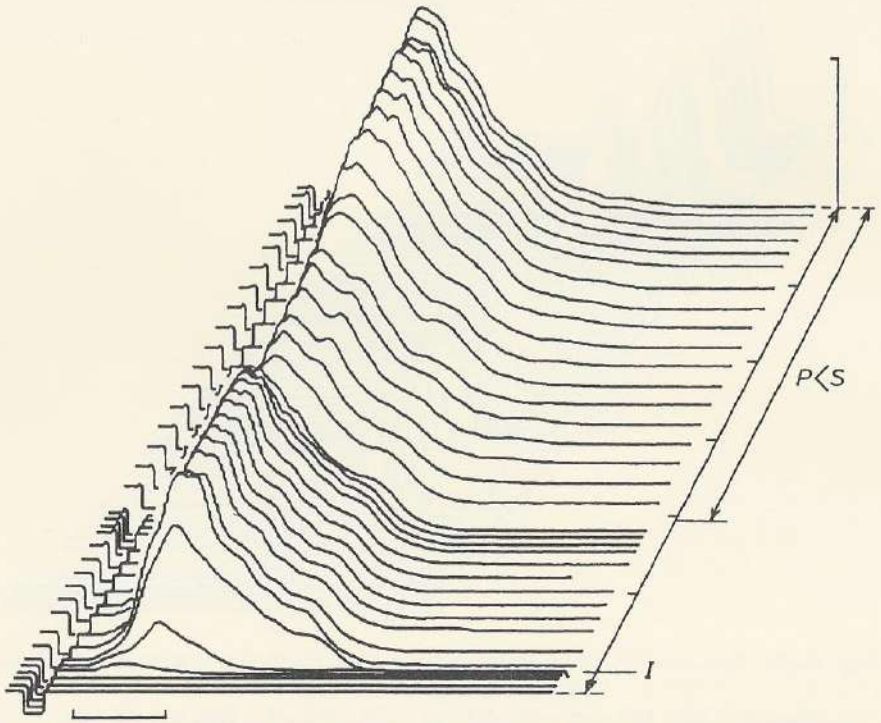


Fig. V–11. Infrasystolic compression of 243 minutes duration. (P < S: infrasystolic compression).
Exp. 87 – SD: 200 μsec – SI: 500 μA – MA: 292 μV – RT: 6 h. 3 min.

Twenty-four minutes of infrasystolic pressure was followed by a slight decrease in amplitude of the nerve action potential (223 to 194 μV). During subsequent suprasystolic infusion the action potential decreased rapidly, following the criteria described for successful suprasystolic infusion compression. After about 3 hours a distinct deterioration appeared in the nervous activity. Two hours after release of pressure no nervous activity could be recorded.

3. INFLUENCE OF COMPRESSION AND ISCHEMIA ON THE FACIAL NERVE

In eleven cats the influences of pressure and of ischemia was recorded during the same experiment. A few examples are given in Fig. V–15 a and b. In comparison to compression, stagnant ischemia results in:

1. A more rapid decrease of the action potential.
2. Disappearance of the nervous activity.
3. A faster change in the ascending part of the action potential than in the descending part.

V – 4. DISCUSSION

It was decided to use the infusion compression method of JAIN and SHARMA (1964) for compression of the facial nerve because, as will be

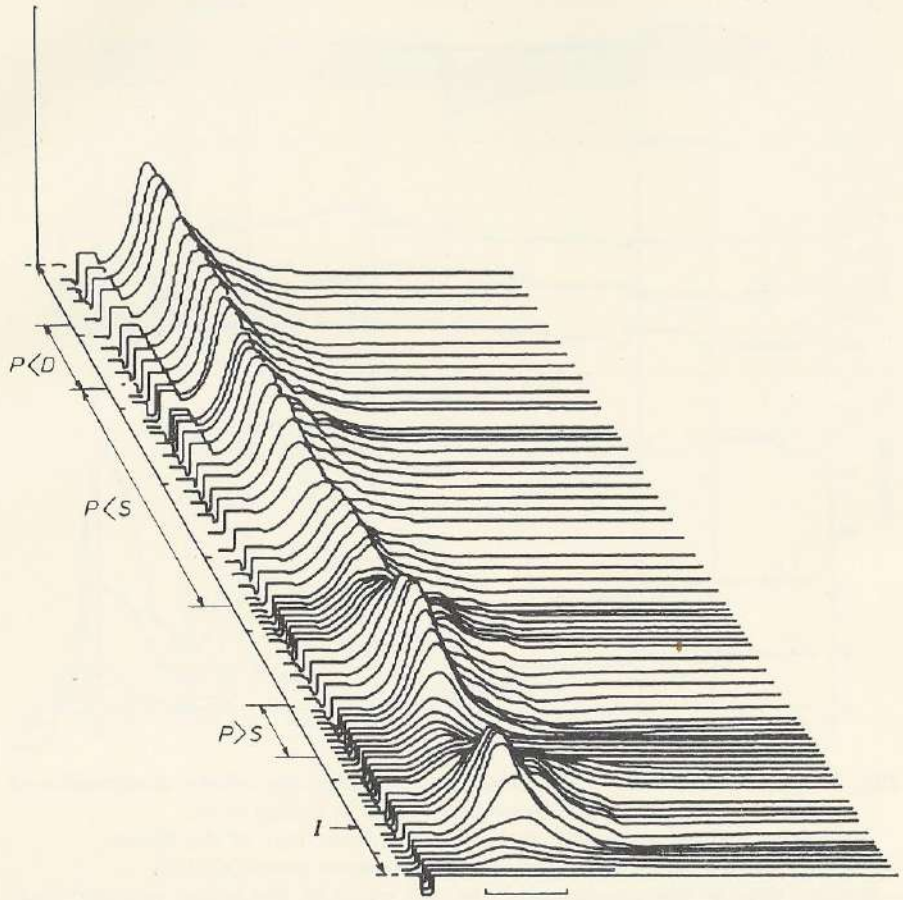


Fig. V–12. Three periods of compression. Infradiastolic (50 minutes), infrasystolic (180 minutes), and suprasystolic compression (42 minutes).
Exp. 72 – SD: 140 μsec – SI: 600 μA – MA: 97 μV – RT: 8 h. 21 min.

explained in Chapter VI, it is generally assumed that the nerve fibers are damaged by edema in ischemic paralysis in man. The same method has been used in two dogs by MCGOVERN et al. (1966); they removed the fibrous collar around the nerve at the stylomastoid foramen and injected a solution of saline into the nerve.

The infusion compression method of JAIN and SHARMA was modified by us as described. With regard to this it should be stressed that in the first place, the facial nerve was totally compressed in its vertical portion, whereas only a group of fibers herein was stimulated and, secondly, that the influence of infusion compression was judged by observation of the nerve action potential in acute experiments and not by its effect on the innervated muscles. On the other hand, our experimental setup enabled us to perform the experiments under standardized conditions, so that the experiments are well comparable between each other.

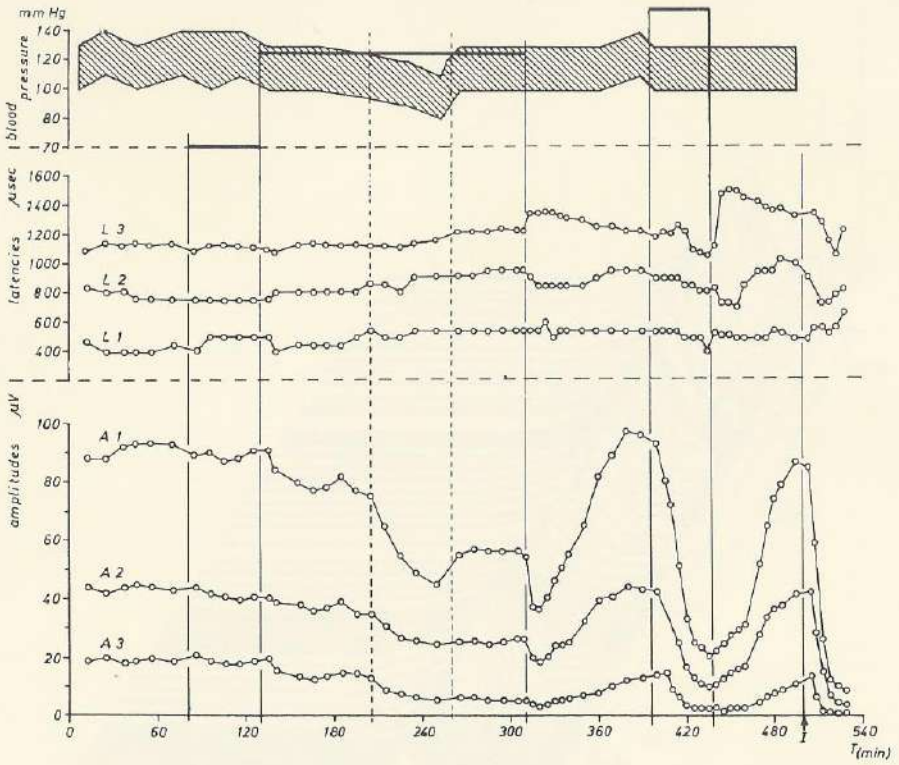


Fig. V–13. Infradiastolic, infrasystolic, and suprasystolic infusion compression of the facial nerve. Same experiment as in Fig. V–12.

Six parameters are presented; from upper to lower part of the Figure:
- latency time to the center of gravity of the action potential (L_3);
- latency time to the maximum of the derivative of the action potential (L_2);
- latency time to the onset of the action potential (L_1);
- maximum amplitude of the action potential (A_1);
- "surface" of the action potential (A_2);
- amplitude of the derivative of the action potential (A_3);

Stimulus; 140 μsec, 600 μA.

Between 360 and 370 minutes the stimulus duration was adjusted from 145–140 μsec.

1. SUPRASYSTOLIC INFUSION COMPRESSION

Electrophysiology

Only the experiments following the criteria described before (V-3) will be considered for the following reasons. (a) An amplitude of less than 100 μV after insertion of the needle could have indicated too heavy damage to the nerve even before compression was started. (b) A slow decrease in amplitude, less than 70 per cent in the first 30 minutes of compression, could have indicated insufficient increase of pressure inside the nerve by clogging of the needle, a bad position of the needle in the nerve or insufficient pressure in the pressure system in relation to the blood pressure.

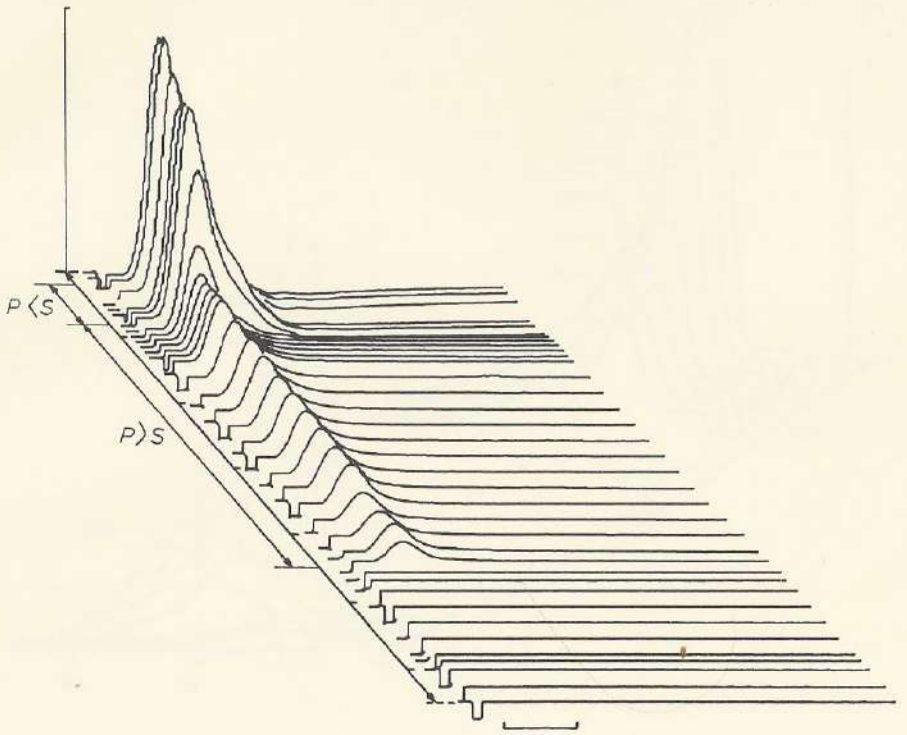


Fig. V–14. Infrasystolic compression of 24 minutes duration, followed by 4 hours of suprasystolic compression.
Exp. 68 – SD: 140 μsec – SI: 150 μA – MA: 223 μV – RT: 6 h. 30 min.

(c) A residual amplitude of less than 20 per cent was chosen as a "minimum of paralysis"; this is, of course, an arbitrary limit. It could generally be reached after about 40 minutes of compression.

It was assumed, by doing so, that the observed changes in action potentials could then reasonably be attributed to suprasystolic infusion compression.

The difference between the normal action potential and that observed after a pressure block of the nerve must be due either to inactivity in nerve fibers or to slower conduction in the depressed area, or to both (Gasser and Erlanger, 1929). Generally the action potential decreased as a whole which would suggest that compression caused inactivity in the nerve fibers rather than slowing of conduction. Most authors, though, agree that the largest nerve fibers are the most susceptible to pressure (Gasser and Erlanger, 1929; Bentley and Schlapp, 1943b; Ogushi, 1969; and others). Although the different fiber types are represented in a single curved action potential, due to the short distance of conduction, the top of the action potential which includes the activities of the largest fibers seemed to be the most affected. The nerve fiber groups, however, are not

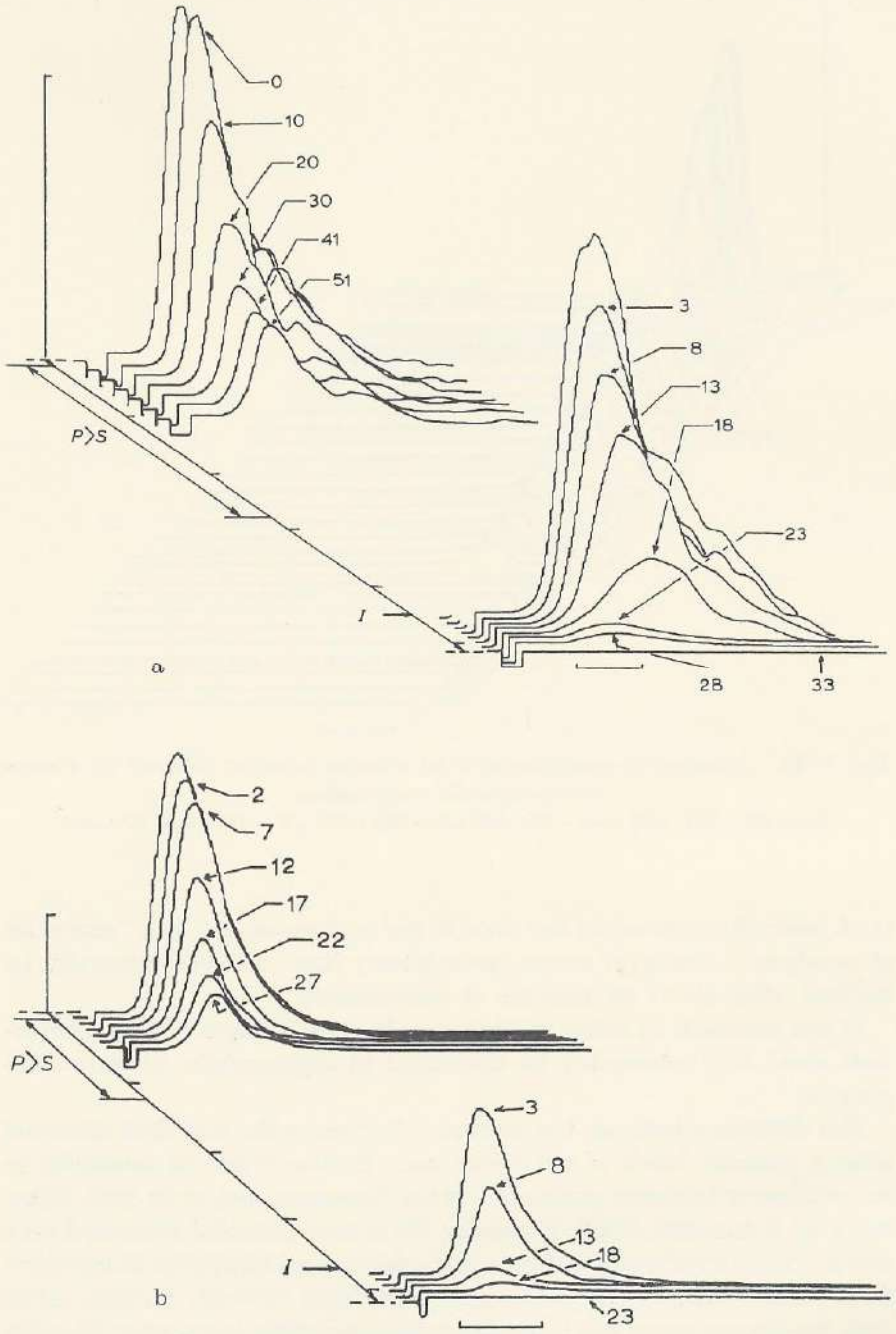


Fig. V–15. Recordings of influence of suprasystolic compression and ischemia during the same experiment. The numbers indicate the duration of compression or ischemia, in minutes.

a. Exp. 45 – SD: 300 μsec – SI: 700 μA – MA: 347 μV – RT: 5 h. 6 min. Fig. V–15.

b. Exp. 56 – SD: 150 μsec – SI: 800 μA – MA: 640 μV – RT: 4 h. 7 min.

sufficiently differentiated in the action potential to confirm or deny this concept.

The *changes due to the compression* were most obvious in the amplitudes, especially with regard to the maximal amplitude of the action potential. The other calculated amplitudes followed an equal course, but less pronounced. There was no constant pattern in the behavior of the latencies. The action potential never disappeared completely during infusion compression; at least 10 per cent of the activity remained visible. Maintenance of conduction in nerves under compression has been explained by diffusion of oxygen from surrounding tissue or nerve (Lewis et al., 1931; Bentley and Schlapp, 1943b). It is highly probable, however, that the action potentials excited in the uncompressed part of the nerve before the block are responsible for the potential changes at the recording electrode, giving the residual signal. It should be stressed that the stimulating electrode, the recording electrode, and the indifferent electrode were located at a small distance (about 5 cm) from each other.

On *release of pressure* characteristic changes were observed in the latencies. In the experiments where the amplitude recovered completely an abrupt increase was seen in the latencies to the center of gravity of the action potential and the maximum of the derivative; afterwards they returned to normal. In the other experiments (the numbers 60, 62 and 67) a small and delayed increase was seen in the latency to the gravity point only. In none of these experiments was a distinct change in the latency of the onset of the action potential seen. If the nervous activity returned, it returned first in the slower (i.e. smaller) conducting fibers or in larger fibers conducting more slowly than normal.

When reviewing the experiments (in Fig. V–8) it can be seen that after a relatively short period of infusion compression, the nerve action potential returns immediately after release of the pressure. Occasionally it even exceeds the initial amplitude (experiment 41). The longer the infusion lasted, the slower was the return of the nervous activity. In experiment 48 (Fig. V–4) the action potential recovered after 180 minutes of compression. The change in shape and the delay of the action potential indicate a more profound influence of the infusion compression on the nervous activity. This influence on conduction velocity, however, does not necessarily imply a decrease in normal peripheral function (Mayer and Denny-Brown, 1964). After compression of 210 and of 245 minutes duration no fast return of action potential could be observed. This is in sharp contrast with our other experiments. Even in experiment 48, the amplitude of the action potential already recovered for 50 per cent in a period of 80 minutes after release of pressure. This cannot be attributed to a much higher degree of infusion compression in these experiments, although no precise measurements hereof were performed in the first experiments. Neither could there be observed a relationship to excessive

damage of the nerve by insertion of the needle or to the decrease of amplitude before application of pressure.

Three and a half hours appeared to be the critical duration of suprasystolic infusion compression compatible with early functional recovery of the facial nerve. It should be noted that after 180 minutes of compression the form of the action potential after recovery was different from the normal shape while after shorter durations of compressions no such effect was seen. The decline in capacity of complete recovery may well have started after 180 minutes of compression.

Our results differ from those obtained by JAIN and SHARMA (1964) in the following aspects:

1. The insertion of a hypodermic needle into the nerve of the rabbit did not produce any subsequent observable functional loss in the experiments of these authors. In our hands and under observation of the action potential, insertion of the needle proved to be very delicate; it often resulted in serious damage to the nerve function. It was therefore made less traumatic by polishing the needle and fixation of it on a micromanipulator.

2. With JAIN and SHARMA, a paralysis lasted 2 to 4 days after 5 to 7 minutes of infusion compression of 100 to 120 mm of mercury and as long as 7 to 10 days after compression of 8 to 10 minutes. The authors concluded that the duration of infusion compression and the time taken for the onset of complete recovery were directly proportional. Since pulsating pressure was exerced with the needle inside the facial nerve with a syringe, damage to the nerve fibers was most likely to occur. The method as used by these authors may therefore not have been sensitive enough to detect early damage to the nerve. The long-lasting effects of very short compressions as observed by these authors might have been caused by mechanical damage of the nerve fibers and the unphysiological saline solution rather than by nerve blocks by edema.

Histopathology

The histopathological lesions described for in our experiments illustrate that the fluid really entered the nerve. The nerve could even be disrupted in some places by the procedure; this could be due, however, to insertion of the needle. The vascular system showed signs of occlusion too, as demonstrated by the vascular stasis observed. Nerve fiber degeneration could not be demonstrated; this does not seem strange, however, in view of the short period of observation. DENNY-BROWN and BRENNER (1944a) observed no change in myelin or axis cylinders after compression by a tourniquet or after direct pressure on the nerve as high as 200 cm of mercury, in all cases in which the animal was killed on the same day of the experiment. On the other hand, conductivity in nerves may be restored before the normal histology has been restored and nerve fibers with a grossly modified myelin sheath structure may continue to function efficiently (DENNY-BROWN and BRENNER, 1944a, b). Two weeks after a

tourniquet lesion of 450 to 800 mm of mercury disappearance of myelin over short segments was demonstrated, beginning at the nodes of Ranvier, with preservation of axis cylinders (MAYER and DENNY-BROWN, 1964).

As reviewed before (Chapter I), ischemic lesions of nerves by compression may be grouped into three categories (SUNDERLAND, 1968), although mixed forms are common:

1. Transient conduction block followed by complete and rapid recovery on release of pressure.

2. Prolonged conduction block with delayed recovery. If the compression is only arrested, but not relieved, the lesion enters a chronic phase with increasing fibrosis. The nerve becomes swollen and hyperemic proximal to the lesion.

3. Nerve fiber degeneration followed by regeneration. If the endoneurium is preserved, the return of regeneration is delayed but complete. In case of destruction of the endoneurium and disorganisation of the internal architecture of the nerve trunk extensive fibrosis results. Recovery is then incomplete or even negligible.

After infusion compression up to about 180 minutes, recovery was rapid and complete in our experiments (first degree of ischemic lesion). After longer infusion compression a more severe functional lesion resulted.

Prolonged block of conductivity could also have resulted in a complete, but delayed, recovery. The degree of nerve lesion, however, could not be demonstrated by microscopical examination.

2. REPEATED SUPRASYSTOLIC INFUSION COMPRESSION

In experiment 52 (Fig. V–10) with 4 periods of compression of increasing duration, the amplitude of the action potential recovered completely but the shape of the action potential progressively changed toward the end of the experiment. The effects of short periods of compression were not compensated by equal periods of rest. A similar effect was demonstrated after repeated ischemia with a sphygomanometer (BARLOW and POCHIN, 1948; ABRAMSON et al., 1971).

3. INFRASYSTOLIC INFUSION COMPRESSION

Infrasystolic compression above the diastolic blood pressure had little influence on the nerve action potential (Fig. V–11); as soon as it exceeded the systolic blood pressure the influence of suprasystolic compression became distinct (Fig. V–13). Theoretically, it could have been provoked by occlusion of the venous return of the nerve with preservation of the arterial supply.

BENTLEY and SCHLAPP (1944b) also observed some interference with conduction in the sciatic nerve of the cat, by direct pressure of 120 mm of mercury. This effect was variable in different animals (CAUSEY and PALMER, 1949).

4. INFRADIASTOLIC INFUSION COMPRESSION

Infradiastolic infusion compression did not influence the nerve function in our experiments. These findings agree with observations of other workers (LEWIS et al., 1931; BENTLEY and SCHLAPP, 1943b; CAUSEY and PALMER. 1949). BENTLEY and SCHLAPP (1943b) explained the persistence of a normal nervous function in these cases by assuming the occurrence of diffusion of oxygen from the neighboring vascularized parts of the nerve. These authors stated that the circulation inside the nerve was arrested because they could not observe macroscopic evidence of circulation after injecting Evans blue intravenously.

5. MIXED INFUSION COMPRESSION

After twenty-four minutes of infrasystolic, followed by three hours of suprasystolic infusion compression, a distinct deterioration in the nerve function appeared (Fig. V–14). Disappearance of nerve function after release of pressure has occasionally been observed (BENTLEY and SCHLAPP, 1943b).

6. INFLUENCE OF SUPRASYSTOLIC INFUSION COMPRESSION AS COMPARED TO ISCHEMIA

The action potential decreased faster after ischemia than after infusion compression. This can be explained by the fact that infusion compression required about 40 minutes to be maximally effective. This time apparently was needed to build up a nerve block by accumulation of a sufficient amount of fluid between the nerve fibers. That the nervous activity did not disappear completely in case of compression can probably be attributed to action potentials generated in the intact part of the nerve fibers distal to the nerve block.

The earlier blocking of fast nerve fibers by ischemia could also explain the earlier decline in general by ischemia. Moreover the pressure block was localized in a small part of the nerve, whereas ischemia involved the total length of the nerve under study.

GELFAN and TARLOV (1956) stated that reversible conduction block by mechanical pressure is due to mechanical deformation of the nervous tissue and not to lack of oxygen. This conclusion was drawn from observations of compressed nerves in vivo and ischemic nerves in vitro. In fact nerves cannot be compressed in vivo without interference with their circulation.

CONCLUSION.

Although the effects of infusion compression can be compared to those after compression by other methods (as described in Chapter 1), differences in the methods have been noted.

In tourniquet paralysis (DENNY-BROWN and BRENNER, 1944a), the amount and duration of pressure necessary to cause persisting paralysis were extremely variable. Complete recovery in 1 to 2 hours after 4 hours

of compression has been observed (BENTLEY and SCHLAPP, 1943a). These differences can be explained by the differences in magnitude of the deforming forces developed by a tourniquet, by the compartmental structure of the nerve trunk at the site of compression, and by the inevitable ischemia of the surrounding tissues (SUNDERLAND, 1968).

BENTLEY and SCHLAPP (1943b) considered about 130 mm of mercury as a critical value for production of nerve block applied for 2 to 3 hours by means of pneumatic cushions around the sciatic nerve in the cat. The authors argued that this was not related to the carotid blood pressure, because the blood pressure in the small vessels was thought to be of the order of one half of that in the carotid artery.

As demonstrated in experiment 72 (Fig. V–12 and 13) the nerve action potential reacted sharply on small variations in blood pressure around the systolic blood pressure. This also proves that the actual pressure inside the nerve corresponded to the pressure in the infusion compression system.

The following conclusions can be drawn from the experiments:

1. It is possible to paralyse a nerve by injecting fluid into it. Although compression of a nerve within its perineurium is said to be unknown in neurology (DRACHMAN, 1969) the method seems to be effective for this purpose. Attention was directed to the method by JAIN and SHARMA (1964). The method permitted compression without ischemia of the surrounding tissues and without artificial isolation of the nerve in an apparatus outside its normal anatomical relationships; it can therefore be considered less artificial.

2. The effect of infusion compression is clearly related to the blood pressure of the animal. The conclusion therefore is inevitable that the arterial circulation of the nerve plays a determining role. This confirms the theory that the initial changes produced by localized pressure are due to the associated ischemia, even if later and more pronounced structural changes are the result of the anatomical deformation brought about by the compression.

3. About three and a half hours of suprasystolic infusion compression are critical for the nerve. Release of compression is followed no more by a short term recovery as seen after shorter periods of compression. The microscopical examination, however, did not permit a detailed description of the degree of nerve lesion.

PART THREE

CHAPTER VI

ISCHEMIC PARALYSIS OF THE FACIAL NERVE IN MAN

VI – 1. INTRODUCTION

The anatomy of the facial nerve and ischemic paralysis of it are considered. Operative treatment of this condition in the acute phase is discussed.

VI – 2. ANATOMICAL CONSIDERATIONS

Before we proceed to the discussion related to ischemic paralysis of the facial nerve, a short review of the macroscopic and miscroscopic anatomy of the facial nerve inside the human temporal bone is necessary.

NERVE FIBERS AND NERVE SHEATH.

From the relation between the number of cells in the geniculate ganglion and the facial nucleus the conclusion can be drawn that the human facial nerve is composed of motor fibers for the mimetic musculature (58 per cent), of general visceral efferent (preganglionic) fibers for the submandibular and the pterygopalatine ganglions (24 per cent), and of sensory fibers (18 per cent). In the cat these percentages are respectively 80, 2, and 18. The average number of fibers distal to the geniculate ganglion is about 13,000; 83 per cent of these fibers is myelinated. The nerve contains an average of 7,000 motor fibers; most fibers in man have a diameter between 7 and 10 μ, in the cat it varies from 3 to 7 μ. All the above-mentioned data are given by VAN BUSKIRK (1945).

Histograms of myelinated fibers of different sizes in persons of various ages did not show a consistent pattern (KULLMAN, 1971).

According to BRODAL (1969) relatively little is known about the afferent innervation of the facial muscles. Although one would presume that the mimetic muscles are amply provided with proprioceptors, the number of muscle spindles in the mimetic muscles appears to be small. It is also unknown along which route the afferents from the muscle spindles and other proprioceptors reach the brain stem.

SUNDERLAND and COSSAR (1953) noted characteristic variations in the arrangement of the nerve fibers with respect to the supporting tissue between different parts of the nerve.

In the *internal auditory meatus* a small amount of fine connective tissue

is arranged about and through the nerve, so that the latter is surrounded by a very delicate epineural sheath and the fibers are grouped into fascicles. The nerve occupies from 12 to 19 per cent of the cross-sectional area of the meatus.

In the *facial canal* the nerve fibers are collected into a single fascicle distally to a point varying between the origin of the branches to the stapedial muscle and those to the chorda tympani. Beyond this point the nerve is mostly composed of a number of fascicles.

In the *monofuniculated* part, proximally to the geniculate ganglion, the bundle is covered with a thin perineurium. Distally to the ganglion this perineurium is well defined peripherally, a thicker but less dense epineural coat separates the perineurium in the way of an adventitia from the periosteum of the canal. The funiculus occupies 25 to 50 per cent of the cross sectional area of the bony facial canal. In the *multifuniculated section* in the facial canal the nerve bundles are not arranged in totally parallel strands but form a stretched out plexus; this results in local changes in the funicular pattern. Each fascicle has a well defined perineurium. A loosely arranged epineurium separates and surrounds these bundles and is not condensed externally to form a dense limiting sheath. The funiculi occupy 30 to 50 per cent of the canal; the remainder is occupied by extrafunicular blood vessels and by connective tissue. The latter is more loosely arranged than the external sheath around the nerve in the monofascicular part.

SUNDERLAND and COSSAR conclude: "The amount and arrangement of the extrafunicular connective tissue is such that any swelling involved would, in the initial stages, be resisted by the perineurium and not by the epineurium or the bony canal".

According to JAMES (1961) the perineurium between the stylomastoid foramen and the stapedial nerve is very thick in the human facial nerve (25 to 35 μ); it is formed by concentric layers of dense collagenous connective tissue with few elastic fibers (Plate 8). More proximally, in the horizontal part, the external nerve sheath is much thinner (5 to 8 μ) and has a looser texture (Plate 9).

THE STYLOMASTOID FORAMEN

There is no uniform agreement about the local narrowing of the bony facial canal at the stylomastoid foramen, as reported by CAWTHORNE (1946). This finding could not be confirmed by SUNDERLAND and COSSAR (1953); there is also no increase of connective tissue at this point (LINDEMAN, 1960).

THE CHORDA TYMPANI

The origin of the chorda tympani from the facial canal may vary from 1.2 mm distal to 10.9 mm proximal to the stylomastoid foramen; in 75 per cent of the cases the branches arise more than 3 mm proximally to the

foramen. The chorda tympani may also sprout from the main nerve stem outside the temporal bone. The nerve branch is composed almost exclusively of small myelinated fibers (KULLMAN et al., 1971).

VASCULARIZATION

Within the facial canal the proximal part of the nerve is supplied by the petrosal branch of the middle meningeal artery and the distal part by the stylomastoid artery (AUDIBERT et al., 1936).

Between the brain stem and the geniculate ganglion small branches of the internal auditory, the anterior inferior cerebellar and the basilar artery supply the nerve.

According to SUNDERLAND and COSSAR (1953) the branches of the nutrient arteries are situated superficially in the epineurium. From these superficial arterioles arise intraneural arterioles, precapillaries, and capillaries which outline a continuous longitudinal intraneural vascular network. In the multifuniculated section of the nerve the arterioles and precapillaries are also located between the nerve bundles. Finer branches pass into the funiculus or funiculi through the perineurium from which they enter the bundle. The topographical distribution of the vasa nervorum is such that the nerve is supplied by a series of anastomosing arterial systems in such a way that, at any level, the nerve is supplied from two separate sources. This arrangement ensures an adequate blood supply to the nerve in the event of occlusion of either one of the contributing systems. Following surgical exposure of the nerve the longitudinal arrangement of the larger vessels on the surface of the nerve might be mistaken for hemorrhagic streaks as evidence of pathological change.

The junction between the stylomastoid artery and the superficial petrosal artery usually occurs near the junction of the middle and upper thirds of the descending part of the facial canal (NAGER and NAGER, 1953).

According to BLUNT (1954), the intrinsic vascular anatomy distal to the geniculate ganglion resembles that seen in peripheral nerves in limbs; according to this author no anatomical evidence exists of a vulnerable blood supply to this section of the facial nerve which would especially predispose it to vascular disturbances.

Immediately proximal to the geniculate ganglion no anastomoses between the arterial systems are encountered other than through the capillary plexus of the facial nerve; this section of the nerve is therefore considered by BLUNT (1956) as the one weak link in an otherwise continuous arterial anastomotic chain. In the geniculate ganglion irregular capillary and precapillary dilatations are present (BLUNT, 1954; CLARKE, 1965).

BOSATRA (1956) observed intima cushions with muscular cells along the main arteries of the facial canal and arteriovenous anastomoses along the smallest arteries of the periosteum and the perineurium. These structures may be of importance for the blood circulation, serum exudation, and consequent edema.

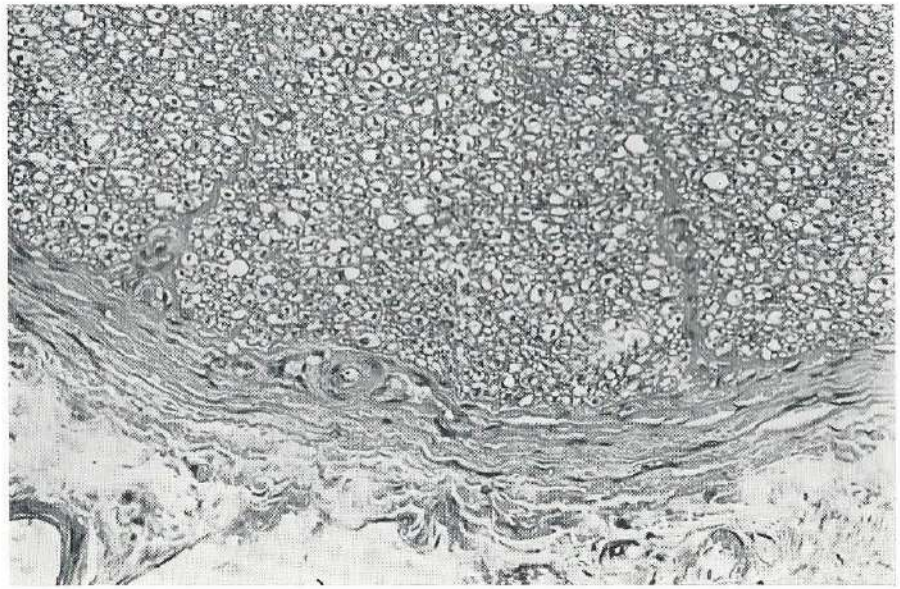


Plate 8. Human facial nerve in its descending part, showing the thick perineural capsula with an arteriole just inside the nerve. Hem-Phlox, 180 ×.
Unpublished microphotograph, courtesy of Dr. J. James, Histological Laboratory, University of Amsterdam.

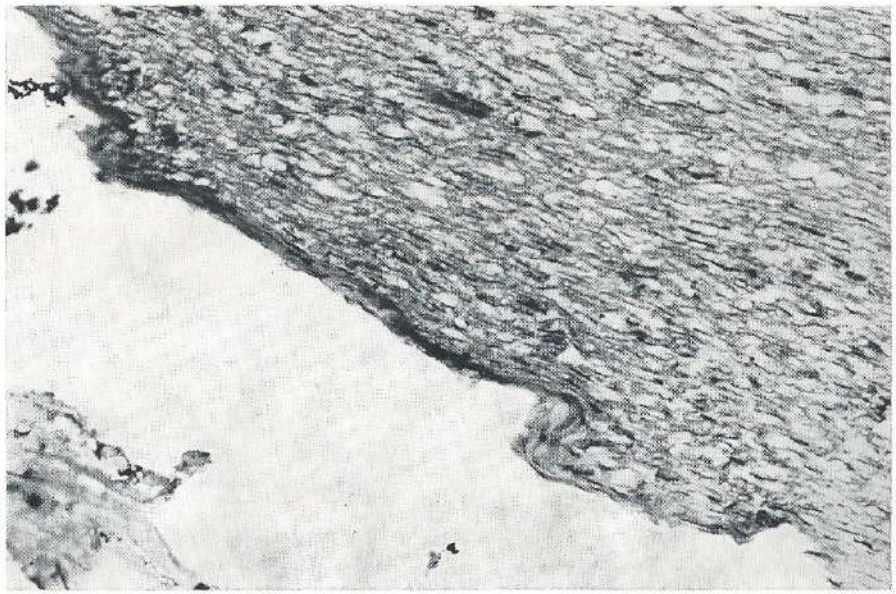


Plate 9. Human facial nerve, horizontal part, of the same series as Plate 8; showing the thin perineural sheath in this part of the nerve. Hem-Phlox, 180 ×, courtesy Dr. J. James, Histological Laboratory, University of Amsterdam.

The intraneural arterioles are situated just inside the nerve sheath. The capillary net ends in small veins in thin septa of connective tissue inside the nerve fascicle. In this way the arterial and venous circulations are separated inside the facial nerve (JAMES, 1961).

CLARKE (1965) performed an X-ray microscopic study of the arterial supply to normal nerves and facial nerves which were idiopathically paralysed for at least 10 years. No ischemic area was detected, but an area of reduced vascularity was noted in the horizontal part of the nerve.

ANSON et al. (1970) observed a striking difference between the extensive circumneural vascularization and the relative avascularity of the nerve itself. The continuity between the vascular network outside the nerve and the intraneural vessels still has to be demonstrated according to these authors. In their opinion segmentally arranged vessels in the category of endarteries and division of the facial canal into compartments could explain the localized nature of edema within the nerve. They remark that microscopic studies of injected specimens which are removed intact together with the temporal bone, in such a way that structures remain in their natural relationships, are needed.

VI – 3. PATHOLOGY

The sudden peripheral paralysis of the facial nerve, as it occurs in man, will be considered now. With this paralysis "there is no obvious cause such as injury, infection or new growth; or there is nothing to suggest a more centrally placed lesion" (CAWTHORNE, 1951).

The name of Charles BELL has been attached to this paralysis; he originally described the facial nerve as the "respiratory nerve of the face" (BELL, 1821).

In addition to "Bell's palsy", the condition is also known as "idiopathic facial paralysis", facial paralysis "a frigore", "rheumatic facial paralysis" and "ischemic paralysis of the facial nerve". The last name has been introduced by KETTEL (1954). Since it seems now to be generally accepted that ischemia is the triggering factor in this paralysis (MIEHLKE, 1960), the name "ischemic paralysis" is used here.

Extensive reviews of the available literature on this disorder have been published by KETTEL (1959), MIEHLKE (1960, 1965) and ZÜLCH (1970).

MACROSCOPIC PATHOLOGY

Most surgeons of the facial canal report swelling of the facial nerve in ischemic paralysis in the lower segment of the descending (mastoid) part of the nerve. The frequency of its occurrence increases as the nerve approaches the stylomastoid foramen (ANSON et al., 1970).

About two months after the onset of the paralysis the swollen and hemorrhagic aspect of the nerve disappears and in cases of paralysis of long duration the nerve sometimes shows a pronounced atrophy (CAW-

THORNE, 1951; JONGKEES, 1954). The swelling and ecchymoses at times may extend to the tympanic portion of the nerve (WILLIAMS, 1959).

According to FISCH and ESSLEN (1970) the most important site of the lesion is situated proximally to the geniculate ganglion. This conclusion was deducted from the swollen and red aspect of the nerve during decompression of the whole facial nerve (in 11 out of 12 cases), from stimulation during operation of different sections of the nerve (in 3 cases), and from preoperative Pantopaque®-cisternography of the internal auditory canal. This has not yet been confirmed by other authors.

Bulging of the nerve after opening of the nerve sheath has been reported in many clinical papers (DUEL, 1932; MORRIS, 1936; KETTEL, 1947; CAWTHORNE, 1946, 1951; and others) and has been regarded as evidence of pathological swelling under pressure (SUNDERLAND and COSSAR, 1953). However, bulging also occurs when the sheath of a normal nerve is incised (SUNDERLAND, 1946). Protrusion of nerve fibers occurs only when the perineurium is opened; the funiculi do not behave in this way as can be shown when the epineurium of a normal nerve is incised (SUNDERLAND and COSSAR, 1953).

According to JONGKEES (1965) the abrupt ending of the bulging of the decompressed edematous nerve, in contrast to the non-bulging part, is the typical aspect of ischemic paralysis.

Normal nerves handled with the necessary care never show this type of circumscript edema.

MICROSCOPIC PATHOLOGY

Microscopic examination of the facial nerve or the chorda tympani in cases of ischemic paralysis (MINKOWSKI, 1891; DÉJÉRINE and THÉOHARI, 1897; JONGKEES, 1954; KETTEL, 1959; MIEHLKE, 1960; and MCGOVERN, 1970) showed edema, degenerative signs of the myelin sheaths (swelling) and less frequently of the axons. The lesions were localized most frequently in the lower part of the mastoid segment of the nerve. No apparent signs of inflammation, such as infiltration by leucocytes, could be found in any cases (JONGKEES, 1957; MIEHLKE, 1960).

JONGKEES (1954) and SADE et al. (1965) found no edema, inflammatory changes, or blood vessel anomalies in biopsies of the epineurium in patients with ischemic paralysis. The appearances were identical with normal postmortem controls.

PATHOGENESIS

BALLANCE and DUEL (1932) suggested that paralysis of the nerve is caused by edema leading to compression of the nerve within the bony canal.

At present the most accepted theory about the mechanism of paralysis is the theory of combined primary and secondary ischemia advanced by KETTEL (1947), HILGER (1949), SULLIVAN and SMITH (1950) and defended by many others. This theory is inspired by the experimental work of

DENNY-BROWN and BRENNER (1944a, b) who stated that conduction failure is primarily a result of ischemia and not of compression. A local arteriolar constriction should then provoke an increase in permeability of the vessels caused by hypoxemia; the ensuing transsudate should then compress the nerve fibers. By impairment of the venous and lymphatic return, more edema fluid could be formed, thus creating a vicious circle of edema and compression. The tendency to arteriolar constriction could originate in the nervous system and be inherited. This could explain familial incidence and recurrences of ischemic paralysis.

Moreover, the dense fibrous sheath of the nerve in the region of the stylomastoid foramen could play a part by preventing collateral arterial supply. Accordingly, treatment must be based on restoration of the circulation in the stylomastoid arterial segments by vasodilating drugs and surgical exposure of the nerve trunk.

According to JAMES (1961) (Plate 8) the dense perineural sheath between the stylomastoid foramen and the stapedial nerve, rather than the bony canal, could be the cause of the predisposition of the facial nerve for vascular disturbances, as the veins might be compressed centrally in the nerve due to the unyielding dense perineural sheath. This would mean that the vicious circle could then be interrupted by incising the nerve sheath after exposure of the nerve and not by merely opening the bony canal.

Electromyographic findings in ischemic paralysis can be explained by a relatively greater loss of the larger motor units as would be produced by ischemia or pressure block (PETAJAN, 1969).

BLATT and FREEMAN (1966, 1969) reject the ischemic origin of the paralysis and ascribe the paralysis to retrograde extension of an inflammation from the chorda tympani from the middle ear to the facial nerve. Their conclusion is based on their observations during operations, submandibular salivary flow studies, and electron microscopy of the chorda tympani and the facial nerve. The findings in the chorda tympani are related to the somatic motor fibers of the nerve trunk: according to these authors transsection of the chorda tympani would prevent degeneration of the facial nerve. Up to now no support has been published for the view of these authors.

ETIOLOGY

An initial spasm of the arterioles provoking edema may be induced by toxic, endocrine, psychic and other factors (MIEHLKE, 1960).

A lower motor neuron paralysis of nuclear origin, possibly of viral etiology, rather than an entrapment syndrome, has been postulated by SADE et al. (1965). This opinion is based on their finding that the continuity of the nerve sheath was not disturbed and on the fact that about 20 per cent of the patients who suffer from an ischemic paralysis show varying degrees of permanent damage. According to these authors, an intact nerve sheath garantees complete recovery if the lesion is entirely in-

franuclear and recovery after decompression should be immediate in case of an entrapment syndrome.

An intact nerve sheath, however, does not exclude intraneural disorganization and, theoretically, decompression of a nerve may relieve a conduction block and favor regrowth of axons to the peripheral muscles.

Subclinical mumps might cause paralysis, as indicated by antibody titer determinations (SAUNDERS and LIPPY, 1959). The finding of epidemics of ischemic paralysis is also said to support the hypothesis of a viral infection (LEIBOWITZ, 1969). However, histological pictures have never demonstrated inflammatory changes.

CONCLUSIONS FROM THE LITERATURE

From the literature on the pathology of ischemic facial paralysis it can be concluded that the prevailing concept concerning the pathogenesis of the paralysis is mainly based on the swollen and red aspect of the facial nerve in the mastoid portion, in the acute phase, and on microscopical observations. However, this swelling may be absent or localized in another segment of the nerve. Bulging of the nerve after incision of the nerve sheath, considered as evidence of increased pressure inside the nerve, has also been reported in normal nerves though not in its typical abruptly ending form.

Although microscopical examination almost always revealed degeneration of the nerve without signs of inflammation, a few examples of inflammatory changes have been reported. Moreover, it is not easy to decide whether the difficult diagnosis per exclusionem is well-founded.

The opinion that the nerve is compressed, was followed logically by attempts to relieve the pressure by opening the facial canal and slitting the nerve sheath. It should be noted that only very few attempts have been made to measure the pressure inside the canal or inside the nerve (JONGKEEZ, 1968).

The mechanism of the paralysis is mostly explained by the following sequence of events: arteriolospasm, ischemia, edema, compression, which again induces ischemia. The particular anatomy of the facial canal and the facial nerve seems to play an important part in the paralysis, but opinions differ widely on details concerning the way the nerve might be predisposed for vascular disturbances.

Different causes might start the vicious circle of ischemia and compression so that an unambiguous explanation probably will never be found, different factors being capable of triggering the same pathogenetic mechanism (ZÜLCH, 1970).

No definite conclusion about the pathology of ischemic paralysis can be reached without studies of normal temporal bones (ANSON et al., 1970), more autopsy studies by advanced histological techniques (FOWLER, 1963) and careful examination of the patients to diagnose or exclude virus infections (SAUNDERS, 1963; PARK and WATKINS, 1949; KETTEL, 1947),

diabetes mellitus (LEIBOWITZ, 1966), and nervous system disorders (DRACHMAN, 1969).

Cases of ischemic paralysis resemble each other "like peas in a pod" (TAVERNER, 1965). Any variation from the usual course should cause the investigator to have doubts about the diagnosis.

VI – 4. GENERAL DISCUSSION

The data from the literature and our own experimental results indicated that the facial nerve is especially vulnerable in its vertical portion of the facial canal. This conclusion is based on anatomical, neurophysiological and clinical observations:

1. Anatomy. The internal structure of the facial nerve is monofascicular inside the facial canal from the internal auditory meatus to the stapedial muscle or the chorda tympani, whereas it is multifascicular in other parts (SUNDERLAND and COSSAR, 1953). It is uncommon for a human peripheral nerve to be composed of a single funiculus. Other outstanding examples are: the ulnar nerve, the lateral popliteal nerve, and the axillary nerve in some part of their course (SUNDERLAND, 1968). The arteriovenous anastomoses inside the nerve (BOSATRA, 1956) and the location of the arterioles (JAMES, 1961) may also be important factors in the pathology of the nerve. Externally, the nerve is surrounded by a bony canal which may also influence the vascular supply to the nerve (ANSON et al., 1970). It has been suggested that the strong nerve sheath is probably the more limiting factor than the bony canal (JAMES, 1961).

2. Neurophysiology. Nerve fibers in monofascicular nerves are more susceptible to compression than fibers in multifascicular ones. Small nerve bundles can more easily escape compression. They can withstand higher pressure, because of their smaller diameter, and the effects can be dispersed by their epineurial packing (SUNDERLAND, 1968). More rapidly conducting nerve fibers proximal to the central nervous system are more sensitive to ischemia than more slowly conducting distal fibers. This is explained by the neuronal environment of closely packed, adjacent nerve fibers. Within their enclosing nerve sheath hyperkalemic depolarization of the nerve fibers may be additive (FOX and KENMORE, 1967).

3. Clinical considerations. Localized swelling in the vertical portion has often been described in ischemic paralysis, although the localization varied. Swelling is a common feature of a nerve proximally of a constriction (WEISS and DAVIS, 1943).

Recurrent paralysis after opening of the vertical portion of the nerve (decompression) has been described. But reexploration of three nerves because of recurrent paralysis after a first operation revealed that the facial canal and the nerve were originally not opened (JONGKEES, 1970). A retrospective study of about 300 decompression operations of the nerve revealed not a single case of recurrent paralysis of the operated nerve. In the

same series of 440 cases of ischemic paralysis, 46 recurrent paralyses were seen among the nonoperated cases (DEVRIESE and PELZ, 1969).

The *surgical treatment* of ischemic paralysis, the operative decompression, is much debated. This is due to the fact that the etiology of the paralysis is not known and that recovery is complete in about 70 per cent of the cases without treatment (PARK and WATKINS, 1949). If sudden ischemic paralysis in man is caused indeed by occlusion of the arterial circulation and by edema, then the situation begins to resemble the pattern observed during experimental infusion compression. In the latter case after about three and a half hours a critical duration of nerve block was reached in our experiments with cats and recovery was delayed (second degree of ischemic lesion). Nerve degeneration, however, might also have followed (third degree of lesion), with or without complete return of function. Almost any treatment is impractible before this critical period. Should surgical decompression of the facial nerve (internal neurolysis) be performed by longitudinal incision of the nerve sheath, in order to relieve compression? The precarious situation of the more vulnerable vertical portion of the facial nerve and disfigurement after defective repair could justify operative treatment. On the other hand, we have no diagnostic means, up to now, to detect in the acute phase which case will subsequently recover with defect, unless the nerve degeneration already started. Consequently, decompression in that stage will not be followed by a complete return of nerve function. This supports the statement of BUNNEL (1952) that the only possible time for prophylaxis or prevention is during the first three hours if the blockade of circulation is complete, and a few hours more if it is partial. Moreover, although decompression of a normal facial nerve is possible without damage to its function (Chapter III), the procedure might be hazardous when edema is present between the nerve fibers and the circulation to the nerve is disturbed (Chapter IV). It can be argued equally well, though, that the perineurium is to be slit to relieve the edema and to restore the circulation. In that case, however, extreme care should be taken not to damage the blood vessels of the nerve.

Finally, it should be noted that the effects of infusion compression on nerve function were studied in the cat and that they could not be observed for more than about 400 minutes. Chronic preparations only can inform about the ultimate repair of the nerve.

SUMMARY

In part one (Chapter I) a short survey is given about the nerve action potential. The literature related to the facial nerve in the cat and to ischemia and compression of peripheral nerve trunks is reviewed. The problems in connection with the experimental setup and ischemic paralysis of the facial nerve are defined.

In part two experiments on the facial nerve in the cat are described and discussed.

In Chapter II the experimental setup to record, to process, and to display monophasic compound action potentials from the facial nerve is described. It permitted stable recordings for about ten hours. The results obtained in control experiments are given; the normal nerve response is discussed.

In Chapter III the literature on decompression of the intratemporal part of the facial nerve in man and on decompressions in animals is first reviewed. Experimental decompressions under observation of submaximal and maximal action potentials are then described. The amplitude of the nerve action potential proved to be the most sensitive parameter of nerve function. Decompression was successful in 5 out of 12 cats. Drilling away of bone proved to be more dangerous than slitting the nerve sheath. The necessary precautions to prevent nerve damage are described. The conclusion is reached that the mastoid portion of the facial nerve in the cat can be uncovered without impairing nerve function.

In Chapter IV the influence of ischemia by circulatory arrest on the nerve action potential is studied. Nerves previously subjected to reversible infusion compression were more vulnerable to ischemia than nerves in control experiments. This is attributed to the presence of fluid between the nerve fibers. The possible consequences for surgery on the facial nerve are discussed.

In Chapter V the literature on damage to the intratemporal part of the facial nerve is first classified and reviewed. A modification of the infusion compression method originally described by JAIN and SHARMA (1964) to compress the facial nerve inside its perineurium is proposed. Suprasystolic infusion compression of increasing duration is reported in a series of experiments. About three and a half hours was critical, since release of compression thereafter was not followed by short term recovery as seen after shorter periods of compression. Histological examinations after one and after four hours of compression demonstrated disruptions in the nerve, fluid between the nerve fibers and vascular stasis. Repeated suprasystolic infusion compression of increasing short durations, in the same nerve, resulted in a progressive change in the shape of the nerve action potential. Infrasystolic compression above the diastolic blood pressure had little

and infradiastolic compression had no influence on the nerve function. Ischemia of the nerve affected the nerve action potential faster and in a different way than compression. It is concluded that a nerve can be paralyzed by injecting fluid into it, that the arterial circulation plays a determining role in this paralysis, and that three and a half hours of suprasystolic compression is critical for short term recovery.

Part three (Chapter VI) deals with ischemic paralysis (Bell's palsy, idiopathic facial paralysis) in man. The anatomy of the facial nerve and the pathology of ischemic paralysis are reviewed. From these data and from own experimental results the conclusion is reached that the facial nerve is especially vulnerable in the vertical portion of the facial canal. Surgical treatment of ischemic paralysis in the acute phase by decompression of the nerve is finally discussed. It is argued that the only possible time for prevention of denervation is during the first three hours when the blockade of circulation is complete. If a decompression of the nerve is performed, extreme care should be taken for the circulation of the nerve, since the presence of edema between the nerve fibers makes them more vulnerable to ischemia.

The Figures II–3, II–5, II–6, III–2 and III–3 are published with the kind permission of the Archives of Otolouryngology.

SAMENVATTING

In het eerste deel (Hoofdstuk I) wordt de zenuwactiepotentiaal in het kort besproken en wordt een overzicht gegeven van de literatuur over de nervus facialis bij de kat en betreffende ischemie en compressie van perifere zenuwen. Tot slot van de inleiding worden de probleemstellingen bij dit onderzoek samengevat.

In deel twee worden de experimenten op de nervus facialis bij de kat behandeld.

In Hoofdstuk II wordt de proefopstelling voor het registreren en het verwerken van monofasische actiepotentialen beschreven. De opstelling maakte het mogelijk gedurende ongeveer tien uur te registreren. De resultaten verkregen uit de controle-experimenten en de normale actiepotentiaal in de opstelling worden geanalyseerd.

In Hoofdstuk III wordt eerst de literatuur over decompressie van het intratemporale deel van de nervus facialis bij de mens en over decompressies bij proefdieren gerefereerd. Dan volgt een beschrijving van experimentele decompressies onder controle van submaximale en maximale actiepotentialen. De amplitude van de actiepotentiaal was hierbij de meest gevoelige parameter. Bij vijf van de twaalf proefdieren kon de nervus facialis gedecomprimeerd worden zonder aantasting van de zenuwfunctie. Wegboren van het bot rondom de zenuw bleek gevaarlijker dan splijten van de zenuwkapsel. De nodige voorzorgen bij het verrichten van deze operatie worden beschreven. Tot slot wordt geconcludeerd dat het mastoid gedeelte van de nervus facialis bij de kat kan gedecomprimeerd worden zonder nadelige gevolgen voor de zenuwfunctie.

In Hoofdstuk IV wordt de invloed op de actiepotentiaal nagegaan van ischemie door circulatie stilstand. Zenuwen die eerst aan reversibele infusie-compressie waren blootgesteld waren kwetsbaarder voor ischemie dan zenuwen in controle-experimenten. Dit wordt toegeschreven aan de aanwezigheid van vloeistof tussen de zenuwvezels. Er wordt gewezen op de mogelijke consequenties van deze waarneming bij operaties op de nervus facialis bij de mens.

In Hoofdstuk V wordt eerst de literatuur over experimenten op het intratemporale deel van de nervus facialis samengevat. Vervolgens wordt een methode beschreven waarmee de druk in de nervus facialis verhoogd kan worden. Deze methode is een modificatie van de methode van Jain en Sharma. Drie en een half uur suprasystolische drukverhoging was een kritische periode van drukverhoging wat betreft vlug herstel van de zenuwfunctie na opheffen van de druk. Bij histologisch onderzoek, na één uur en na vier uren drukverhoging werden scheuren in de zenuw, vocht tussen de zenuwvezels en stase in de bloedvaten gezien. Herhaald verhogen van de druk gedurende steeds langere perioden in dezelfde zenuw resulteerde in

een verandering in de vorm van de actiepotentiaal. Infrasystolische drukverhoging boven de diastolische bloeddruk had weinig invloed en infradiastolische drukverhoging had geen invloed op de zenuwfunctie. In vergelijking met druk werd de actiepotentiaal sneller en op andere wijze kleiner onder invloed van ischemie. Uit deze experimenten wordt geconcludeerd dat een zenuw verlamd kan worden door er vloeistof in te spuiten, dat de arteriële circulatie de determinerende factor bij deze verlammingen is en dat drie en een half uur suprasystolische drukverhoging een kritische periode is voor vlug functieherstel na opheffen van de druk.

In deel drie (Hoofdstuk VI) wordt de ischemische verlamming bij de mens (paralyse van Bell, idiopathische aangezichtsverlamming) besproken. Eerst wordt een literatuuroverzicht gegeven over de nervus facialis bij de mens en de pathologie bij de ischemische verlamming. Uit deze gegevens en uit de resultaten van de experimenten wordt afgeleid dat het verticale gedeelte van de nervus facialis in het facialiskanaal bijzonder kwetsbaar is. Tot slot volgt een discussie over decompressie als behandelingsmethode in de acute fase van de ischemische verlamming. Er wordt gesteld, dat bij *volledige* circulatiestilstand in de zenuw, denervatie alleen tijdens de eerste drie uren van de verlamming kan worden voorkomen. Wanneer een decompressie van de zenuw verricht wordt moet de circulatie in de zenuw zoveel mogelijk intact gelaten worden omdat de aanwezigheid van vloeistof tussen de vezels deze gevoeliger maakt voor ischemie.

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APPENDIX

OFF-LINE PROCESSING OF THE NERVE ACTION POTENTIALS

BY

H. R. DE JONGH AND P. P. DEVRIESE

An *analog-to-digital conversion* of the action potentials recorded during the experiments, was necessary for further processing. The conversion was performed by an analog-to-digital converter (AFO9A, Data Acquisition System) coupled to a general purpose computer (PDP-9) from Digital Equipment Corporation (Maynard, Massachusetts).

All computer programs were written in FORTRAN IV, with some special subroutines in MACRO-9 ASSEMBLER. In preliminary versions the conversational language FOCAL was used.

The separate conversion of each action potential into 128 discrete data values was initialized by a trigger pulse (recorded together with the original action potentials during the experiments) fed into the computer via an event counter (own design). In order to make precise latency calculations possible, a high sampling rate of 40 kHz was applied to the first 500 microseconds following the trigger pulse so that the onset of the stimulus artifact, preceeding the nerve action potential, could be measured from these 20 samples with an accuracy of at least 25 microseconds. Then, for a period of 5.4 miliseconds the sampling was continued at a rate of 20 kHz. This gives a further 108 digital values of the action potential (Fig. A–1).

The first stage of digital processing consisted of a *data reduction procedure*. Five-hundred consecutive action potentials (or calibrations) were averaged and stored on file-oriented magnetic tape (DECTAPE). For identification of the averaged curves one identification block per experiment containing additional information was added to the data blocks.

For *calculation of the parameters* of the action potentials, the most important step was to define for every curve the index b of the data point that represented the beginning of the action potential. This often proved difficult because of noise and irregular oscillations inherent to the stimulus artifact. The index b was found by comparing the values preceeding the data point corresponding to the maximal amplitude of the action potential until no significant difference (less than five microvolts) between 2 consecutive values was encountered. Consequently, the following parameters were calculated:

1. The latency time (in microseconds) from the onset of the stimulus artifact to the onset of the action potential.
2. The latency time (in microseconds) from the onset of the stimulus artifact to the maximum of the derivative of the action potential.

3. The latency time (in microseconds) from the onset of the stimulus artifact to the center of gravity of the action potential. The center of gravity was defined by the formula:

$$\frac{\sum_{i=b+1}^{N} i\Big(x(i)-x(b)\Big)}{\sum_{i=b+1}^{N}\Big(x(i)-x(b)\Big)} \qquad (1)$$

Here $x\ (i)$ is the value of the signal at the time instant with the index i Mostly N, the maximum number of data points, was 128; in case of a positive (downward) deflection, N was the highest index value such that $x(N)>x(b)$. For calculation of the first three parameters the nonuniform sampling rate was taken into account.

4. The maximum amplitude of the action potential (in microvolt).

5. The "surface" of the action potential. To give a measure of the total neural activity the appropriate data values were squared and summed. The square root of this value was named the effective "surface" of the action potential.* The following formula seemed the most appropriate for its calculation; it also includes the positive deflections that sometimes followed large action potentials:

$$\sqrt{\sum_{i=b+1}^{128}\Big\{x(i)-x(b)\Big\}^2} \qquad (2)$$

The main reason for calculating the "surface" in this way was computational simplicity. As long as the wave shapes resemble one another this seems admissible.

6. The maximum amplitude of the derivative of the action potential (in microvolts per millisecond). The calculated parameters were visually checked by individual display of the curves (Fig. A-1). The disturbing oscillations of the stimulus artifact were circumvented by giving it a standard shape of the exact duration of the stimulus used in the experiment. Of every experiment a paper tape, containing the calculated parameters of every curve, together with the original time of recording, was produced.

From the paper tapes the course of the parameters as a function of elapsed time was displayed, taking into account the calibrations (Fig. A-2). Finally, a *three-dimensional display* of all curves was realized in an attempt to obtain a better understanding of the nervous activity under experimental conditions.

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* On point of fact the word surface is used here only to indicate an approximate correspondence to the real surface of the wave form.

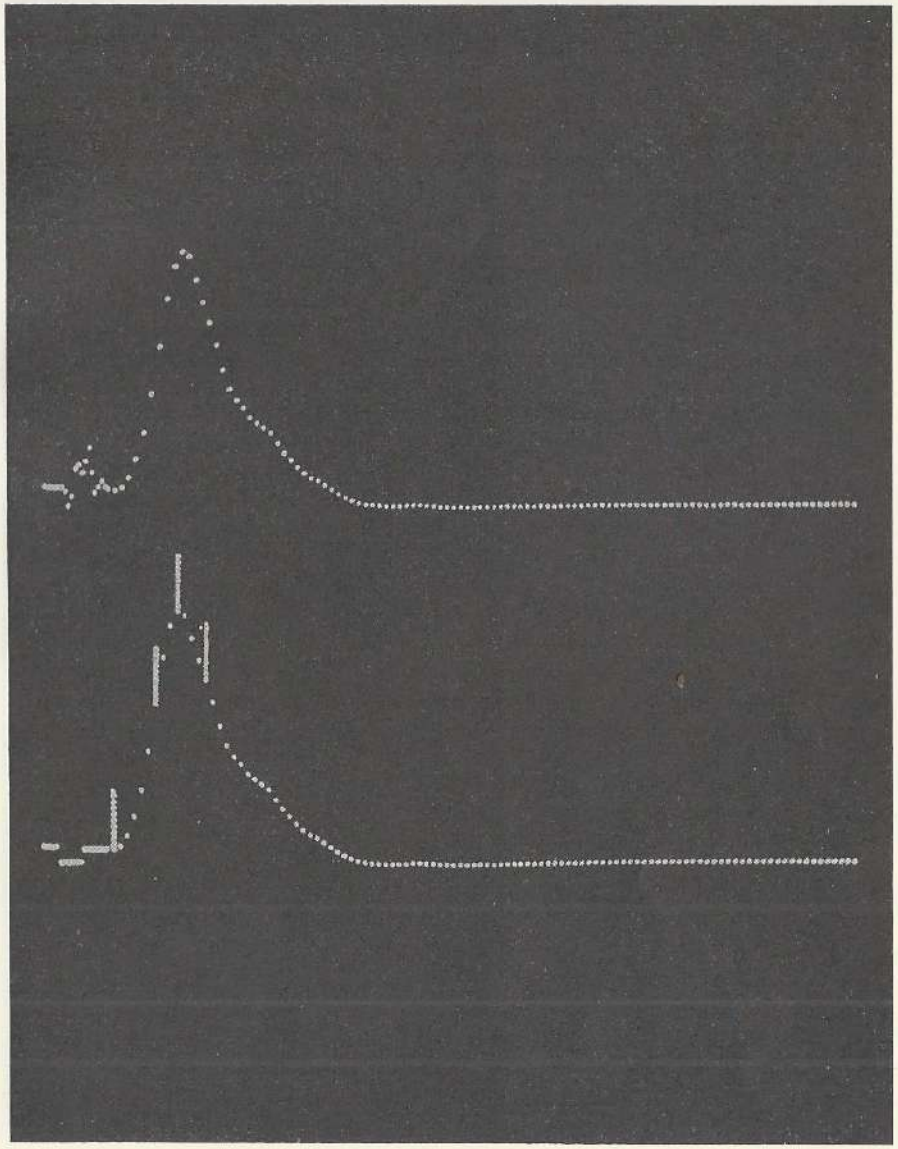

Fig. A–1. Display of action potential, some calculated parameters and the modified stimulus artifact.

Upper curve: Display of action potential (averaged over 500 stimulus presentations). The first 500 microseconds (20 data) were sampled at a rate of 40 kHz; the following 5.4 milliseconds at a rate of 20 kHz.

Lower curve: The same display with the positions of the vertical bars indicating from left to right the data points corresponding with: the onset of the action potential, the maximum of the time derivative of the action potential, the maximum amplitude, and the center of gravity of the action potential. The modified stimulus artifact is also demonstrated. The rectangle indicates the actual duration of the stimulus as used in the experiments (on a uniform time scale).

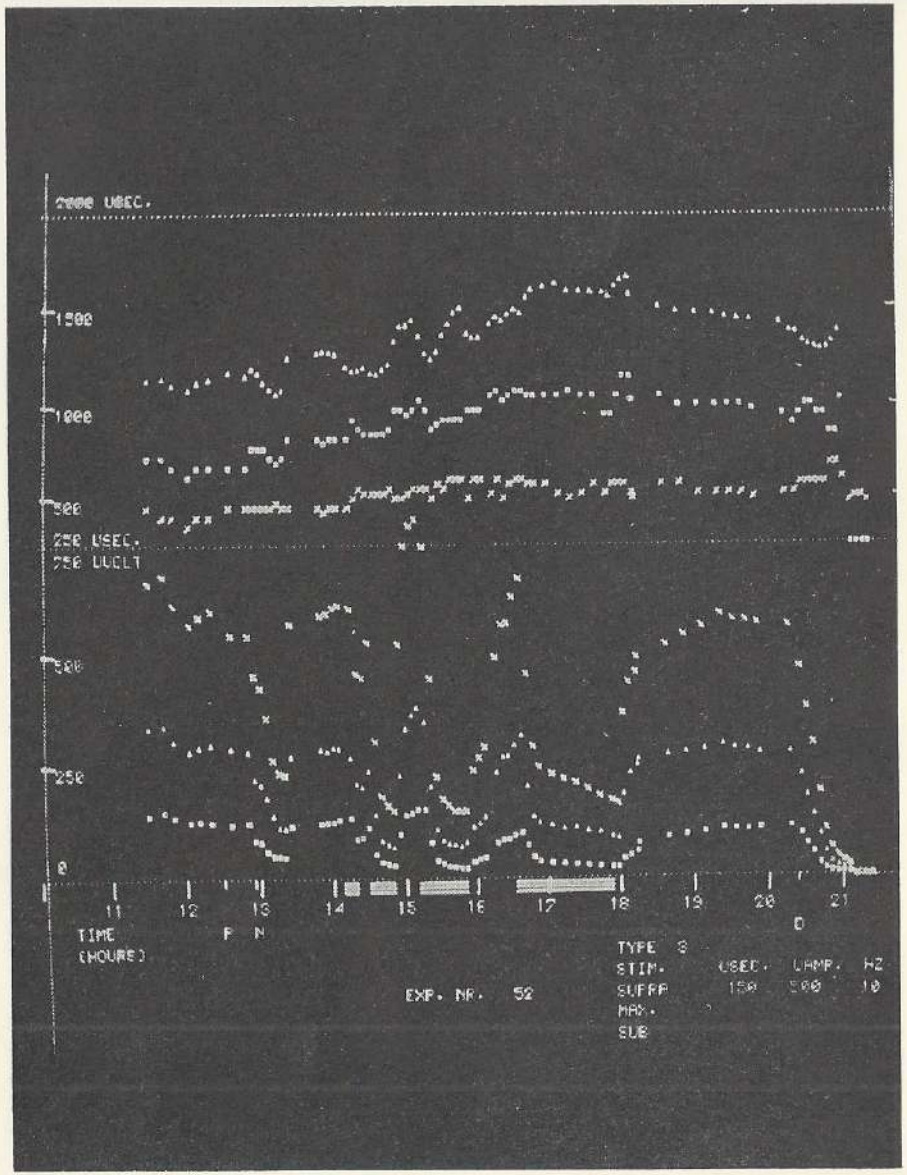

Fig. A–2. Display of the parameters (experiment 52).

Upper three curves: latency of the center of gravity (△), of the maximum of the derivative of the action potential (□) and of the onset of the action potential (×). The time scale is in microseconds.

Lower three curves: maximum amplitude of the action potential (×), "surface" of the action potential (△) and maximum amplitude of the derivative of the action potential (□). The scale is in microvolts. As indicated in the text the time instants are replaced by the indexes of the signal samples. This allows plotting of the "surface" and of the derivatived curve in the same Figure.

On the horizontal axis: time (in hours), P indicates the removal of paraffin, N the insertion of the needle. Furthermore, the various periods of pressure (suprasystolic) are shown as horizontal bars. D marks the point the animal was sacrificed.

STELLINGEN

I

Bij de mens is de nervus facialis in de pars verticalis van de canalis facialis kwetsbaarder dan in de andere intratemporale gedeelten.

II

Het recidiveren van een ischemische facialisverlamming kan door een goed uitgevoerde decompressie-operatie voorkomen worden.

III

Het trommelvlies kan door een onderzoeker met normale ogen dan slechts voldoende beoordeeld worden wanneer een vergroting van minstens twee maal wordt gebruikt.

IV

Kinderen met een gehoorverlies dienen zoveel mogelijk op een normale school geplaatst te worden; ze moeten dan wel begeleid worden.

V

Het getuigt van een gebrek aan verantwoordelijkheidsgevoel wanneer aan abortus provocatus de voorkeur gegeven wordt boven de overal verkrijgbare anticonceptiva.

VI

De succesvolle afloop van een operatie kan en mag aan een patiënt nooit beloofd worden.

VII

Er zijn aanwijzingen dat stoffen uit indische hennep dystrofie van hersenschorscellen kunnen veroorzaken.

Lit. Valk, L.E.M., Ned. T. Geneesk. 116:815–818, 1972.

VIII

Het verschil tussen kwantitatief en kwalitatief onderzoek zit niet in de kwantiteit van de verwerkte gegevens maar in de kwaliteit van de verwerking.

IX

De controle op de naleving van de onderhoudsplicht door de eigenaren van met overheidsgelden gerestaureerde monumenten dient doelmatig georganiseerd te worden; dit zou het mogelijk maken met de beschikbare gelden meer te restaureren.

X

Dat grote hoeveelheden reuzel gebruikt worden bij de produktie van fabrieksbrood is, uit het oogpunt van volksgezondheid, ontoelaatbaar.

XI

Gezien de veel grotere gastvrijheid die door de Roomsen geboden wordt, dienen hun fraaie kerkgebouwen door de Protestanten onmiddellijk teruggegeven te worden.

XII

Ter vermijding van ergernis verdient het aanbeveling bij televisiereclame geen gebruik te maken van geluid.