maximum independence and in a London Unit those not on home dialysis training programmes, had leg shunts and were encouraged to put themselves on the machine after having assembled their own machines. The following morning they stripped and cleaned their machines.

Muscle Power

Decrease in muscle power and orthopaedic type complications were seen in such multitude that I began to wonder whether transplantation was in any way justifiable, especially after seeing such fit and healthy patients in one London Dialysis Unit.

Surely the answer to transplantation cannot merely be good and/or adequate renal function but an active normal human being. If muscule power, especially around the weight-bearing joints can be hypertrophied, can one not hope to prolong the use of that joint? The intense and vigorous rehabilitation while the patient is awaiting transplantation should aim at achieving as "normal" a person as possible, making the actual transplant a much less formidable procedure.

This was, to some extent, confirmed in a London Unit where similar vigorous and comprehensive rehabilitation programmes existed and where these devastating results did not present themselves.

The fact that I have harped on the American hospitals more than elsewhere is partly due to the fact that I spent 46 days in Canada and the U.S.A., four in France, 20 in the U.K., and three in Johannesburg. Furthermore, the standard of physiotherapy — I repeat prophylactic respiratory physiotherapy — in the U.S.A. is something one has to see to believe. It was for this reason that I ended my travels in the U.K. in the hopes of learning. I must stress that I did not visit rehabilitation units, which would have had much valuable material to offer as this is their forte, but I was sent to visit Transplant, Cardio thoracic and Intensive Care Units only. The U.K. in itself was somewhat disappointing. I went eager to learn a great deal and to come back with lots of new ideas. Here the standard of physiotherapy was high, but there was little or no progress since my post-graduate scholarship visit of three months in 1965. At some places more cardiac surgery was being done, but the physiotherapy was routine and unchanged. In fact I felt we could teach quite a lot.

Research in transplantation from the physiotherapeutic aspect is vast. The long term effects of exercise on muscle power, bone and joint involvement and high doses of steroids is an unexplored one and theories alone are not acceptable. It is sad that our programme at Groote Schuur is so small and so slow moving as to get conclusive results one requires greater numbers and over a lengthier time period than our present results range.

SUMMARY

The observations, findings and recommendations relating to I.C.U.'s, Cardiac Units and Transplant Units in the U.S.A. and U.K. have been discussed with special reference to the prophylactic aspect of physiotherapy.

An attempt has been made to demonstrate the great need for this type of physiotherapy in the U.S.A.

In conclusion my sincere thanks are due to John Ackermann, the necessary driving force behind the whole trip, to him, Professor L. Eales, Dr. G. Thatcher, Dr. J. Terblanche, Dr. Stuart Sanders and Dr. T. O'Donovan for the introductions to the various heads of units I visited, to the Cape Provincial Administration for the realisation of this study trip and to Dr. J. G. Burger, Medical Superintendent, Groote Schuur Hospital for permission to publish.

The Use of Muscle Stimulation for Peripheral Nerve Lesions

by

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The use of electrical stimulation has been the subject of much controversy over the years and this paper represents a review of the available literature to date. However, before discussing the value of this treatment, a revision of the structure of a peripheral nerve trunk and the changes associated with a lesion may be of benefit to those concerned with the problem.

THE PERIPHERAL NERVE TRUNK

The structures forming the nerve trunk are:

1. Nerve fibres; 2. Connective tissue;
3. Nervi nervorum; 4. Blood vessels and lymphatics.

1. Nerve Fibres

The neurone is the basic structural and functional unit of the nervous system and its structural division into cell body, dendrites and axon is well known. The axons forming the trunk are myelinated with the myelin forming segments. Around these segments the cytoplasm of the Schwann cells form a sheath, with one nucleus to a segment, and the whole is enclosed in a connective tissue sheath, the *endoneurium.* Where the cytoplasm of the Schwann cell dips in towards the axon at the end of each myelin segment the notch so formed is called the Node of Ranvier.

Functionally the fibres within the nerve trunk are representative of both motor and sensory systems, including the Autonomic system.

2. Connective Tissue

The part played by the connective tissue components of nerve trunks in relation both to injury and recovery of function is often not fully appreciated.

Endoneurium: Composed mainly of collagen fibres this sheath surrounding the axon both protects the fibre and is responsible for maintaining the intracellular pressure of the axon.

Perineurium: Bundles of fibres are enclosed by this connective tissue wrapping, the whole being named a *funiculus.* The perineurium contains both elastic and collagen fibres which are arranged in oblique, circular and longitudinal layers around the bundles of fibres. These funiculi engage in repeated divisions and re-uniting along the length of the nerve so that the cross-sectional pattern of the nerve trunk varies as does the amount of perineurium present at any one place. Where there are many small funiculi and therefore more perineurium, the nerve trunk is strongest. This arrangement tends to occour where nerves cross joints and are subject to stretch. It is also of interest to note that the lateral popliteal nerve contains very much fewer funiculi than the medial popliteal nerve in the region of the knee joint. This factor may well account for the greater proportion of lateral popliteal nerve lesions following traction injuries in this area.

Where no perineurium exists, as at the nerve roots, the fibres are more vulnerable to traction injury; this often occurs with brachial plexus lesions.

The perineurium acts in protecting the nerve fibre and maintaining the intrafunicular pressure in much the same way as the endoneurium. In addition, because of its strength, it is largely responsible for maintaining the integrity of the nerve trunk as a whole under stretch. If sufficient of the funiculi are stretched beyond the elastic limit of the perineurium the whole nerve tends to elongate rapidly.

Epineurium: The funiculi are embedded in an amount of loose areolar tissue with an enclosing sheath at the periphery. This sheath, the epineurium, separates the nerve trunk from the surrounding structures and allows a degree of mobility of the nerve along its course. Branches given off along the length of the nerve tend to anchor it as do blood vessels concerned with supplying its structures.

Although capable of stretching and therefore protective to the funiculi, it is not as strong as the perineurium and serves more to cushion the bundles of fibres within it and protect them from compression. A nerve is therefore less liable to damage from compression where there are a number of small funiculi embedded in a large amount of epineurium.

3. Nervi Nervorum

Small branches from the nerve supply the connective tissue and form perivascular plexuses. They contain sympathetic and sensory fibres.

4. Blood Vessels and Lymphatics

These provide nutrition for the connective tissues and the axon along the length of the nerve. The vessels arise from arteries in the vicinity and are concerned with supplying both the connective tissues and the axon, although its survival and ability to function fully depends on the integrity of the cell body.

Damage to a Peripheral Nerve Trunk

This may occur in a number of ways.

Stretch: The strength of the perineurium is the prime factor in protecting a nerve trunk against damage due to stretch. Once the elastic limit of this structure has been reached it does not immediately break but elongates rapidly, rather like chewing gum. However, the endoneurium, being not as strong, gives before the perineurium and the axon is disrupted first. Continued stretch will progressively rupture the various connective tissue layers until finally the entire trunk is torn apart.

A compression factor also exists with stretching a nerve as the diameter decreases. Another problem is that lesions may be spread along the length of the nerve and not necessarily be confined to one area as the fibres will give where they are weakest.

Compression: Mechanical pressure will interfere with function by both disrupting the molecular organisation of the axon as well as causing ischaemia by pressure on the blood supply.

Friction: Here stretch or compression factors may operate where a nerve is stretched over callus or some other obstruction, or protective fibrosis may cause compression.

Ischaemia: Other than local compression mentioned above, interference with the main vascular supply to the area may cause nerve damage. Volkmann's Ischaemia is usually due to damage to the median nerve in this way.

Classification of Nerve Damage

The three well-known headings usually used to classify nerve damage, i.e. neurapraxia, axonotmesis and neurotmesis, fail to account adequately for the different degrees of connective tissue damage which occurs and which affect the prognosis. Sunderland[1] has tabulated five degrees of damage which more fully cover the field.

1st Degree (*Neurapraxia*) This is the well-known conduction block with disorganisation of the molecular structure of the axon only. The integrity of the axoplasm remains intact and this lesion is fully reversible. The length of time taken for recovery depends on the severity of the deforming force and may take anything from a few minutes to two to three months; recovery of muscle function does not necessarily follow a distributional pattern.

2nd Degree (*Axonotmesis*): Here again the pattern is familiar, with disintegration of the axon at the site of the lesion, distal to the lesion and for a short distance proximally; the cell body also undergoes changes in structure. Wallerian degeneration occurs, with the nerve retaining its ability to conduct for 48-72 hours after which this function diminishes until after anything from 14-28 days the axoplasm has been completely removed. The endoneurium shrinks as the intracellular pressure is not present but the tissue remains intact.

Recovery depends on the cell body remaining intact and the length of time is variable. The structure of the cell body must first return to normal; the axoplasm must grow down the endoneurial tube, grow across the site of the lesion and distally to the end-plate. As the return to full function depends on the return of the nerve fibre to its former diameter recovery must not be so delayed as to cause the endoneurial tube to thicken with fibrous tissue as well as shrink; the former irreversibly reduces the lumen and impairs function of the fibre. With the endoneurium intact in 2nd degree injuries recovery is usually uncomplicated and once the former end-organ relationship has been established function is possible. The pattern of functional recovery will be distributional, i.e. it will occur in the order of nerve supply from proximal to distal.

3rd Degree (*Neurotmesis*): Damage has been sufficient to not only disrupt the axon but the endoneurium as well. The perineurium is intact but the fibres within the funiculi are disorganised. Recovery is delayed and may be incomplete. A new factor is involved in the crossing of the site of the lesion by the regenerating axon. The axoplasm may not necessarily grow down its former endoneurial tube or for that matter any endoneurial tube, and normal end-organ relationships may not be restored. If the time factor is long enough the endoneurial tube may no longer be capable of expanding to its former diameter as explained previously; changes in the end-organs themselves due to prolonged denervation may be such as to preclude recovery and function will depend on sufficient number of fibres returning to normal.

4th Degree: This degree of injury damages the perineurium with subsequent disruption of the funiculi. Functional recovery is incomplete and delayed due to the amount of connective tissue damage and the difficulties involved when the axoplasm has to bridge the site of the lesion; not only are the endoneurial tubes disorganised but the guiding perineurium is not present to maintain the integrity of the funiculi.

5th Degree: Disruption of the epineurium and therefore loss of continuity of the entire nerve trunk is the feature of this stage of damage. Recovery depends on suturing the nerve ends and is never complete.

With these last two degrees of injury changes in the structures supplied by the nerve may be such that even with regeneration of the axon, functional recovery is not possible. Time for recovery to occur plays a large part in determining these changes and a brief discussion is necessary here before reviewing the possibilities of benefit by electrical stimulation to the denervated muscle.

Changes in Structures Deprived of their Nerve Supply

Loss of voluntary muscle power and cutaneous sensation are the most obvious presenting changes following denervation. It must be remembered that joints and soft tissues will have lost their sensory supply and be more susceptible to trauma; vaso-motor disturbances occur following loss of sympathetic control of blood vessels; abnormal sensation will also be experienced with recovering or progressing lesions; disturbances of conduction, and false localisation due to misrepresentation in the cortex after wrong regeneration of an axon to an end-organ.

Changes in Muscle

Loss of voluntary movement, which may be complete paralysis or partial paresis of the muscle or muscles supplied by the nerve follows a lesion to a peripheral nerve trunk.

The muscle first shows a reduction in size and weight and a change in colour. This atrophy proceeds rapidly for the first 60 days, 70 per cent of the original muscle bulk having diminished in this time. The rate of atrophy then slows down and reaches a more or less constant level at 120 days when there has been 80 per cent to 90 per cent reduction in size.[2, 3, 4, 5]

It has been shown experimentally[6, 7] that 18 months to three years after denervation, muscle fibre, although atrophied, had not lost its ability to contract. In addition the end-plates have been observed to remain intact for periods up to 17 months.[8]

Changes in the connective tissues also occur, this tissue following the same pattern in muscle as in nerve, with individual fibres enclosed in endomysium, bundles of fibres (fasiculi) in perimysium and then the whole ensheathed in epimysium. Following denervation the connective tissue proliferates, the perimysium first and then the endomysium. This proliferation appears from the second month onwards and progresses for varying periods, finally reaching a steady state where the muscle fibres are separated by the thickened connective tissue but retain their own characteristic histological appearance and fasicular pattern within the muscle. Ultimately the muscle cell itself fragments, thins or swells and disintegrates, the muscle unit being replaced by fat and fibrous tissue.

Many of these findings have been experimentally induced in animals, but studies on human muscle show a close correlation.

Bowden and Gutmann[9] showed vascular changes in human muscle as well with obliteration of arteries and capillaries after 12 months' denervation.

Time is once more an element which emerges in the consideration of nerve lesions. If the axon regenerates within the period, while the muscle fibre remains intact, return of function is possible, and this has been shown to sometimes last as long as three years. However, other factors operate to jeopardise the survival of the muscle fibre and cause its degeneration and replacement by fibrous tissue to occur more rapidly.

(*a*) Reduction in circulation, due to vaso-motor paralysis and lack of muscular activity impairs the nutrition to the part and accelerates degeneration of the fibres.[10] Lack of muscular activity may be due both to the paralysis of muscles and to immobilisation of the part following other injuries incurred at the same time. It may also be due to bad splinting of the part to support the paralysed muscles.

(*b*) Paralysed muscles are very susceptible to trauma and over-stretching which accelerates degeneration.[11]

(*c*) Non-neural complications at the time of injury may lead to impairment of circulation and the formation of interstitial fluid which will impair nutrition and hasten the formation of fibrous tissue.

The end picture in long standing denervation is one of widespread interstitial fibrosis involving muscles, joints, tendon and fascial planes with contractures of muscles. From the above experiments and clinical findings it seems reasonable to believe that the prognosis on functional recovery of human muscle for periods up to 12 months following denervation is good, and that it thereafter depends on conditions prevailing both at the time of injury and during the period of denervation.

THE ROLE OF ELECTROTHERAPY

To date no procedure is known which will influence axon regeneration and treatment must be directed towards maintaining the part in as good a condition as possible until reinnervation occurs and to restore full function and muscle power following recovery.

The questions one asks are:

Can electrical stimulation of denervated muscle prevent atrophy and fibrosis? and

Can it accelerate the period between reinnervation and restoration of full power?

Experiments have shown the answer to the second question o be in the negative[49, 25] but widely conflicting views are held on the first question.

Experimental Findings

While some animal experiments have shown that atrophy of muscle following denervation can be retarded, but not prevented by electrical stimulation,[12, 13, 14, 15, 16, 17, 2, 23, 24] others have shown that electrical stimulation is of no value in the prevention of denervation atrophy.[18, 19, 20, 21] Still other workers have found the value unproven or unsure.[5 22]

The differences which arise are due, apparently to:—

1. different methods of assessing degree of atrophy;
2. different animals used;
3. different strength of contraction;
4. different duration and frequency of treatment.

1. Methods of Assessing Atrophy

These have included weight of the muscle as compared to normal side, wet and dry weight after death, measurement of maximal tension, volumetric readings, histological studies, oxygen consumption comparisons, strength duration curves and measurement of double refraction. Measurement of weight has proved difficult due to the fact that connective tissue proliferation will increase weight and is liable to increase if there has been inflammation and trauma, and there is therefore no indication of how much weight maintenance is due to muscle bulk and how much due to connective tissue proliferation. Volumetric measurements are complicated by the increase in fluid in the muscle following contraction and must not be done immediately after exercise. The most reliable method is the histological study of muscle sections which show degree of muscle fibre, the size of the fibres and amount of connective tissue present.

2. Different Animals Used

Most experiments which showed a positive finding were carried out on the gastrocnemius muscle of the rat or rabbit. When experiments were tried on monkeys, cats and dogs they were found to be unsuccessful and the theory that there was a species difference was postulated. However, histological studies on rat gastrocnemius muscle showed that only the peripheral muscle fibres retained some bulk, but that the deeper fibres had atrophied.[15] The muscles of the other species are larger than those of the rat and perhaps this fact is responsible for the species difference, i.e. that stimulation can only affect superficial fibres and very small muscles.

3. Different Strength of Contraction

Here the more successful experimentalists anaesthetised the animal to produce strong contractions, often against resistance. Trophic changes on the skin caused difficulties and the electrodes had to be moved continually from place to place. In the experiments showing no beneficial results the animals were not anaesthetised and treatment was within the patient's tolerance. It would appear that success in retarding denervation atrophy depends on producing strong contractions preferably against a load.

4. Different Duration and Frequency of Treatment

Many different opinions were voiced on this matter. As far as the type of current used was concerned, whatever type produced the required contraction at the lowest intensity appeared to meet the requirements. Duration of treatment varied from several seconds a day to throughout an eight-hour period. Some experimentalists felt there was no duration difference, a short treatment being as effective as prolonged treatment, but in those experiments which produced positive findings 20-30 minutes treatment was given. Fatigue was found to have no detrimental effect on the muscle, and the rest periods were given to allow the muscle to recover before continuing stimulation.

Frequency of treatment worked on the more the better principle, twice daily better than daily and only twice a week being ineffective.

CLINICAL TRIALS

Clinical trials on man introduce many more problems for accurate assessment, suitability of patients and time available for treatment. Of the six trials reported, four showed negative results.[27, 28, 29, 30] The trials were carried out on patients with radial, ulnar and median nerve lesions and facial paralysis. Two trials on denervated hand muscles, where volumetric measurement was used to assess muscle atrophy showed retardation of atrophy about seven per cent better on the treated muscles than on the untreated muscles.[25, 26]

In all trials only patients who continued the treatment daily were assessed. In the trials showing retardation of atrophy[25, 26] it was stated that any break in continuity did not produce results and full daily treatment was essential. Duration of treatment was 90 contractions strong enough to move the hand with gravity eliminated. The difference in atrophy between treated and untreated muscles was only in the region of seven per cent and as no patients were stimulated before 60 days, the muscles had already atrophied 60 per cent. The prevention of seven per cent further atrophy as the result of daily treatment for periods up to 400 days does not really seem very significant. This view was held by the authors of a report on similar clinical trials with the same results[30] who felt that they did not justify the expenditure of time, money and personnel in the routine application of electrical stimulation to denervated muscle.

Trials on facial muscles[27, 29] treated daily with 90 contractions per day (never less than 30), for up to seven months, were completely negative, no shortening of the interval between initial movement and full recovery or time of onset of initial movement being shown as compared to untreated muscles.

In a comprehensive study of denervated muscles supplied by the radial nerve[28] patients' tolerance allowed moderate contractions of which only 30 were given daily. No benefit was shown from electrical stimulation which may have been due to the short duration of daily treatment. However, the authors then carried out trials to assess the amount of work done by a muscle during electrical stimulation and used the amount of increased blood flow as an index. A sleeve plethysmograph recorded volume increase and lesions where the distal part of the limb was totally denervated, such as sciatic nerve and brachial plexus lesions, were chosen. To produce contractions of sufficient intensity, the skin had to be anaesthetised in sciatic nerve lesions, the arm already having lost sensation in the brachial plexus lesions. Comparisons were made of the injured limb after stimulation using five times the skin tolerance intensity for four minutes at 30 and 60 contractions per minute and the uninjured limb after one hour's rest and then active movement against slight resistance for half a minute. The increase in blood flow after stimulation at far above normal tolerance of current intensity produced no increase in circulation whilst the active movement produced a marked increase. Electrical stimulation was carried out in the same way on normal muscle also with no effect and the authors felt that it is impossible to produce a useful degree of exercise by electrical stimulation.

CLINICAL AND EXPERIMENTAL EVALUATIONS

There have been numerous articles written on the subject after clinical experience in which opinions differ widely,[31, 32] some feeling that, if the lesion will take long to recover, treatment should be given[35] while others feel the exact opposite.[41] Earlier workers felt only the weakest contraction possible should be used[33] whilst still others that a large number of vigorous contractions[37] should be used. Finally there have been those who feel that the value of stimulation is unproven and balanced against the length of time for which the patient needs to attend and the use of the physiotherapy department it is not worth undertaking.[34, 36]

This latter view is held in the Medical Research Council Report on Peripheral Nerve Lesions.[38]

The difficulty of assessing which of the modalities one is employing during treatment has the most benefit, is obvious.

In the more recent articles, opinion has very markedly swung away from the need to use electrical stimulation in nerve lesions.[39, 40, 41, 42, 43, 44, 45, 46, 47, 48] If the treatment is mentioned at all, it is stated that it is not used or that its value is doubtful with the above conclusions.

The most comprehensive physiotherapy article read[44] on the treatment of nerve lesions puts emphasis on the control of oedema, maintenance of joint range, constant usage of the limb and intensive re-education once re-innervation has occurred of both motor and sensory function. Good functional results have been attained without resorting to electrotherapy at any stage.

Of interest is that the facial muscles do not benefit from treatment[27, 29] and physiotherapy is not advocated by leading workers treating facial paralysis.[40, 46, 47]

SUMMARY

1. The structure of a Peripheral Nerve trunk is discussed.
2. Causation of damage to a peripheral nerve is discussed and five degrees of nerve damage listed.
3. Changes in structure deprived of their nerve supply, particularly muscles, are explained.
4. It has been shown experimentally that electrical stimulation producing strong contractions of denervated muscle against a load, given daily for 20-30 minutes will not prevent muscle atrophy but will retard it in the smaller animals such as the rat and the rabbit.
5. Clinical trials have failed to prove any marked benefit from the treatment of denervated muscle by electrical stimulation.
6. Recent views on the subject show that while it may be of some value, it is not a necessary part of treatment and does not justify the time, money and effort needed to carry out the treatment.