Rehabilitation of the elderly in the 21st century

The F. E. Williams Lecture 1992

The future burden of disability in old age

The expectation that the greatest increases in the population over the next few decades will be in the very elderly [1] has created considerable anxiety because the incidence of major neurological and musculoskeletal causes of disability—stroke [2], Alzheimer's disease [3], Parkinson's disease [4], osteoarthritis [5], fractured neck of femur [6]—increases almost exponentially with age. Even epileptic seizures occur more commonly in old age and great old age [7]. An increasing incidence resulting from an ageing population may not translate into a proportional rise in prevalence, if survival in older patients is reduced. This has been suggested for stroke [8]; however, a higher case fatality rate is cold comfort indeed. Although it is difficult to predict how age-related trends for chronic diseases will translate into disability and dependency, the 1988 Office of Population Censuses and Surveys (OPCS) census of disability, which showed an exponential relationship between age and the prevalence of disability, indicates how they might [9] (Fig. 1).

It is easy to dwell morbidly on statistics and speak of rising tides and silent epidemics. At present, there is as little evidence of the pandemic of dementia and disability predicted by Gruenberg and Kramer as of the compression of morbidity at the end of the lifespan anticipated by Fries [10,11]. Trends in disability-free life expectancy as a proportion of life expectancy in old age [12] do not support the belief that increased survival is being bought at the cost of a dramatic increase in disability in old age. Nevertheless, one might reasonably assume that one aspect of the future of rehabilitation of the elderly is that there will be more of it. How are we going to meet the challenge? To address this question, we need to understand where we are now, and try to define what present-day rehabilitation is.

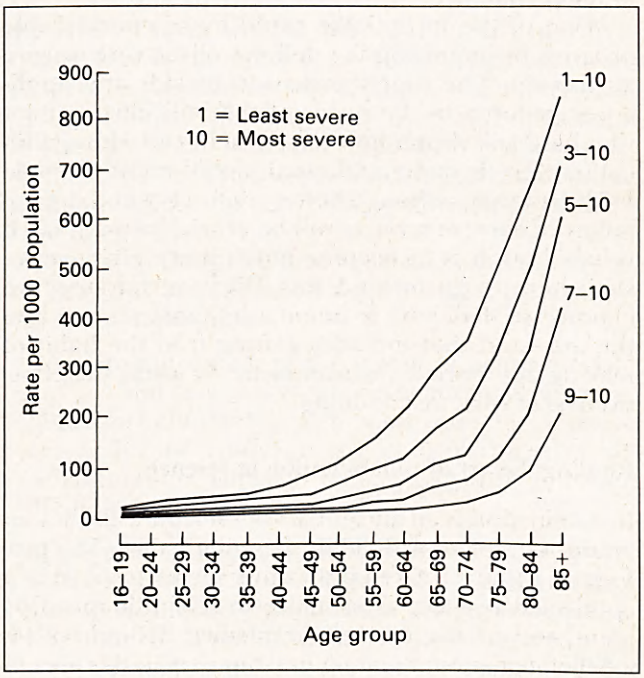

Fig. 1. *The prevalence of disability against age (OPCS data).*

Rehabilitation now

Rehabilitation eludes easy definition [13], perhaps because it is a dimension of medicine rather than a particular specialty or set of procedures. It predominates when pathology proves unresponsive, or only partially responsive, to attempts at correction. It may be thought of as the reduction of functional deficits without necessarily reversing the underlying biology of the disease.

Clarifying the distinction between impairment, disability, and handicap has sharpened our concept of rehabilitation [14]. We may think of impairment as a deficiency in the body; of disability as the consequent limitation in acitivities of daily living; and of handicap as the resultant social disadvantage and dependency. The handicap is the 'cash value' of the impairment or the disease.

It is essential to appreciate that the amount of handicap need not be proportional to the amount of impairment. An individual may have only a small

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impairment and yet be enormously handicapped; or, on the other hand, be severely impaired and yet be relatively free of handicap. Current rehabilitation is mainly about intervening between impairments and disabilities and between disabilities and handicaps. It explicitly recognises that the person suffering from a disease is not simply an organ or organism but an individual in a complex environment. Rehabilitation therefore takes place at several levels (Fig. 2) and the resultant package is complex, as illustrated for stroke in Table 1. The central concern is with the restoration of independence.

What of the future? We could make considerable progress by improving the delivery of the care we give at present. The timely provision of aids and appliances, tailored to the needs of the individual patient who has been shown how to use them and whose difficulties with them are addressed, would be an example [15]. However, although better resourcing and organisation of current services will be crucial to progress, if rehabilitation is to become much more effective we shall have to go beyond this. Whatever strategy we choose, we shall have to admit a bit more science into the art—and that includes getting into the habit of making appropriate measurements to assess the effectiveness of what we are doing.

Table 1. The rehabilitation package in stroke.

Direct treatment of damaged organ
e.g. Bobath therapy (?)
language therapy (?)
anti-spastic agents
Prevention of damage to other organs
e.g. skin
shoulder
Prevention of secondary psychological damage
Assessment of impact of stroke on physical, cognitive, emotional, and social function
Retraining skills in activities of daily living
Provision of aids and appliances
Modifying the material environment
Mobilising a social support network
Education of patient and carer(s)
Counselling and support of patient and carer(s)

Rooting the art of rehabilitation in science

Rehabilitation is an ancient art but scientifically it is an infant—an infant with adult spending habits. The prolonged infancy of rehabilitation seems to have two connected reasons: a resistance to scientific measurement, and a faith in untried remedies. Behind both is a belief, commoner among practitioners in this area of medicine than in any other, that compassion, support, kindness and enthusiasm and, indeed, whole patient care, are incompatible with science. This is of course nonsense: effective therapy is more humane than ineffective therapy, and therapy that has been subjected to test is more likely to be effective than therapy that has not been so tested; from which it follows that rigorous scientific standards are not merely compatible with humane rehabilitation but are its necessary condition.

Rehabilitationists rarely doubt that most of what is done in rehabilitation is effective, in the sense of helping the patient physically, mentally, socially, and spiritually to adapt to impairments. There is little in the rehabilitation package in stroke (Table 1) with which one could quarrel, and little one could ethically put to the test by denying it to some patients in a randomised controlled trial (except perhaps the biggest, and least visible, part of nearly every rehabilitation package: the

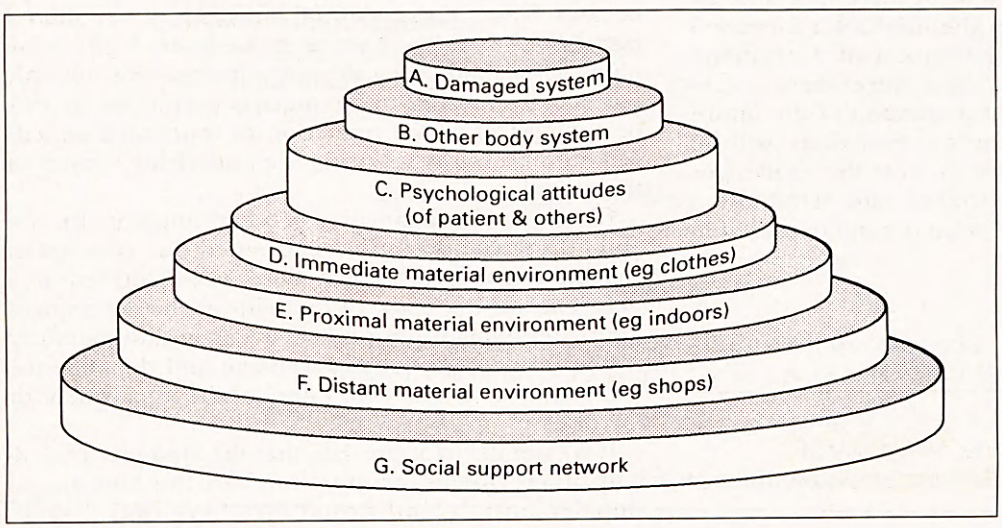


Fig. 2. *Rehabilitation is multi-levelled.*

hours of waiting while nothing is happening). However, two points need to be made.

First, strong claims are being advanced for the effectiveness of certain privileged resource-intensive elements of the current package—namely, the specific therapies. These claims have never been properly put to the test. What little literature there is, is unsatisfactory and does not, for example, support the belief that some forms of physiotherapy are better than others [16–21], or that they add much to the crucial work of the therapist in educating, advising, training, informing, and counselling patients and their families and providing aids and appliances when appropriate and teaching their use.

Second, we really should be doing better than we are doing now. Recent careful studies of the effectiveness of rehabilitation—the overall package [22], that heterogeneous collection of interventions called 'physiotherapy' [23] and highly specific intensive therapies for upper-limb function [24]—have shown that while rehabilitation is effective the benefits are only modest [22–24], that they are often short-lived [23], and that they make little or no impact on the isolation and psychosocial dysfunction of patients and their carers [22]. Many patients are bitterly disappointed with what we have to offer them and they subject themselves to prolonged courses of treatment—packaged with prolonged courses of hanging about—in the hope of getting much better than we can make them. It is about time we began to share the impatience of our patients and were less satisfied with our modest achievements.

Making rehabilitation more effective presupposes a decided prejudice in favour of treatments that have been demonstrated in quantitative studies to be effective. Increasingly, measurement should be used to weed out useless treatments from the useful ones. This is easier to advocate than to act upon, for there are difficulties unique to rehabilitation research. Some of them are intrinsic and some of them are man-made [13]. Nevertheless, they are not insuperable and should not be used as an excuse for not carrying out satisfactory research.

Future rehabilitation research may focus to a much greater extent than at present on specific, precisely defined treatments of particular impairments. Unfortunately, the current trend is mainly in the opposite direction, with one or two notable exceptions, such as the detailed single case studies of the use of training techniques for neuropsychological deficits [25]; the Bristol study of intensive therapy for arm dysfunction after stroke [24]; and the Oxford study of physiotherapy interventions late in stroke [23]. Otherwise, the emphasis tends to be on assessing the overall impact on the patient of global packages of treatment; the effect on overall activities of daily living (ADL) scores of, say, three half-days a week of outpatient rehabilitation as opposed to four whole days [26]. Such studies are immensely labour intensive, encounter enormous methodological problems, and do not, in the end, tell us what we really want to know: namely, exactly what part of what treatment does what to which patients [13].

I envisage a future in which discrete new elements, assessed one by one, will be introduced into the rehabilitation package and others will be discarded. In 10 or 20 years' time the overall package may have been totally changed, like a living body whose entire tissues are replaced while daily living continues. For each element it is essential to make the initial evaluation at the right level [13]. Where claims are being made for an effect on impairment, appropriate measures of impairment must first be made; then, if a positive result is found, the impact on disability and handicap will need to be assessed. In this way, real effects will have less chance of being overlooked and imaginary effects will not be 'observed'. The further one's measurement is from the level at which the treatment has its putative effect, the more will confounding variables cloud the picture.

Hostility to this piecemeal approach is considerable and it is not unconnected with hostility to directly addressing impairments:

> 'Impairment measures are useful in acute specialties . . . and they are superficially attractive in rehabilitation because they seem more scientific. However, relief of impairment is not the aim of rehabilitation, which is concerned with relieving disability and handicap.' [27].

It is as if by taking impairments seriously, one somehow fails to respect the wholeness of the patient. This is connected with the fundamental insight of rehabilitation: that the amount of impairment need not be reflected in what matters to the patient, namely handicap. It does not, however, follow from this that impairments are irrelevant. The belief that we should not look at the individual parts, since what matters is the whole patient, or quality of life (which cannot be measured), exemplifies 'the *fallacy of misplaced holism*'. Of course, quality of life and the whole patient *do* matter; they matter throughout all of medicine. They matter, for example, to diabetic patients and to diabetologists. But diabetologists have not thereby been inhibited from measuring, and trying to treat, metabolic impairments. A diabetologist whose concern for the whole patient discouraged the assessment of blood sugars would not, I submit, provide a better service than one who regularly measured blood sugars.

The directions of future development (Fig. 3)

The present package consists of half-hearted attempts at physical reintegration of the patient in the environment (aids, appliances, etc.); half-hearted attempts at psychosocial rehabilitation (a bit of support, a bit of counselling, sporadic provision of carers' groups, etc); and quarter-hearted attempts at reversing impairments. In the future I anticipate, we shall make

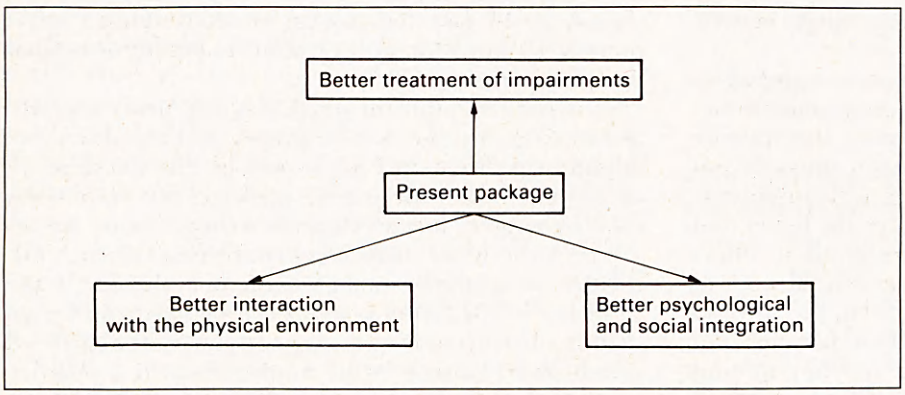


Fig. 3 *Directions of future development in neurological rehabilitation.*

progress in all three areas, as well as improving our delivery of those elements in the present package that are worth retaining.

Improved physical integration will depend upon more than better aids and orthoses. We may expect advances in orthotics not only because of the development of new materials that are light yet robust, flexible yet reliable, but also because of a willingness to tailor design to the needs of individual patients, paying attention to ease of use and cosmetic appearance. Already bioengineers are employing their skills to create more sophisticated devices that can respond to movement, and incorporate a better appreciation of the dynamics of posture and of that dynamically unfolding posture we call mobility. The new generation of orthoses is exemplified by the reciprocating gait orthosis (RGO) [28], which is clearly an advance on the simple devices that have gone before.

Orthoses, however, often seem to be the mark of disability rather than of liberation from it. They are obtrusive, add to the energy requirements of locomotion, and have to be donned and doffed, procedures that may require the help of others and so perpetuate the dependency they are designed to evade. The RGO is unlikely to have a major part to play in rehabilitating frail elderly people, many of whom have difficulties with support stockings. Nevertheless, it is possible to imagine a future in which orthoses merge with more effective and versatile environmental control systems [29], perhaps with the intervention of robotics. The development of a virtual reality to emulate or simulate the world to which disabled elderly people may be denied access raises ethical [30] and other problems; nevertheless, it should be considered seriously, as a 'table-served' environment neatly inverts the problem of giving disabled people access to a richer experience.

Improving the psycho-social integration of disabled elderly people will depend on several things: a deeper understanding of successful adjustment to disablement; more effective, universally available, and sustained counselling of the patient, the family members, and other supporters; less marginalisation of elderly disabled people, which will depend as much upon legislation and economic support—cash as well as more freely available customised and co-ordinated services—as upon encouragement of positive attitudes and education; and the technological transformation of the world. The world of the 21st century will be one in which labour, the exertion of power, and even ordinary social interactions, will be based on microelectronic technology and depend less upon strength and mobility.

Strategies for reducing impairments: drugs and neural implantation

What are the prospects for making progress in improving impairments, particularly in neuromuscular rehabilitation, which presents the greatest challenges to patients, their families, and their rehabilitationists? There seem to be several possible strategies for diminishing impairments: new drugs, neural implantation, and electrotherapy.

Pharmacological Strategies

New drugs may be designed to:

1. promote plastic changes;
2. replace deficient neurotransmitters;
3. normalise neurophysiology;
4. have direct effects on end organs.

Research into the control of axonal growth, guidance, and synapse formation by receptors on neuronal growth cones that respond to positive and inhibitory cues in the local microenvironment has brought nearer an understanding of why the regenerative capacity of the central nervous system is so poor compared with that of the peripheral nervous system [31]. This raises the possibility of using soluble growth factors to enhance natural recovery processes. However, exuberant growth *per se* may not necessarily be a good thing: it may produce a neural hairball rather than useful circuitry. Growth needs to be directed by information—as it is in development—if it is to produce functional recovery.

Neurotransmitter replacement seems a logical strategy where the deficit is a neuropharmacologically tidy one, as was once believed of Parkinson's disease and hoped of Alzheimer's dementia. However stroke, the most important neurological disease of old age, is neuropharmacologically illiterate.

Drugs may play a part in dealing with the effects of pathophysiology and normalising physiology. Increased understanding of, for example, the neurophysiology of spasticity may permit the development of antispastic agents to reverse established spasticity, or prevent its development, without global loss of tone or weakness.

The problem with centrally acting drugs is not only of physiological, but also of physical (spatial and temporal) targetting. The use of intrathecal baclofen [32] for severe spasticity avoids the blood brain barrier and ensures localised effects, through controlled concentrations of the drug in the target tissue. But there is a long way to go before individualised, modular, microminiaturised, closed-loop patient implant systems, able to deliver multiple drugs with precise location in space and controlled variation in time, are developed. Enriching the pharmacological conversation with the brain is one thing; emulating the pharmacological conversation of the brain with itself quite another.

Direct pharmacological effects on end organs have few exemplars, but there is one impressive instance. Beta-agonists not only exert trophic effects, preventing muscle wasting in ageing laboratory animals [33], but also have an acute effect in increasing strength [34]. Growth hormone increases muscle bulk and decreases lean body mass in elderly subjects, thus improving the engine-to-chassis ratio in two ways [35], and can prevent muscle wasting in association with operations [36].

Neural implantation

From the point of view of a geriatrician, the most interesting research developments suggest that implants might reverse laboratory models of age-related declines in sensorimotor co-ordination and memory and learning [37]. However, translating these fascinating experimental findings into clinical benefit has proved to be even more difficult than anticipated. Meanwhile geriatricians, aware that, so far, 'more patients have probably been harmed than helped' [38], follow the debate as to whether implants act as biological depot preparations, as replacement circuitry, or merely as a source of growth factors [39].

Electrotherapy (Table 2)

Electrical techniques to restore lost strength or neural control are based on the observation that an electrical stimulus, when applied to a nerve, can induce an action potential indistinguishable from the naturally occurring signal, and that this can cause effector systems to respond. Until the advent of cardiac pacemaking, electrotherapy was totally unscientific. This has changed dramatically, and attempts to develop rational electrotherapies have gone side by side with honest endeavours to evaluate their effects scientifically. In this brief survey of a huge topic, I shall merely allude to some representative endeavours that may find application in the future.

Table 2. Electrotherapy in neuromuscular rehabilitation.

EXCITATORY
Continuous
Cerebellum (epilepsy; cerebral palsy)
Thalamic vim nucleus (Parkinsonian tremor)
Spinal cord (pain; multiple sclerosis)
Functional
Contingent:
Thalamic (multiple sclerosis)
Sacral root (neuropathic bladder in SCI)*
Lateral popliteal nerve (foot drop)
Sensory cueing (tactile neglect)
Programmed:
Diaphragmatic (neurogenic hypoventilation)
Paraplegic walking devices (SCI)*
Open loop (incomplete lesion)
Closed loop (complete lesion)
TROPHIC
Spinal cord stimulation
Muscle stimulation
'Machine-blind' irrational
Empirical non-tailored uniform frequency (quadriceps)
Tailored uniform frequency (facial nerve)
Patterned neuromuscular stimulation
Intrinsic hand muscles
(Quadriceps)

* SCI = spinal cord injury

Excitatory electrotherapy

Excitatory stimulation may be divided into continuous and functional electrical stimulation (FES); FES divides into contingency stimulation, where stimulation is contingent upon some other event, and programmed sequential stimulation.

Continuous stimulation makes less physiological sense but is technically easier and presupposes less physiological knowledge. Cooper introduced chronic cerebellar hemisphere stimulation for the control of intractable epilepsy and for the treatment of cerebral palsy [40]. The rationale was that the output of the cerebellum was predominantly inhibitory. Initial

results were promising but controlled trials failed to show effectiveness [41]. While this was disappointing it did demonstrate that the central nervous system (CNS) could safely be chronically stimulated with implanted electrodes. More recently, Benabid *et al* reported [42] the beneficial effects of chronic stimulation of the ventral intermediate thalamic nucleus for drug-resistant Parkinsonian and essential tremor. This effect was highly specific as regards both site and frequency of stimulation. The results of larger series [43] have been even more impressive in the case of Parkinson's disease. Since this treatment is relatively non-invasive (the electrodes are inserted under local anaesthetic), it has implications for elderly Parkinsonian patients. A third example of continuous excitatory electrotherapy is spinal-cord stimulation, which has both excitatory and trophic effects (see below).

Contingent functional electrical stimulation ranges from the dramatic and central to the homely and peripheral. The first demonstration of the feasibility of chronic contingency stimulation is the lateral popliteal nerve stimulator for foot-drop, which has been in use for nearly 30 years [44]. A heel switch activates stimulating electrodes placed over the lateral popliteal nerve, causing dorsiflexion of the foot and thus preventing the foot catching on the ground during the swing phase of gait.

A more spectacular example was thalamic stimulation for suppressing severe tremor in patients with multiple sclerosis [45]. Stimulation was triggered by the electromyogram from the deltoid muscles, so that the onset of upper-limb movement switched off tremor. Since current was delivered only when needed, the target was spared constant stimulation. This technique was reported over 10 years ago but has been used only in a very few patients with severe multiple sclerosis; the long-term results, as one might expect in patients with a progressive disease, have not been good; moreover, the response was unpredictable and there were peri-operative problems. Nevertheless, individual patients in the small reported series showed remarkable responsiveness.

Functional electrical stimulation systems have been used in patients with neuropathic bladders. The physiological challenge is to cause detrusor contraction without co-contraction of the urethral sphincter [46]. This can be achieved by choosing trains of stimulation such that detrusor contraction outlasts sphincter contraction and, in the interstimulus interval, voiding takes place. There are many unsolved problems, but recent long-term follow-up of paraplegic patients using sacral anterior root stimulators to achieve bladder control [47] found that the vast majority were still using the devices regularly and over 90% of these were always or usually continent. We may now look forward to devices that mobilise descending inhibition which may help with the problem, common in the elderly, of idiopathic detrusor instability where drugs seem either dangerous or ineffective.

The simplest example of a *programmed stimulator* is the so-called diaphragmatic pacemaker, which stimulates the phrenic nerve, used to maintain respiration in subjects with neurogenic hypoventilation, as in high cervical-cord injury [48]. More widely known are the programmed muscle and nerve stimulators designed to achieve walking in people with paraplegia or paraparesis [49]. Advances have continued and systems for patients with incomplete spinal-cord injury look promising [50]. Progress has followed from a greater understanding of how to exploit reflexes, for example, the flexor withdrawal response to achieve flexion at the hip and knee; of the parameters of stimulation that will cause maximally effective muscle response with least discomfort; of the need for the patient to achieve cardiovascular fitness prior to undertaking paraplegic walking; and for conditioning the relevant muscles by chronic electrical stimulation to increase their strength and fatigue resistance. Programming the sequence of muscle and other stimulations so that walking can take place over even rough ground is yet more complicated. Nevertheless, progress has been made, especially with hybrid devices that combine mechanical orthotic support with electrical stimulation. Closed-loop devices that give the subject feedback regarding the position of the limbs—necessary for patients with complete lesions and no proprioception—are becoming increasingly sophisticated. While the young paraplegic patient is a far cry from the elderly stroke patient, progress made with one will help us towards progress with the other; and analogous devices have been tried in patients with hemiparetic stroke [51].

Trophic electrotherapy: central nervous system

Excitatory devices have their limitations. Patients do not welcome being cluttered up with bits of apparatus, however carefully designed. The sense of being manipulated from without is unpleasant. The complexity of the software and hardware required to emulate the activity of the nervous system becomes overwhelming when we are trying to assist patients with tasks more complex and variable than putting one leg in front of another.

The control mechanisms of the nervous system itself show a remarkable propensity to recover after neurological damage, however late in life the injury occurs. We need, therefore, to consider the possibility of exploiting the trophic effects of chronic stimulation of the neuromuscular system in the hope of restoring its structure or assisting spontaneous recovery.

Over the past few decades much has been learned about mechanisms of recovery, such as the unmasking of dormant synapses and collateral sprouting. These, however, may be maladaptive as well as adaptive. Indeed, plastic changes have been suggested as the basis for spasticity [52].

It is now understood that after initial neurological

damage, more widespread adverse changes may take place [53]. Some of these are due to lack of information being transmitted along the relevant tracts. The paralysed arm not only fails to receive the correct neurological input; its lack of normal activity also starves the CNS of relevant information to direct plastic change in ways that are beneficial rather than adverse. The corollary of this is that introducing the correct information may hasten structural recovery. It has been suggested that the normal movement physiotherapists try to encourage or simulate with patients does this [54]. Function does not passively reflect the form or structure of the nervous system: structure is, equally, shaped by function. Just how profound this influence may be has been known for a long time by those who have studied the influence of sensory input on connectivity in the developing sensory system.

How shall we introduce the relevant information into the neuromuscular system to prevent maladaptive changes and to promote adaptive ones? We are a long way yet from being able to talk to the cerebral cortex, or even the spinal cord, in a language it understands. Nevertheless, even in the case of the CNS, some encouraging observations have been made.

Nearly 20 years ago, Cook in New York made the serendipitous observation that patients with multiple sclerosis (MS) coincidentally receiving chronic electrical stimulation of the spinal cord (SCS) for intractable pain also had improvements in their neurological impairments [55]. Further observation in the UK [56–59] showed remarkable and repeatable reductions in lower-limb spasticity, though these did not translate into improved walking and lasted a short time only. Various neurophysiological measurements, such as cervical stomatosensory evoked potentials, also became more normal. More interesting from the point of view of the prospect of inducing useful plastic changes in the nervous system were improvements in longstanding bladder dysfunction: reduced detrusor instability and sphincter dyssynergia with greatly improved bladder capacity and, in some patients, restoration of continence. The mechanism was unclear but the direct mobilisation of propriospinal inhibitory mechanisms, or of descending inhibition from higher levels, or indirect mobilisation of inhibition via activating the central excitatory state, were possibilities. Most interestingly, some of the effects were of slow onset and in many cases outlived the period of stimulation, by months or years, suggesting structural changes.

Spinal cord stimulation is not used routinely in MS because of the technical difficulties of the procedure, the unpredictability of the response, and the continual emergence of new problems in patients with a progressive disease. Nevertheless, the observations supported the belief that longstanding beneficial plastic changes could be brought about by electrotherapy in patients with neurological damage. It was as if the nervous system could extract information from the noise poured into it.

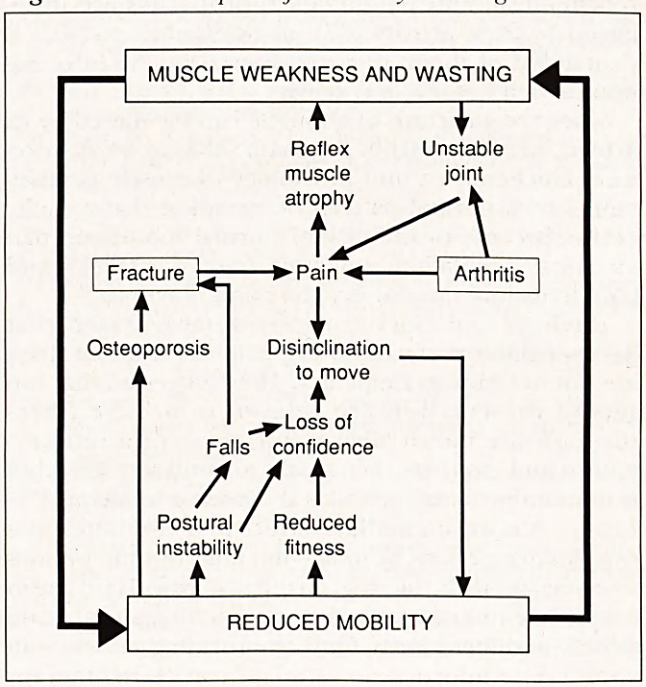
Trophic electrotherapy: skeletal muscle (Fig. 4)

Several layers of darkness separate us from understanding fully what we are doing when we are trying to induce beneficial structural changes in the CNS by stimulating it. We do not know: how far the current spreads and which structures we are stimulating (though advances are being made here [60]); what parameters of stimulation will most favour appropriate growth; or what the more widespread effects of stimulation are and how the nervous system will adapt to them. It is possible that, with more sophisticated computer analysis of neural activity using parallel devices [61], we may be able in future to extract important codes for adaptation.

Until then, it will be sensible to apply our trophic efforts to simpler and more accessible systems. One such system is skeletal muscle. In the elderly the common occurrence of combinations of illnesses can readily lead to a vicious spiral of muscular weakness, immobility, dependency, and loss of confidence (Fig. 4). How shall we break into this vicious spiral? Exercise is often impossible and conventional physiotherapy of limited effectiveness. We need novel methods of restoring muscle strength. They include the potential of drug treatment—beta-agonists and growth hormones, as mentioned earlier. It is here that electrotherapy, directed at bringing about plastic change, will yield its earliest dividends—perhaps even in advance of the 21st century.

Electrotherapy has been used to attempt to restore muscles for many years, usually without any scientific basis: the output of stimulators is frequently unknown to their operators [62] and does not bear any relation

Fig. 4. *The vicious spiral of immobility in old age.*



to plasticity. There has yet to be any study showing that such treatments, given rather haphazardly and half-heartedly, achieve anything. They have rarely been tested and, where tested, as in the case of Bell's palsy [63], have been found to be useless.

Recently, sustained uniform frequency stimulation has been used on an empirical basis and has been found to be effective. Rennie and his colleagues [64] found that relatively low intensity (5% MVC), relatively low frequency (30Hz), percutaneous faradic stimulation of quadriceps muscles for an hour a day over six weeks prevented muscle atrophy and the reduced muscle protein synthesis associated with limb immobilisation following tibial fracture.

More rational attempts to stimulate muscle growth or to reverse wasting are based on the recent understanding that it is not merely the fact or quantity of stimulation that matters but its pattern. The influence of the pattern rather than the mere quantity of muscle activity upon its structure was first suggested by Buller and colleagues [65] in cross-innervation experiments. Reinnervation of a slow-twitch or fast-twitch muscle with a nerve normally supplying a fast-twitch or slow-twitch muscle respectively leads to a transformation of a muscle's phenotype from fast to slow and *vice versa.*

Buller thought that the changes were caused by trophic influences carried by the nerve from some more central source, but Salmons and Vrbova [66] using implanted stimulators showed that transformation of a fast-twitch to a slow-twitch muscle can be brought about without altering innervation by transmitting a low frequency pattern corresponding to that which is normally delivered by action potential traffic to a slow-twitch muscle. This was clear proof of the plasticity of muscle fibres and of how their structural, biochemical, and physiological properties are influenced by their activity [67]: muscles adapt to what is demanded of them; they are altered by the information carried in their own activity.

Since the structure of a muscle can be altered by its activity, it is reasonable to assume that normal structure, biochemistry, and physiology of muscle is maintained by a normal pattern of impulses; that atrophy occurs because of the lack of normal input; and that electrical stimulation emulating normal activity would tend to restore normal structure and function.

Dayhoff and Gerstein [68] demonstrated that nerves exhibit preferred firing patterns and that these are not of uniform frequency. They suggested that two sets of information are relayed to muscle fibres through the motor nerve: one set is of a uniform nature and instructs the muscle to contract; the other is non-uniform and instructs the muscle to adapt. The former has an immediate effect and the latter acts over longer periods of time—the kind of time periods we associate with training. Artificially calculated mean frequencies do not carry this latter information; or do so only in diluted form. Only by providing muscle with appropriate information to adapt can electrotherapy alter phenotype so that both force and endurance are increased. The quantity of adaptive information increases as the muscle is stressed, just as improved muscle performance results from stressful, fatiguing exercise. If this argument is correct, electrotherapy using a stimulator programmed to replicate the information relayed by a motor nerve to fatigued muscle should have the most potent effects.

On the basis of these ideas [54], new electrotherapies are being investigated. Studies have been carried out evaluating the therapeutic effects of 'physiological' patterns of stimulation. The first of these [69] was in patients with traumatic and Bell's facial palsy. In this study truly patterned stimulation was not used, but a stimulation train that corresponded rather precisely to the mean firing frequencies, extracted by analytical EMG techniques, of the fatigued facial muscles of normal subjects. These not-entirely-meaningless modes of stimulation produced encouraging results. This success with uniform frequency stimulation was probably due to using rather more precisely tailored frequencies of stimulation—low frequency stimulation to muscles that are predominantly postural—based on observations of the behaviour of motor units in normal subjects.

However, the really important step was taken by Kidd and Oldham [70,71], using patterned neuromuscular stimulation that emulated the firing patterns of motor units in fatigued muscle. They treated atrophied first dorsal interosseus in patients with rheumatoid arthritis and observed a marked increase in both the force of maximum voluntary contraction (MVC) and fatigue resistance. Significantly, these benefits were not seen with random, unpatterned stimulation. The clinical significance of these changes may be judged by the fact that over 50% of patients awaiting hand surgery cancelled their operations because of improvement in their function. Our group is currently investigating the role of patterned neuromuscular stimulation for patients with quadriceps wasting caught up in the vicious spiral of immobility.

We can imagine future approaches to the vicious spiral of immobility in which patients are given better temporary orthoses, supplemented by functional electrical devices stimulating muscles that have been conditioned—or prevented from wasting—by patterned neuromuscular stimulation and a combination of beta-agonists and growth hormone. If this sounds a daunting package, it is rather less invasive than what surgeons routinely do to their patients. If the alternative is permanent disability and institutionalisation, then the effort must surely be worth it.

The future of rehabilitation of the elderly: wider considerations

This may seem a long way from conventional geriatric rehabilitation, but in a decade I shall be surprised and disappointed if stimulation techniques have not suffi-

ciently advanced to make the devices more comfortable to use, less invasive to have inserted, less obtrusive, more reliable, and carrying more precise neurophysiological prescriptions. Even so, it is time to return to earth. For even when the new treatments have been shown to be effective, even when we are able to talk to the cerebral cortex in the language it understands, so that we can think about tackling problems like the failure of motor programming in the apraxic gait, or even dysphasia—perhaps a job for the 22nd century—the basic principles of good geriatric medicine and good rehabilitation will be no less important.

Much disability in old age, even among those who have a clear medical cause for being disabled, is avoidable. Many patients could be kept out of the vicious spiral of immobility by early intervention, and good whole patient care in an appropriate setting. In addition, we need to take seriously the contribution of medication to the development, or exacerbation, of neurological disability [72]. Inappropriate prescribing for elderly people—'cacapharmacy'—is common [73–76], and up to half of adverse drug reactions and of drug-related admissions may be due to this cause. [77]. It will therefore come as no surprise that frequently the most effective intervention in a disabled elderly person is the careful and considered alteration of medication.

Even in the 21st century, geriatricians will still need the multi-disciplinary, multi-talented team to deal with the complex physical, emotional, and social impacts of disability and its treatments; and to instruct, support, and counsel as well as to treat patients in the narrower 'hi-tech' or 'lower-tech' sense. I do not see a diminished role for therapists in the future—only a changed one. Disablement at any age is complex, never more so than in the elderly. A response that respects that complexity will itself be no less complex.

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References

1 *Population Projections by the Government Actuary 1985–2025,* OPCS, series PP2, 15.
2 Bamford J, Sandercock P, Dennis M, *et al.* A prospective study of acute cerebrovascular disease in the community: the Oxfordshire Community Stroke Project 1981–1986. 1. Methodology, demography and incident cases of first-ever stroke. *J Neurol Neurosurg Psych* 1988;**51:**1373–80.
3 Kay DWK. The epidemiology of dementia: a review of recent work. *Rev Clin Gerontol* 1991;**1:**55–66.
4 Mutch WJ, Dingwall-Fordyce I, Downie AW, *et al.* Parkinson's disease in a Scottish city. *Br Med J* 1986;**292:**534–6.
5 Lawrence JS. Generalised osteoarthrosis in a population sample. *Am J Epidem* 1969;**90:**381–4.
6 Sainsbury R. Hip fracture. *Rev Clin Gerontol* 1991;**1:**67–80.
7 Tallis R, Hall G, Craig I, Dean A. How common are epileptic seizures in old age? *Age Ageing* 1991;**20:**442–8.
8 Malmgren R, Warlow C, Bamford J, *et al.* Projecting the number of first-ever strokes and patients newly handicapped by strokes in England and Wales. *Br Med J* 1989;**20:**333–9.
9 OPCS. Surveys of Disability in Great Britain, *Report 1: The Prevalence of Disability Among Adults.* HMSO, London: 1988.
10 Grundy E. Future patterns of morbidity in old age. In: *Advanced Geriatric Medicine,* vol 6, Caird FI, Evans JG (eds). Bristol, J Wright, 1987.
11 Fries, JF. Ageing, natural death and the compression of morbidity. *New Engl J Med* 1980;**303:**130–5.
12 Robine JM, Ritchie K. Healthy life expectancy: an evaluation of global indicators of change in population health. *Br Med J* 1991;**302:**457–60.
13 Tallis RC. Assessing outcome in rehabilitation. In: *Horizons in Medicine No. 3,* Seymour CA, Summerfield JA (eds). London: Transmedica, 1991.
14 World Health Organisation. *International Classification of Impairments, Disabilities and Handicaps. A Manual of Classification Relating to the Consequences of Disease.* Geneva, Switzerland: WHO, 1980.
15 Mulley GP. Aids for old people living at home. *Rev Clin Gerontol* 1992;**2:**157–69.
16 Bobath B. *Adult Hemiplegia: Evaluation and Treatment.* London: William Heinemann, 1978.
17 Stern PH, McDowell F, Miller JM. Effects of facilitation exercise techniques in stroke rehabilitation. *Arch Phys Med Rehab* 1970;**51:**526–31.
18 Inaba M, Edberg E, Montgomery J. Effectiveness of functional training, active exercise and resistive exercise for patients with hemiplegia. *Phys Ther* 1973;**52:**28–35.
19 Logigian MK, Samuels MA, Falconer J, Zagar R. Clinical exercise trial for stroke patients. *Arch Phys Med Rehab* 1983;**64(8):** 364–7.
20 Dickstein R, Hockerman S, Pillar T, Shaham R. Stroke rehabilitation. Three exercise therapy approaches. *Phys Ther* 1986;**66:**1233–8.
21 Basmajian JV, Gowland CA, Finlayson MA, *et al.* Stroke treatment: comparison of integrated behaviorist physical therapy versus traditional physical therapy programs. *Arch Phys Med Rehab* 1987;**68:**267–72.
22 Young JB, Forster A. The Bradford community stroke trial: results at six months. *Br Med J* 1992;**304:**1085–9.
23 Wade DT, Collen FN, Robb GF, Warlow CP. Physiotherapy intervention late after stroke and mobility. *Br Med J* 1992;**304:**609–13.
24 Sunderland A, Tinson DJ, Bradley EL, *et al.* Enhanced physical therapy improves recovery of arm function after stroke. *J Neurol Neurosurg Psychiat* 1992;**55**:530–5.
25 Sunderland A. Single case experiments in neurological rehabilitation. *Clin Rehab* 1990;**4:**181–92.
26 Smith DS, Goldenberg E, Ashburn A, *et al.* Remedial therapy after stroke: a randomised controlled trial. *Br Med J* 1981;**282:**517–20.
27 Gompertz P, Ebrahim S. (Letter). *Health Trends* 1991;**23:**128.
28 Douglas R, Parson P. The LSU Reciprocating Gait Orthosis. *Orthopaedics* 1983;**6:**834–9.
29 Woods BM, Jones RD. Environmental control systems in a spinal injuries unit: A review of 10 years' experience. *Int Disabil Studies* 1990;**12:**137–40.
30 Leading Article. Being and believing: ethics of virtual reality. *Lancet* 1991;**338:**283–4.
31 Doherty P, Moolenaar CECK, Ashton SV, *et al.* The VASE exon downregulates the neurite growth-promoting activity of NCAM 140. *Nature* 1992;**356**:791–3.
32 Ochs G, Struppler A, Meyerson BA, *et al.* Intrathecal baclofen for long-term treatment of spasticity: a multi-centre study. *J Neurol Neurosurg Psychiat* 1989;**52:**933–9.
33 Rothwell NJ, Stock NJ. Effect of a selective beta 2-agonist (clenbuterol) on energy balance and body composition in normal and protein deficient rats. *Bioscience Reports* 1987;**7:**933–40.
34 Martineau L, Horan MA, Little RA, Rothwell NJ. Effect of salbutamol on skeletal muscle functional capacity in man. *Proc Nutr Soc* (in press).

35 Rudman D, Feller AG, Nagraj HS, *et al.* Effects of human growth hormone in men over 60 years old. *N Engl J Med* 1990;**323:**1–6.

36 Jiang Z–M, He G–Z, Zhang, S–Y, *et al.* Low-dose growth hormone and hypocaloric nutrition attenuate the protein-caloric response after major operation. *Ann Surg* 1989;**210:**513–24.

37 Gage FH, Bjorkland A. Neural grafting in the aged brain. *Ann Rev Physiol* 1986;**48:**447–59.

38 Williams A. Cell implantation in Parkinson's disease. *Br Med J* 1990;**301:**301–2.

39 Gage FH, Buzsaki G. CNS grafting: potential mechanisms of action. In: *Neural Regeneration and Transplantation,* Seil FJ (ed). New York: Alan Liss, 1989.

40 Cooper IS. Effect of chronic stimulation of anterior cerebellum on neurological disease. *Lancet* 1973:**1:**206.

41 Wright GD, McLellan DL, Brice JG. A double-blind trial of chronic cerebellar stimulation in twelve patients with severe epilepsy. *J Neurol Neurosurg Psychiat* 1984;**47:**769–74.

42 Benabid AL, Pollak P, Gervason C, *et al.* Long-term suppression of tremor by chronic stimulation of the ventral intermediate thalamic nucleus. *Lancet* 1991;**337:**403–6.

43 Benabid AL. (Oral presentation). First Congress of the International Neuromodulation Society. Rome, May 3–6, 1992.

44 Liberson WT, Holmquist HJ, Scott D, Dow A. Functional electrotherapy: stimulation of the peroneal nerve synchronised with the swing phase of gait in hemiplegia. *Arch Phys Med Rehab* 1961;**42:**101–4.

45 Brice J, McLellan L. Suppression of intention tremor by contingent deep-brain stimulation. *Lancet* 1980;**i:**1221–2.

46 Brindley GS. Treatment of urinary and faecal incontinence by surgically implanted stimulators. *Ciba Foundation Symposium* (Netherlands) 1990;**151:**267–74.

47 Brindley GS, Rushton DB. Long-term follow-up of patients with sacral anterior root stimulator implants. *Paraplegia* 1990;**28:** 469–75.

48 Miller JL, Farmer JA. Stuart W, Apple D. Phrenic nerve pacing of the quadriplegic patient. *J Thorac Cardiovasc Surg* 1990;**99:** 35–9.

49 Kralj A, Bajd T. *Functional Electrical Stimulation: Standing and Walking after Spinal Cord Injury.* Boca Raton, Florida; CRC Press, 1989.

50 Granat M, Keating JF, Smith ACB, *et al.* The use of functional electrical stimulation to assist gait in patients with incomplete spinal cord injury. *Disabil Rehab* 1992;**14:**93–7.

51 Marsolais EB, Kobetic R. Functional walking in paralysed patients by means of electrical stimulation. *Clin Orthopaed Rel Res* 1983;**175:**30–6.

52 Devor M. Plasticity in the adult nervous system. In: *Rehabilitation of the Neurological Patient.* Illis LS, Sedgwick EM, Glanville HJ (eds). Oxford: Blackwell Scientific Publications, 1982.

53 Illis LS. Determinants of recovery. *Int Rehab Med* 1982;**4:**166–72.

54 Kidd G, Musa I, Lawes N. *Understanding Neuromuscular Plasticity.* London: Edward Arnold, 1992.

55 Cook AW, Weinstein SP. Chronic dorsal column stimulation in multiple sclerosis: preliminary report. *N Y State J Med* 1973;**73:** 2868–72.

56 Illis LS, Sedgwick EM, Tallis RC. Spinal cord stimulation in multiple sclerosis: clinical results. *J Neurol Psychiat* 1980;**43:**1–14.

57 Sedgwick EM, Illis LS, Tallis RC, *et al.* Electrophysiological responses and stimulus parameters associated with spinal cord stimulation. *J Neurol Neurosurg Psychiat* 1980;**43**(1):15–24.

58 Illis LS, Read DJ, Sedgwick EM, Tallis RC. Spinal cord stimulation in the United Kingdom. *J Neurol Neurosurg Psychiat* 1983;**46:**299–304.

59 Read DJ, Mathews WB, Higson RH. The effect of spinal cord stimulation on function in patients with multiple sclerosis. *Brain* 1980;**103:**803–33.

60 Coburn B, Sin WK. A theoretical study of epidural electrical stimulation of the spinal cord—Part 2: Effects on long myelinated fibres. *Trans Biomed Engineering* 1985;**32:**971–7.

61 Bechtel W, Abrahamsen A. *Connectionism and the Mind: An Introduction to Parallel Processing in Networks.* Oxford: Blackwell Scientific Publications, 1991.

62 Lloyd JJ, Evans JA. A calibration survey of physiotherapy equipment in North Wales. *Physiotherapy* 1988;**74:**56–61.

63 Taverner D. Bell's palsy. *Brain* 1958;**78:**209–27.

64 Gibson JNA, Smith K, Rennie MJ. Prevention of disuse muscle atrophy by means of electrical stimulation: maintenance of protein synthesis. *Lancet* 1988;**ii:**767–9.

65 Buller AJ, Eccles JC, Eccles RM. Interactions between motor neurones and muscles in respect of characteristic speeds of their responses. *J Physiol* 1960;**150:**417–39.

66 Salmons S, Vrbova G. The influence of activity on some contractile characteristics of mammalian fast and slow muscles. *J Physiol* 1969;**210:**535–49.

67 Nix WA, Vrbova G. (eds) *Electrical Stimulation and Neuromuscular Disorders.* Berlin: Springer Verlag, 1986.

68 Dayhoff JE, Gerstein GL. Favoured patterns in spike trains. 2-Application. *J Neurophysiol* 1983;**49(6):**1349–63.

69 Faragher D, Kidd GL, Tallis RC. Eutrophic electrical stimulation for Bell's palsy. *Clin Rehab* 1987;**1:**265–71.

70 Kidd GL, Oldham JA. Motor unit action potential (MUAP) sequence and electrotherapy. *Clin Rehab* 1988;**2:**22–33.

71. Kidd GL, Oldham JA, Stanley JK. A comparison of uniform patterned and eutrophic electrotherapies in a clinical procedure of rehabilitation of some movements in the arthritic hand. *Clin Rehab* 1989;**3:**27–39.

72 Swift C. Drug-induced neurological disease. In: *The Clinical Neurology of Old Age.* Tallis RC (ed). Chichester: John Wiley, 1989.

73 Gosney M, Tallis RC. Prescription of contraindicated and interacting drugs in elderly patients admitted to hospital. *Lancet* 1984;**i:**564–67.

74 Adams K, Al-Hamouz S, Edmond E, *et al.* Inappropriate prescribing in the elderly. *J Roy Coll Physicians Lond* 1987;**21**(1): 39–41.

75 Browne M, Tallis RC, Vellodi C, *et al.* A computer-assisted survey of contraindicated and interacting drugs in long-stay geriatric patients. *Brit J Pharm Prac* 1987;**9(7):**250–4.

76 Gosney M, Vellodi C, Tallis RC, *et al.* Inappropriate prescribing in Part 3 residential homes for the elderly. *Health Trends* 1989;**21**(4):129–31.

77 Lindley CM, Tully MP. Paramsothy V, Tallis RC. Adverse drug reactions in the elderly: the contribution of inappropriate prescribing. *Age Ageing* (in press).