

# Neurophysiology

In all the clever affairs of man, a consistent pattern runs through his search for knowledge and for better ways of doing a set task.

First there is the steady accumulation of learning, based on the results of years of work and the experience of many other people.

Then there is a continued modification and adaptation, made possible by the use of new techniques, and then a steady forward march again to new objectives.

But sometimes there occurs a lightning flash which illuminates the whole field, and a breakthrough is established which will affect all future work in that particular area.

Within the compass of this chapter, I can only give a brief resume of the direction our research patterns are trending. Our funds are necessarily limited, and require us to carefully equate the value of a dollar spent on a research project with the demands of CP children's therapy and adults' work training procedures. We could advance the argument that all therapy and surgical operations are a waste of time; that we could spend the money more effectively, if we concentrated on the next generation, in finding why cerebral palsy happens in the first place. The Spastic Centre exists only for its present CP children and adults. Therefore, the levels of medical therapy and social maturation must be considered first, and only then can our research programmes be done, on a shoestring, depending upon the interest and the fibre of the people involved. We owe our Director of The Spastic Research Unit, Dr Peter Neilson, and all his staff, a lot!

For this chapter, I have asked Dr Neilson to give me a resume of his work during the eighteen years that have elapsed since he accepted the challenge of researching the reasons for 'spasm' of the muscle of cerebral palsy.

We know full well that there are no magic wands in research, or in therapy generally. What we have put in, necessarily limits what we can expect to get out, in effort, or in finance. Part of the problem is to interpret the findings of the research into the neurophysiology of the muscles of the cerebral palsied, into the associated areas of therapy, where we are dealing with abnormal muscle groups affecting posture and movement. We hope that the development of better muscle and speech training facilities, associated with better educational techniques and more advanced work training, would give the CP, in the years to come, a much better opportunity of making use of their undoubted intelligence.

The Spastic Centre of New South Wales has an active policy of encouraging both basic and applied research into cerebral palsy. Since The Spastic Centre was established in 1945, conservative therapy and management has reduced the consequential orthopaedic defects of this condition, probably about as far as this approach can be taken. Research into the neurophysiology of the basic problem of abnormal muscle tone has contributed little to its control. However, we continue to hope that it will do more in the immediate future.

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The term 'cerebral palsy' implies damage to parts of the brain concerned with the control of movement. This damage can occur before, during, or shortly after birth. Cerebral palsy does not imply epilepsy perceptual problems, behavioural problems or mental retardation. All of these terms including cerebral palsy, are covered by the more general term 'brain damage syndrome'.

Cerebral palsy is only the motor component of the brain damage syndrome. Individuals with cerebral palsy often have diffused brain damage, however, many display multiple aspects of the brain damage syndrome, together with cerebral palsy. Nevertheless, it is important to realise that even severely disabled individuals with cerebral palsy are not necessarily mentally retarded.

As a result of damage to motor control centres of the brain, cerebral palsied persons often display bizarre postures and stereotyped facial expressions. They frequently have difficulty with swallowing and drooling is common. They may develop deformities of bones and joints, particularly as the ankle, hip, hand and neck muscles may become physically shortened, a state known as 'muscle contracture', and have a limited range of movement.

Individuals with cerebral palsy (CPs), often display writhing, sinuous involuntary movements, particularly of the hands and fingers, as well as tremors. These involuntary movements are referred to as the involuntary movements of athetosis. Their attempt at volitional movement is usually uncoordinated, slow and clumsy, and often non-functional. Their speech is frequently unintelligible and is referred to as 'dysarthric speech'. They suffer from intermittent spasms or vigorous involuntary contractions of muscles, which often lead to deformities of joints and ligaments, requiring surgical correction. Automatic postural responses are often abnormal. Attempts to move the limbs of a CP individual are opposed by active contractions of the muscles, producing a feeling of rigidity and tightness, referred to as 'rigidospasticity'.

A newborn cerebral palsied baby may have all of the infantile reflex responses of a normal newborn, and so it is often difficult to make an early diagnosis. As the weeks and months go by, however, normal motor development is not observed. The motor development process is slowed down and in many cases, the motor milestones are not reached. It is usually possible, within the first two to three months, to make a diagnosis of cerebral palsy, although cerebral palsied babies are often not diagnosed until towards the end of the first year.

CP children retain many of the so-called primitive or infantile reflex responses, and also display abnormal or pathological reflex responses. If a severely disabled CP child is placed on his back, he may display a whole body extensor spasm, so he forms an arching bridge between his heels and the back of his head. If the same child is then rolled onto his stomach he may switch into a flexor spasm, in which his knees are brought up towards his chest and his head is flexed forward. Rotation of the head can produce reflex movement of the arms and legs, the so called 'tonic neck reflexes'

What are the causes of these problems? What are the neurophysiological mechanisms which underlie these motor disabilities of cerebral palsy? What can be done to ameliorate these problems? If we recognise that brain damage is irreversible, what can be done to ensure that an individual CP leads a life in which he can achieve his full potential and contacts with others, in which he has a minimum deformity and requires a minimum of medical or surgical intervention?

Before these questions can be answered, we must develop an understanding of the underlying neurophysiological mechanisms responsible for the various motor symptoms of cerebral palsy. Little is currently known about these mechanisms; the prevailing therapy techniques recognise the major problem as being associated with abnormal postural reflex control. In the Bobath method, the CP child is encouraged to remain in postures opposite to those in which he is normally pushed by reflexes. He is encouraged to inhibit the reflexes, so he can remain in so-called 'reflex inhibiting postures'. The focus of Bobath therapies on reflex inhibition and proprioceptive neuromuscular facilitation (PNF), is one reason for starting our research programme with an investigation of reflex behaviour and reflex mechanisms. Another reason was that, as shown at the beginning of the century, there already existed an animal model for rigidospasticity. Although it is now appreciated that the decerebrate animal is not a good model for human rigidospasticity, this research provided motivation for us to begin our research programme with an investigation of stretch reflex behaviour in cerebral palsy.

At the beginning of our studies, the twitch contraction produced by tapping the tendon with a reflex hammer was the best known illustration of the stretch reflex. The tendon jerk had been used for many years by neurologists to test the integrity of spinal circuits. The H-reflex, an electrical equivalent of the tendon jerk, had also been studied. Instead of tapping the tendon with a reflex hammer, an electrical stimulus is applied to the nerve to evoke a stretch reflex twitch contraction of the muscle. The characteristics of this electrical tendon jerk reflex have been much studied and were well known at the beginning of our research.

It was generally believed by neurologists that rigidospasticity, assessed clinically by asking the patient to relax while passively moving the limb back and forth about the joint and feeling the resistance to movement, was caused by an exaggerated sensitivity of the same stretch reflex circuit responsible for the tendon jerk reflex. Patients with exaggerated resistance to passive movement usually have brisk tendon jerk reflexes. It was argued, therefore, that the hypersensitive tendon jerk stretch reflex circuit was also responsible for the exaggerated resistance to passive movement.

There were some neurologists, however, among them Professor James Lance, head of the Department of Neurology at Prince Henry Hospital (The Spastic Centre Research Unit is one of the laboratories within this Department), who were not satisfied with this explanation of rigidospasticity, because they often saw rigidospastic patients with no brisk tendon jerk reflex responses, or vice versa. The best example is observed in cerebellar patients, who display a so-called 'pendular' knee jerk. These patients are hypotonic; their limbs feel loose and floppy. Nevertheless, a brisk tendon jerk reflex response can be elicited in these patients. The foot swings forward, driven by the hypersensitive twitch contraction of the tendon jerk reflex, and then swings back and forth

like a loose pendulum, because of a lack of muscle tone. Due to these observations, Professor Lance recognised the importance of an experimental finding in our laboratory, which later became known as the Tonic Vibration Reflex. We found that when vibration was applied to a muscle, it suppressed the tendon jerk reflex responses. As soon as the vibration was removed from the muscles, the tendon jerk reflex responses reappeared. At the same time, however, the vibration evoked its own sort of reflex response which consisted of a slowly augmenting tonic contraction of muscle. This differential effect indicated that there were two reflex mechanisms – a phasic reflex mechanism, which produced the tendon jerk reflex responses, and a tonic reflex mechanism that was excited by vibration and produced a sustained contraction of the muscle.

This provided the first experimental evidence that two or more stretch reflex mechanisms exist in human muscles, and provided a possible explanation for the observed dissociation between the sensitivity of tendon jerk reflexes and the level of rigidospasticity assessed clinically. However, the characteristics of the Tonic Vibration Reflex do not correlate well with clinical measures of rigidospasticity. Nevertheless, the differential effect of vibration on phasic and tonic reflex mechanisms demonstrated in the original experiment emphasised the possibility that other reflex mechanisms may exist which are responsible for rigidospasticity.

The problem now was to find those reflexes. It was decided to study the electromyographic characteristics of tonic stretch reflexes by slowly moving the limbs backwards and forwards about the joint, in the same way as during clinical assessment of rigidospasticity, while recording joint angle movements, thereby measuring the magnitude, amplitude, and velocity of stretch applied to the muscles, and at the same time using electromyography to record the reflex contraction patterns of the various muscles operating across the joint. In brief, the results of these studies showed that if a normal subject is asked to relax while his limbs are moved passively back and forth by an examiner, there is no abnormal resistance to the movement and no reflex contractions of muscles, and the joint feels loose. When this test is applied to a CP patient, however, there are reflex responses in the muscles, and there is an abnormal resistance to the movement. The resistance is associated with abnormal asynchronous bursts of reflex contraction, which occur at various times in different muscles operating across the joint. In any one muscle, an asynchronous burst of EMG activity is usually produced during the early part of the stretching cycle. For example, if one were stretching the limb backwards and forwards rhythmically, say at 1 – 2 Hz through about thirty degrees change of joint angle, every time the movement swings into the stretching phase a burst of EMG activity is recorded from the muscles being stretched. The muscles then remain silent for the rest of the stretching cycle. Such reflex contractions could be responsible for the feeling of tightness experienced in rigidospastic patients. In CP patients there is a velocity threshold of muscle stretch, below which no reflex response is produced in any of the muscles. As the velocity of stretching is increased above the velocity threshold, the size of the EMG burst increases in proportion to the velocity. It was thought that this might provide a method of quantifying the amount of rigidospasticity in a muscle. If the size of the EMG reflex response is graphed against the velocity of stretch, then both the threshold and the slope of the straight-line relationship provides a measure of the degree of rigidospasticity. The lower the velocity threshold, the more spastic the muscle, and the steeper the slope, the more spastic the muscle. This method of assessing spasticity has since been used by a number of researchers investigating the effects of drugs on rigidospasticity.

At The Spastic Centre Research Unit, we were still not convinced, however, that this method of studying tonic stretch reflexes revealed the physiological mechanisms responsible for rigidospasticity and disruption of voluntary movement in cerebral palsy. The prevailing clinical idea was that abnormal postural reflexes in cerebral palsy disrupt voluntary movement. That is, abnormal postural reflexes compete with the voluntary system for control of muscles, and the reflexes dominate because of their hypersensitivity. The reflexes push or pull the patient into various postures – reflexively splinting him in those postures, thereby disrupting control of movement. Thus, the stretch reflexes responsible for disrupting control of movement in cerebral palsy must be those abnormal reflexes operating in competition with voluntary movement. The tonic stretch reflexes described above were measured while the subject was attempting to relax. It seemed highly probable, therefore, that a different stretch reflex mechanism might be activated when the subject attempted to voluntarily contract muscles.

Another line of reasoning also indicated that we should attempt to measure stretch reflex responses during voluntary activity. The tonic stretch reflexes described above were not observed in normal relaxed subjects, but only observed in movement-disordered patients, such as spinal injured patients, hemiplegic patients, CP patients, Parkinsonian patients and patients with cerebellar damage. This had led to the view that the tonic stretch reflex was a pathological phenomenon. It seemed to us that pathways for such complex stretch reflex mechanism were unlikely to exist within the nervous system without there being some reason for their existence in terms of normal mechanisms. Although there were no tonic stretch reflex responses present in normal subjects when they were relaxed, it seemed likely that the tonic stretch reflex mechanisms might be activated during voluntary activity.

For both of the reasons mentioned, it was decided to investigate tonic stretch reflexes while the subject was voluntarily active. This requirement considerably complicated the measurement analysis technique. When a muscle is being contracted voluntarily, a sustained EMG signal is produced, which consists of an interference pattern of electrical spikes. When a stretch is applied to the muscle, the tonic stretch reflex response is also indicated by a burst of electrical impulses in the EMG, mixed with the

background activity. Consequently, we have the problem of extracting the tonic stretch reflex responses from the background activity. This can be achieved by using an averaging technique in which the reflex responses to a series of stretches are averaged together. Only the part of the EMG activity consistently present in each response averages up; the irregular varying background activity averages to zero.

More sophisticated computer analysis techniques, employing cross-correlation and spectrographic analysis, have been developed for technological applications such as seismographic analysis. These procedures have the advantage, that not only can they solve the problem of separating reflex EMG responses from the background activity, but also they provide a mathematical description of the transmission characteristics of the stretch reflex pathways. Consequently, at the Spastic Centre Research Unit, we developed computer programmes to perform this cross-correlational and spectrographic analysis procedure, and applied these programmes to analysis of EMG signals and joint angle signals recorded from normal and cerebral palsied subjects during sustained voluntary activity.

Large tonic stretch reflex (TSR) responses were clearly present in normal subjects during voluntary activity. The very process of voluntarily contracting muscle seemed to activate the TSR circuits associated with that muscle. These TSR responses recorded during voluntary activity in normal subjects were very powerful responses, producing large swings in the contraction of the muscle. A small displacement of five to thirty degrees at the joint can drive the muscle through almost its full range of contraction. The responses were certainly large enough to be of functional importance during movement. The computer analysis procedure provided detailed technical specifications of the closed-loop transfer function transmission characteristics of the reflex loops involved in generating the responses. This was exactly the knowledge we needed to investigate the behaviour and the functional role of these tonic stretch reflexes during voluntary movement.

*We had discovered a new and powerful TSR, brought into play during voluntary activity. We had revealed a muscle control mechanism that was involved in the pathological process responsible for the generation of rigidospasticity in cerebral palsy.*

The next step was to study TSR during voluntary activity in CP subjects. Although the TSR responses were grossly abnormal in CP subjects, TSR pathways were also functionally reorganised in CP during voluntary activity. Although CP patients displayed TSR responses when they were trying to relax, when the same CP subjects voluntarily contracted muscles, the TSRs were reorganised. The reorganised responses were more sensitive to stretch, the responses were much larger, and the timing of the responses was completely different from that measured when they were relaxed. In other words, both the magnitude and timing of the TSR responses measured during voluntary activity were completely different from the TSR responses measured in the same CP subjects attempting to relax. It was clear that we were studying a different reflex mechanism during voluntary activity from that studied in the same CP subject at rest. *We coined the terms 'Resting Tonic Stretch Reflex' and 'Action Tonic Stretch Reflex' to differentiate between these two types of reflexes.* An important consequence of this discovery was that the rigidospasticity assessed clinically in CP subjects (by asking them to relax and passively moving their limbs back and forth about the joint and experiencing the resistance to the movement) was very different from the rigidospasticity experienced by the CP person himself when he attempted voluntarily to move his own limbs. In other words, the rigidospasticity experienced by a CP individual during voluntary movement was quite different from the rigidospasticity assessed by the clinician with the subject at rest.

The functional importance of the difference between the resting TSR, and the action TSR, in cerebral palsy was further emphasised by the results of a double-blind drug study conducted to assess the efficacy of the drug Phenoxybenzamine in controlling the involuntary movements of athetosis. Results of this investigation revealed that the drug Phenoxybenzamine Veldopa suppressed resting TSR responses and was effective in reducing rigidospasticity as assessed clinically. The drug had no influence, however, on the action TSR responses and produced no improvement in functional control of movement as assessed by visual pursuit tracking tests. This differential effect of the drug Phenoxybenzamine on resting and action TSR responses provided further experimental evidence in support of the hypothesis, that additional reflex circuitry is brought into play by descending neural activity during voluntary activity. It also supports the notion that it is the action TSR and rigidospasticity assessed during voluntary activity that is important with respect to functional control of movement in cerebral palsy, rather than the rigidospasticity assessed clinically when the CP patient is at rest.

Action TSR studies were performed on two groups of CP subjects. The first group was predominantly spastic, the second group was predominantly athetoid. Results showed that the characteristics of the action TSR in CP subjects are grossly abnormal, with distinct and significant differences between the spastic and athetoid groups. Action TSR characteristics were also investigated in cerebellar patients and found to represent yet a third type of abnormality. The abnormality seen in different motor-disordered patient groups was found to be consistent with the clinical picture of the disorder.

Nevertheless, results of these studies of action TSRs in CP indicated that the disorder of action TSR transmission was not so much a disorder of the stretch reflex circuits. This indicated that the pathophysiological mechanisms responsible for rigidospasticity and

spasm in CP would be found, not in the stretch reflex circuits themselves, but in the parts of the brain involved in descending control of reflex transmission. *In our search for the neurological course of rigidospasticity and spasm, it was now clear that we should investigate the brain mechanisms involved in descending control of action TSR transmission.*

Normal subjects are usually not conscious of descending control of stretch reflex transmission. In fact, when you think about it, almost all of the mechanisms that are involved in producing a voluntary movement are not available for conscious examination. We are usually not aware which muscles we are contracting, let alone what nerve signals we are sending down which motor nerves. Reflex transmission control seems, subjectively, to be an automatic path of voluntary movement. Presumably, if the descending control systems were damaged in CP, the individual CP would be unaware he was inappropriately tuning reflex transmission. The consequences of faulty descending control would be experienced, however, as spasm and rigidospasticity.

There are two important reasons for asking the question, 'Can reflex transmission be brought under conscious control?' Firstly, if it were possible to teach CPs to switch off defective stretch reflexes, this would be equivalent to suppression of rigidospasticity and spasm. This would represent an important advance in therapy for CP, particularly with respect to prevention of muscle and joint deformity. The second reason for asking this question is, that if we could teach normal subjects consciously to control reflex transmission, we would be able to study the associated changes and this would give us some idea of the range of normal descending control and of its functional significance during voluntary movement. We would then be able to recognise abnormalities of descending reflex transmission control in CP.

One of the first things we studied concerning the nature of this descending control of TSR transmission, was the effect of changing the level of voluntary contraction of the muscle. Able bodied subjects contracted elbow muscles to various proportions of a maximum contraction while transmission characteristics of the action TSR were measured. We found a systematic change in the characteristics of the TSR with changes in contraction level – as the contraction level increased, sensitivity of the TSR increased. We also found a systematic change in the timing of the reflexes with changes in contraction level – as the contraction level increased, the phase lead of the EMG responses ahead of muscle stretch increased. It was thus demonstrated, that descending control of TSR transmission is not a simple switching of the reflexes on or off, but a graded control in which the sensitivity and the timing of the stretch reflexes change in proportion with the contraction level of the muscle.

We repeated this experiment with a group of CP subjects, and found that graded descending control of reflex transmission was absent or at least disrupted in CP. As soon as a CP contracted the muscle, the descending TSR control acted like a switch and an abnormal action TSR was switched on. When a CP subject produced even a small voluntary contraction, the TSR was switched on with an abnormally high sensitivity, and then remained switched on, despite subsequent changes in contraction level. A clear abnormality was thus demonstrated in the descending control of stretch reflex transmission in CP. Normally, the sensitivity of the TSR increases in proportion with the level of contraction of the muscle, but in CP subjects the TSR has high sensitivity even at relatively low contraction levels. In other words, in CP, the descending control of reflex transmission does not appear to be coordinated with voluntary control of muscle contraction as it is in normal subjects. This is consistent with the notion that stretch reflexes compete with, rather than cooperate with, voluntary signals for control of muscle contraction. But, does the sensitivity of the TSR in normal subjects always increase or decrease in proportion with the contraction level of the muscle, or can the sensitivity vary independently of the contraction level? Independent control of sensitivity would greatly increase the adaptive control capabilities of the nervous system, and would indicate the existence of additional brain mechanisms involved in coordinating reflex transmission with voluntary activity. This is an important question to answer, since it helps define the brain mechanisms, which when damaged in CP, lead to faulty descending control of TSRs and hence to rigidospasticity and spasm.

To resolve this question, we developed computer programmes to provide an on-line visual display of both the sensitivity of the reflex, and the contraction level of the muscle. We then asked normal subjects to try and hold the contraction level constant as indicated on one of the visual displays, and to attempt to reduce the reflex sensitivity indicated on the other display. We used this biofeedback training method to discover whether or not it is possible for the central nervous system to vary the sensitivity of tonic stretch reflexes independently of the contraction level of the muscle. Within one hour's training, ten of the eleven normal subjects tested were able to reduce the sensitivity of the TSR three to fourfold while holding the contraction level constant at 30 to 50 per cent of a maximum contraction. This was an exciting result. It demonstrated the potential of using biofeedback to teach

CP subjects consciously to suppress rigidospasticity and spasm. *It also indicated the existence of complex central mechanisms involved in descending control reflexes that could be damaged in CP. Almost nothing was known about this mechanism.*

What is the functional significance of descending control of reflex transmission during voluntary movement? How is the descending control system disrupted in CP? An important clue to the role of descending modulation of reflex transmission was noticed during the biofeedback experiments. Despite the fact that subjects held the contraction levels of the muscles constant, the stiffness of the limb, as experienced by the experimenter, felt looser when the stretch reflex sensitivity was set low than when it was set high.

Following this observation, we performed a series of experiments, in which the mechanical force-displacement characteristics of the elbow joint were measured, while the contraction levels of the muscles were held constant. The elbow joint was shown to be mechanically equivalent to an inertia restrained by both stiffness and viscous elements. In other words, the mechanical characteristics of the elbow resembled those of an automobile suspension system with a spring and a dashpot damper. As the descending influences controlling the transmission characteristics of the stretch reflex pathways varied, the stiffness and viscosity of the equivalent spring and dashpot damper varied. These data showed that the stiffness and viscous mechanical characteristics of the elbow are determined predominantly by the involuntary tonic stretch reflex contractions of the muscles. Moreover, both the stiffness and viscosity can be varied by the nervous stem controlling the descending modulation of interneurons involved in stretch reflex transmission. In turn, the sensitivity and timing characteristics of the stretch reflex pathways determine the stiffness and viscous characteristics of the joints. This data provide compelling evidence in support of the hypothesis that the central nervous system normally tunes the mechanical stiffness and viscosity of the skeletal joints by modulating the transmission characteristics of the TSR pathways within the central nervous system. The data supports the view that brain damage sustained in cerebral palsy disrupts descending modulation of TSR transmission, and thereby produces abnormal stiffness and viscosity of the joints, which is experienced clinically as rigidospasticity.

*We now had to determine whether or not conscious control of stretch reflex transmission could be taught to severely disabled CP individuals.* Benefits, such as reduction in spasm, reduced muscle tightness, increased range of joint movement and prevention of muscle and joint deformities would ensue. According to the prevailing views, if a CP could learn this descending control of TSRs there might be an unmasking of the unrelying voluntary control ability, which would otherwise be 'splintered' by spasm and rigidospasticity. Even if it took years for a CP to learn to control TSR transmission, the benefits would be more than worth the effort.

Firstly, we conducted a pilot study. Two intelligent adult CPs volunteered to work with us for two weeks. Using the biofeedback visual displays, they attempted to hold the contraction level of the muscle constant while decreasing reflex sensitivity. After two weeks training they were able to produce small but significant changes in stretch reflex sensitivity. This finding indicated that it is possible for a CP to learn consciously to suppress spasm and rigidospasticity. It also indicted, however, that training would be difficult and slow. A training period of one or two years might be required to learn to control rigidospasticity, and perhaps a lifetime of continuous retraining to maintain the skill. Nevertheless, the technique had such exciting potential it was considered worthwhile setting up a training programme. Accordingly, an Action Reflex Training room was established at Centre Industries and a small group of adult CPs volunteered to attend the training room for one hour each working day. We provided biofeedback displays of muscle contraction level and reflex sensitivity, and we recorded progress in learning to self-regulate TSR transmission, that is, spasm and rigidospasticity. We also employed a visual pursuit-tracking test to assess functional control of movement; movement of the elbow was used to control the vertical movement of a response marker on a visual display screen. The task for the subject was to move the elbow in such a manner that the response marker followed a slowly moving stimulus marker on the display. Movements of both stimulus and response markers were recorded and analysed. This provided detailed objective measures of the CPs functional control of elbow movement, since we knew exactly the movement the CP was trying to make and had precise measures of the movement actually made. It also provided an objective description of inappropriate and involuntary movements at the elbow, and enabled us to describe three types of movements, which characterise the involuntary movements of athetosis. This work led to the discovery of the so called 'athetoid action tremor', a vigorous tremor or bursting in the contraction of athetoid muscles which varies irregularly at frequencies between 2 - 4 Hz, and is uncorrelated in agonist and antagonist muscles.

After twelve months training, all of the CPs in the group succeeded in controlling action tonic stretch reflex sensitivity independently of contraction, and showed a reduction in the amount of spasm and rigidospasticity. The training led to a reduction in muscle tightness and increased the range of movement in the joint.

*This experimental result is clearly of great importance with respect to therapy, particularly with respect to prevention of muscle contractures and joint deformity.* It demonstrated that the damaged brain in CP has sufficient plasticity to acquire, given appropriate training, some inhibitory control over reflex transmission. This is an essential first step in any therapy programme, not only to reduce muscle tightness and prevent deformity, but also to unmask underlying functional control of movement

From a scientific point of view, however, we still have the problem of tracing these descending signals into the brain and finding which parts of the brain are involved in modulating reflex transmission, and what goes wrong with those parts of the brain in CP.

A problem in assessing rigidospasticity in CP was revealed by this data. We found that reflex sensitivity can be influenced by the size of the stretch applied to the muscle. Although we had been carefully controlling the contraction levels of the muscles and

the velocities of the stretch, we found that the sensitivity of the stretch reflex increased as the magnitude of the stretch was decreased. This is very important in clinical assessment of rigidospasticity, because, as the rigidospasticity of the joint decreases with training, there is a tendency for the examiner to move the limb (which now feels looser) through a larger range of movement at a lower frequency. Consequently, the dependence of TSR sensitivity on the magnitude of stretch confounds the measurement of rigidospasticity and therefore must be taken into account during future design of spasticity meters. We had not controlled the magnitude of stretch during the tests with the first group of CP subjects, so we found it necessary to start a new group of CP subjects, this time with contraction level, velocity, frequency and magnitude of stretch all carefully controlled. For this second group of CPs, we obtained the same results as with the first group, but of course the data was no longer confounded by the effects of changing the magnitude of stretch. *Within twelve months, all the subjects learned to self-regulate spasm and rigidospasticity. The limb felt loose, and involuntary contractions of the muscles were reduced.*

We are continuing to experiment with the use of EMG biofeedback training to teach CPs to self-regulate spasm and rigidospasticity. We are attempting to determine which particular aspect of training is most responsible for the observed improvement. During training, CPs learn to contract the muscles being studied without producing contractions in neighbouring muscles. They learn to relax the muscle (which is different from reflex sensitivity control during active contraction), and they learn to sustain a voluntary contraction with a minimum of involuntary fluctuation. Any or all of these skills could be contributing to the observed improvement. We are also experimenting with the use of EMG biofeedback training with four to seven year old CP children at risk of developing contracture of the calf muscles and deformities of the ankle joints. The question being investigated is, Can the tightness of the calf muscles be reduced sufficiently to prevent or even reverse the development of muscle contractures in children?

Another important result was obtained from our studies of action reflex training in CP. Although treatment of spasm and rigidospasticity is an essential component of therapy for CP, results from the biofeedback training studies indicate that spasm, rigidospasticity and involuntary contractions are not the primary cause of motor disability in cerebral palsy. All of the CPs in the training programme succeeded in reducing spasm, rigidospasticity and involuntary contractions of the elbow muscles, but their ability to control elbow movements, as assessed by pursuit tracking tests, remained very poor. It seems that the lack of functional control is caused, not so much by abnormal reflexes competing for control of muscles, but by a fundamental inability to formulate an appropriate sequence of motor commands to achieve a desired movement. This is an important deviation from the traditional explanation for motor deficiency in cerebral palsy. The notion of a movement programming defect as the primary cause of disability in cerebral palsy was further supported by our work on the contraction patterns of lip, tongue and jaw muscles during speech in CP.

CPs often lack functional control of the speech muscles and their speech is often unintelligible. The only form of communication is frequently via a point board. Such a communication barrier makes education, and psychological development in general, very difficult. The lack of communication resulting from dysarthric speech can be seen as one of the major handicaps in CP. But what is known about the physiological mechanisms underlying control of speech? What goes wrong with these mechanisms in CP? A review of speech science literature showed that the prevailing explanations of the cause of dysarthric speech in CP could be classified into five different theories: weakness of speech muscles, rigidospasticity of speech muscles, release of primitive and pathological reflexes involving speech muscles, imbalance of approach and avoidance reflexes influencing the speech mechanism, and disruption of speech by involuntary contractions of the speech muscles.

We decided to use hook-wire electrodes to record EMG activity from lip, tongue and jaw muscles during a variety of speech and non-speech tasks in both normal and CP subjects. The CP subjects involved in this study all had severely dysarthric speech. The EMG activity from up to nineteen lip, tongue and jaw muscles was recorded simultaneously onto a 14-track FM tape recorder, providing a library of tapes.

Many important findings have come from this data, but the result we found first, and perhaps one of the most important, was the discovery that TSR responses are not present in lip and tongue muscles, although clearly present in jaw muscles. This was true both in normal subjects and CPs, which implied that rigidospasticity cannot be present in lip and tongue muscles, and therefore, contrary to the prevailing view, rigidospasticity of lip and tongue muscles cannot be responsible for the dysarthric speech of the CP subjects. This challenges the notion that lack of motor control in CP is a consequence of a competition between reflexes and a relatively normal voluntary control system. *It would appear that one cannot attribute the lack of intelligible speech in CP to the presence of rigidospasticity in speech articulator muscles.* This of course, is an amazing conclusion, especially in the light of speech therapy aimed at CP, which often includes many hours of oro-facial desensitisation in an attempt to reduce the spasticity of these muscles.

The subjects were nevertheless grossly abnormal. This indicated that the abnormal contraction patterns of muscles in CP are not necessarily produced by abnormal TSR behaviour, and indicated an abnormality in the central mechanisms responsible for

programming motor commands. This result is most important, because it clearly challenges the notion that lack of movement control in CP is the result of a competition between a relatively normal voluntary control system and an abnormal reflex control system. *This result was the first clear pointer to the conclusion that the primary movement disability in CP results, not from reflex abnormality per se, but from abnormalities in the voluntary control system itself.*

This conclusion is consistent with results from the action reflex training programme discussed earlier. After many months of training, our CPs were able to reduce spasm and rigidospasticity of the elbow and their limbs felt loose. There was no dramatic improvement in functional control of movement as assessed by visual tracking tests. Movement control was poor and remained poor, despite reduction in spasm and rigidospasticity. So, although spasm and rigidospasticity are important symptoms of CP, they are not the symptoms most responsible for the movement disability of CP. This is not to say that treatment of rigidospasticity is unimportant; voluntary movements cannot be made while muscles are in spasm, and consequently reduction of spasm and rigidospasticity is a necessary step towards improving functional control of movement, but it is not sufficient. *Even if problems of spasm and rigidospasticity were eliminated, CPs would remain seriously disabled, because of an inability to transform desired perceptual goals into appropriate motor commands to achieve them.*

Detailed examination of the EMGs recorded from lip, tongue and jaw muscles in the CP patients during speech, showed that each CP had an idiosyncratic abnormal pattern of muscle activity. There were, however, a number of features in common: they all used an excessive number of inappropriate muscles, the muscles contracted to excessive levels and the contractions were prolonged compared to normal subjects, but the details of the coordination of the muscles, the timing patterns of muscle contractions were quite different from one CP to the next. Nevertheless, even in athetoid CP subjects who display pronounced involuntary movement, it was found that each individual produced the same abnormal pattern of muscle contraction every time he repeated a particular syllable.

The results of these studies provided evidence that the primary cause of the motor defect in CP was an inability of the damaged nervous system to programme the appropriate sequence of motor commands to produce a desired movement.

*It was time to switch all attention to the brain mechanisms involved in programming voluntary movement.* We studied the literature relevant to voluntary control of movement in the physiological, psychological, behavioural, sports science, work load and ergonomic fields, and in the process have collected a library of reprints which are now stored at The Spastic Centre Research Unit. We decided that pursuit tracking provided the most powerful experimental method of investigating voluntary control of movement. We already had some experience with visual pursuit tracking, and developed computer programmes for analysis of such data. In visual pursuit tracking, the subject operates a control in order to move a visual response marker on a display screen, and attempts to keep the response marker aligned with a continuously moving target or stimulus marker also on the display screen. This test has the advantage that the experimenter knows exactly the movement of the control that the subject is trying to produce, and also has a precise measurement of the movement actually produced, and so is in a position to assess the accuracy and speed and appropriateness of the voluntary movement responses. From our own tracking experiments, and studies of the voluntary movement research literature, we knew some things about the behaviour of the voluntary control system. We knew, for example, that the maximum speed at which one can track a moving target is considerably less than the maximum speed at which one can move the limb. In other words, while the maximum speed for voluntary movement is limited by the mechanical properties of the limb and the maximum forces that can be generated by the muscles, the maximum speed at which one can track a moving target is limited by the time taken by the nervous system to analyse sensory information and programme an appropriate motor response. The maximum speed of tracking is less than 2 Hz, while the maximum frequency at which one can move the elbow, for example, is 4-6 Hz. We also knew that the voluntary control system updates movement programmes intermittently, in the sense that a reaction time interval is required to detect input and initiate an appropriate motor response.

The concept of intermittent motor programming is of fundamental importance to an understanding of brain mechanisms involved in voluntary movement control.

*In essence, it implies that central mechanisms require a finite interval of time (related to reaction time) to analyse sensory information and formulate a desired movement response.*

Once the desired response has been programmed, it is transferred to other parts of the brain concerned with response execution, thereby freeing the central mechanisms concerned with movement programming, enabling them to commence work on programming the next response. Thus, while muscles perform continuous contractions during the execution of movements, the underlying sequences of motor commands prepared by the central motor programming mechanisms are updated only intermittently and, once formulated, the motor programmes are executed in an open loop fashion; that is, they are performed, for at least a reaction time interval, without being modified by feedback from the evolving response.

A range of evidence exists in support of intermittency in movement programming, including our own work showing intermittent

updating of correction responses, following unexpected changes in the sensitivity of the control in a visual pursuit-tracking task. The most compelling evidence for intermittency comes, however, from double stimulus reaction time tasks and studies of the so-called psychological refractory period. It is as if a central mechanism is busy programming the first response, and consequently the second stimulus has to be held in short-term memory until the first response is programmed.

Thus we have a picture of voluntary control, in which the central nervous system requires a reaction time interval of about two hundred milliseconds to analyse sensory information and programme an appropriate response. While the response is being performed, central mechanisms can analyse sensory feedback from the movement, detect errors and, if necessary, programme a correction response. Consequently, movement programmes can be updated intermittently at reaction time rates.

Equally important to an understanding of the brain's movement control systems, is the observation that voluntary control is highly adaptable. A newborn baby has to learn to move, and there is a natural time course for development of motor skill.

*This implies that the voluntary control system is a learning machine.* This learning ability underlies the voluntary control system's adaptability. For example, a child can continue to perform complex skilled movements despite changes due to body growth. The nervous system is able to compensate for changes in mechanical loads applied to the limbs. For example, one can move an arm and point to objects in three dimensions, then pick up a heavy weight and move the arm in exactly the same way, compensating for the heavy weight. One can put on a pair of heavy boots and walk just as well as without heavy boots, even though completely different muscle contraction patterns are required. The voluntary system can learn the characteristics of external systems, so we can learn to operate tools, drive cars and sail boats, and so on. Moreover, we can rapidly compensate for changes in the characteristics of these external systems. The voluntary control system can also adapt for a variety of surgical changes. For example, the nervous system can learn to control movements following surgical relocation of muscles and nerves. The nervous system can often recover function following damage to descending tracts in the spinal cord, and even following damage to the motor cortex itself. The wearing of prism glasses, which invert images, at first disturbs movement control, but after a week or two, adaptation occurs and the subject sees images normally and can control movement skillfully. Clearly, adaptability is a key feature of the voluntary control system.

Apart from intermittency and adaptability, one other factor strongly influenced our thinking about the nature of the movement programming deficit in CP. After many years of studying motor ability and motor disability in CP, Beatrice Le Gay Breton, a psychologist at The Spastic Centre of New South Wales, presented the following insight into the mechanism of motor disability of CP at a Spastic Centre Conference.

*She proposed that a CP child may have difficulty in performing a task, such as drawing a square, because, although he appreciates the task involved and could readily find a similar square, he is unable to transfer this appreciation into appropriate movement.*

She suggested that the difficulty experienced by a CP in performing a voluntary movement is analogous with the confusion experienced by an able bodied person in drawing a square when the hand and pencil can only be seen reflected in a mirror.

Based on the above observations, we proposed the following Sensory-Motor (S-M) Model Theory of voluntary movement control. Volitional movement is programmed at a cortical level in terms of the desired perceptual consequences of the movement and transformed in subcortical circuits into an appropriate input to the motor cortex to produce the movement. The transformation of desired perceptual consequences into appropriate motor commands is based on previous learning of the relationships between central motor activity and the resulting perceptual consequences of the movement. In other words, the nervous system continuously monitors its own motor commands, and computes and stores internal models of the relationships between motor commands and their perceptual consequences. These internal models are used during voluntary movement to transform desired perceptual consequences into appropriate motor commands to achieve them. The continuous updating of internal models enables the voluntary system to adapt and compensate for changes in muscle control systems, limb mechanics, and/or external systems.

We suggest that periventricular lesions in the deep white matter of the brain, produced by ischemic and haemorrhagic lesions in the newborn CP, prevent the nervous system from monitoring its own motor activity and therefore prevents the learning of internal sensory-motor learning essential for transforming movements planned in terms of desired perceptual consequences into appropriate motor activity. Thus a CP might know precisely the movement he wants to make, but his nervous system is unable to transform this appreciation into appropriate motor commands, just as suggested by Le Gay Breton.

Pursuit tracking tests and the computer analysis technique developed at our laboratory provided an experimental approach for studying adaptability of voluntary motor control in CP. We first investigated tracking performance in a group of spastic and a group of athetoid adult CPs and found that they moved only at low speeds, had long time delays, and produced a large amount of movement unrelated to the motion of the target. An interesting observation was that although this residual movement might

be regarded as involuntary movement, and certainly included the involuntary movements of athetosis, the spastic group produced a larger amount of residual movement than did the athetoid group. This led us to the notion of 'inappropriate voluntary movement' rather than 'involuntary movement'. Spastic CPs do not display involuntary movement when they are at rest, but when they attempt to make a voluntary movement, the contraction patterns of their muscles are inappropriate and the voluntary movement is disrupted. This distinction between involuntary movement and inappropriate voluntary movement we consider to be important.

When an operator first begins visual tracking, let us say he uses a joystick to control the position of a response marker on a display screen, and tries to drive the response marker so it will track or follow a target marker, he must first calibrate himself. That is, he must learn the relationship between movement of the joystick and the resulting movement of the response marker on the screen. According to our S-M Model Theory of movement control, this calibration procedure represents the CNS learning an internal model of the relationship between movement of the joystick and the resulting visual deflection of the response marker on the display

Visual tracking experiments with normal subjects demonstrated that acquisition of skill is represented, to a large extent, by the subject learning the relationship between movement of the control and the resulting deflection of the response marker.

We investigated how quickly normal subjects could adapt following sudden changes in the relationship between movement of the control and the resulting movement of the response marker. Normal subjects tracked sudden steps of the target from one position on the screen to another. After practice, sensitivity of the joystick was suddenly and unexpectedly increased, so the same movement of the joystick produced a bigger movement of the response marker on the screen. We found that after the target jumped, there followed a reaction time interval, and then the subject initiated a fast movement of the joystick appropriate for the old joystick sensitivity. Consequently, the response marker would overshoot the target. A second reaction time interval would follow before a new response was initiated which, 70 per cent of the time, was appropriately adjusted for the increased sensitivity of the joystick.

*In other words, subjects could detect a change in sensitivity of the joystick and adaptively adjust their internal model within one or two reaction time intervals.*

Generally speaking, we found that the operator adjusts his characteristics so he behaves like the inverse or reciprocal of the characteristics of the system he is controlling; in engineering terms, he behaves like an inverse model of the external system. The nervous system learns the relationship between the kinaesthetic and visual sensory information by monitoring feedback during the movement, and then uses the information in programming succeeding responses.

If we assume that the same neurological mechanisms involved in learning to control an external system (such as a visual tracking system), are involved in learning to control the movement of one's own body and in adapting for growth and for changes in mechanical loads on limbs, then we can develop a theoretical view concerning the brain mechanisms involved in programming voluntary movement and what goes wrong with those mechanisms in cerebral palsy. One can argue that in order for the nervous system to learn to control the movements of the body's mechanical systems, it must establish an internal model of the relationships between the tensions generated in muscles and the resulting movements of the body parts. These relationships include reaction forces due to the body's inertia, visco-elastic properties, Coriolis forces and the effects of gravity. Just as the nervous system can establish internal models of the relationships between kinaesthetic and visual information representing the relationships between movement of the control and the resulting deflection of the response marker in a visual tracking test, it can establish internal models of the relationships between proprioceptive information describing the tensions generated in the muscles, and proprioceptive information describing the resulting movements of the body parts. Furthermore, the nervous system can also establish internal models of the relationships between central motor activity in the motor cortex and the resulting tensions generated in the muscles. In other words, just as the nervous system can learn the inverse characteristics of the external system in a visual tracking task, it can learn the inverse characteristics of the body's biomechanics and of the body's neuromuscular control systems. The same adaptive mechanism involved in learning to control an external system is involved in learning to control movements of the body. This view accounts for the nervous system's ability to compensate for changes in mechanical loads on limbs, for changes due to growth, surgical relocation of muscles and nerves, and damage within the nervous system itself.

The Sensory-Motor Model Theory of voluntary movement control can be summarised as follows: Movement is programmed initially at a cortical level in terms of the desired perceptual consequences of the movement. It is then transformed at a sub-cortical level, via an internal model of the external system, into desired body movement as represented by sensory information detected by joint receptors, muscle spindles and skin receptors. The desired body movement information is then transformed by an internal model of the body's biomechanical system into the desired pattern of muscle tensions, as detected by Golgi tendon organs, required to produce such a movement. The desired pattern of muscle tensions is then transformed via an internal model

of the body's neuromuscular control systems into appropriate motor cortex neural activity to generate the desired muscular tensions and produce the desired movement. The nervous system must learn the internal models (this learning represents acquisition of motor skill) and maintain their accuracy by monitoring sensory feedback of the motor commands, muscle tensions, body movements and exteroceptive consequences and by computing and updating the internal models of the relationships between these sensory signals. Furthermore, it is proposed that the movement programming defect in CP is consistent with the notion that brain damage in CP disrupts internal feedback of motor commands, thereby preventing the nervous system from forming an accurate internal model of the body's neuromuscular control systems. Consequently, during movement programming, the damaged nervous system experiences difficulty in translating desired muscle tensions into central motor activity capable of producing those muscle tensions. As a result, the nervous system generates inappropriate motor activity and this constitutes the primary disability of cerebral palsy.


The notion of the motor programming deficit in CP being a motor learning defect, caused by disruption of internal feedback of motor commands preventing the nervous system from establishing an accurate internal model of the body's neuromuscular control systems, is consistent with the observation that diagnosis of cerebral palsy only becomes possible as the CP falls further and further behind in normal motor development and fails to reach various motor milestones. Clearly, the movement deficiency in CP is an expression of a motor learning defect. CPs know exactly what they want to do, they know what movements they need to make to achieve the goal, and they can compute the muscle tensions required to achieve those movements, but they cannot compute the central motor cortex activity needed to produce the muscular tensions required to produce the movement. In other words, they have a breakdown of voluntary control at the level of learning the relationships between central neural activity in the motor cortex and the resulting tensions generated by the muscles.

All of this of course is speculation; a hypothesis is to be tested experimentally. If it is confirmed, it will represent an important new direction in our understanding of the mechanisms responsible for the motor programming disabilities of cerebral palsy. *It will shift the emphasis of therapy away from spasm, rigidospasticity and involuntary movement and focus it on motor learning and lack of motor adaptability as the primary cause of disability in cerebral palsy.*

A neuroanatomical picture, relevant to understanding the mechanisms of cerebral palsy, is beginning to appear in the neonatal intensive care and neurodevelopmental research literature. This picture is consistent with the Sensory Motor Model Theory presented above. In the developing brain there is an area of brain tissue which lies at the boundary between two different blood supply circuits; one blood supply circuit originates on the surface of the brain and radiates inward. If the developing brain is deprived of oxygen in some way, say by haemorrhage or occlusion of the blood supply vessels, so that there is a massive drop in the amount of oxygen reaching the brain, this vulnerable area of the brain at the boundary of these two blood supply systems is the region most likely to be damaged. This region is deep in the white matter of the brain, and is referred to as the periventricular white matter. It includes those nerve fibres which pass through the internal capsule of the brain on their way to the spinal cord. Brain sections at autopsy and brain scans of babies with brain damage have shown a patchy mottling degeneration within this white matter, which is referred to as leukomalacia – the sort of lesions which commonly occur when babies survive an anoxic insult of the developing brain. The blood vessels in this region of the developing brain are particularly thin and are vulnerable to damage in premature infants. Haemorrhage in this region is common and bleeding often extends into the ventricles.

The idea that the lesions responsible for motor disorder in CP are located in deep white matter in the internal capsule is consistent with the traditional explanation of cerebral palsy; that is, lesions disrupt the nerve fibres which transmit motor impulses from the brain to the muscles. Hemiplegic spasticity due to stroke is commonly associated with haemorrhage into the internal capsule and damage to the descending motor fibres, but CP is only superficially similar to stroke hemiplegia and the muscle contraction patterns observed in CP indicate a motor programming defect not observed in stroke patients.

Recent reviews of the neuroanatomy and neurophysiology of the deep structures in the brain known as the basal ganglia, thalamus, and cerebellum, in the nervous system section of the recently revised Handbook of Physiology, indicate that movement programming involves subcortical circuits which project from the cortex through the basal ganglia and cerebellum and back to the motor cortex. These circuits involve multiple pathways concerned with control of different body parts. The multiple pathways through the basal ganglia are not concerned with control of the forces generated by muscles per se, but rather are involved in specifying the parameters of limb movement, the speed and direction of movement of the limb as a whole. This is consistent with the notion that the subcortical circuit through the basal ganglia behave like an internal model of the external system which transforms desired perceptual goals into a neural code representing the body movements required to produce those perceptual consequences. For example, in a visual tracking task, the basal ganglia would transform the desired visual displacement of the response marker into the hand movement required to operate the joystick. Next, the information flows through the subcortical loops through the cerebellum and back to the motor cortex via the thalamus. In these subcortical loops, the desired body movements are transformed into the required patterns of tensions to be developed by the muscles and then the desired patterns



of tensions are transformed into the appropriate central neural motor activity (central motor commands) to activate the neuromuscular control systems. In other words, the subcortical loops through the cerebellum, behave like internal models of the body's biomechanics and neuromuscular control systems. The transformations involved in these loops represent learned relationships between perceptual consequences of movement, kinaesthetic sensations of movement, patterns of tensions developed by muscles, and central motor cortex neural activity. All of these transformations involve flow of neural signals from the cortex through the basal ganglia back via the thalamus to the pre-motor cortex, to the cerebellum, and back via the thalamus to the motor cortex. In other words, as best as we can tell from neurophysiological and neuroanatomical research, the nerve fibre pathways involved in transforming desired perceptual consequences of a movement into appropriate motor cortex activity to produce the movement, include the very pathways in the brain which are most vulnerable to damage by lack of oxygen in the developing brain.

The location of brain lesions in CP determined in one field of research, and the function of the neural circuits in which those lesions are located from another field of research, are consistent with the nature of the motor learning disability observed in CP in yet another branch of research.

*Thus the pieces of the jigsaw of CP so far assembled, suggest that the primary motor disability in cerebral palsy results from haemorrhage or ischemic lesions in the deep periventricular white matter of the developing brain.*

This white matter includes nerve fibres involved in the subcortical circuits through the basal ganglia and cerebellum involved in transforming desired perceptual consequences of a movement into appropriate motor commands to produce the movement. As a result, a CP can appreciate precisely the task he wants to perform, but is unable to translate this appreciation into appropriate motor commands to perform the task, just as suggested by Le Gay Brereton. As a consequence, the nervous system generates inappropriate motor commands, which are communicated to muscle control systems through intact descending pathways producing the powerful, but inappropriate, muscle contractions typical of CP.

*Notice, this view differs from the traditional explanation of CP, which asserts that motor commands are disrupted by lesions in the descending pathways from the motor cortex to the muscles.*

According to this new point of view, lesions in the periventricular white matter disrupt internal feedback of motor commands. Although the motor learning mechanisms receive sensory feedback from muscles, tendons, joints, skin, etc., it has no feedback of the motor commands which produce volitional movement, and so it is unable to learn the characteristics of its own neuromuscular control systems. The damaged nervous system can compute the muscular tensions required to produce a desired movement, but is unable to translate these desired tensions into the appropriate central motor activity needed to produce the movement. The inappropriate activity, which is formed by some compensatory mechanism, leads to inappropriate contractions of muscles and inappropriate reflex behaviour. Notice that this view implies that CPs have difficulty in controlling the tensions of individual muscles, rather than in computing the appropriate patterns of tensions to be developed across groups of muscles. In other words, strictly speaking it is not a problem of muscles coordination per se, but a problem in controlling the individual muscles within a coordinated pattern.

If these experiments, which will require some years to complete, are successful, we will have provided experimental evidence in support of the notion that the primary motor programming defect in CP results from disruption by brain lesions of internal feedback of motor commands, thereby preventing the nervous system from establishing internal models of the body's own neuromuscular control systems. We will also have obtained learning curves demonstrating the extent to which improvement in functional control of muscle contraction level can be obtained by appropriate EMG tracking training.

*These data will then form the experimental basis for establishing new therapy techniques, aimed not only at reduction of spasm, rigidospasticity and involuntary movement, and prevention of development of deformities, but also continuing that training to acquire improved functional control of muscles.*

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Una Holibone



A suit of Samurai armour presented to The Spastic Centre by Dr S. Yamada of Okinawa.