THE BOBATH CONCEPT IN THE TREATMENT OF PATIENTS WITH BRAIN DAMAGE[1]

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Dr. and Mrs. Bobath have worked mainly with patients suffering from cerebral palsy, and with those who are hemiplegic as a result of a cerebro-vascular accident. However, their methods of treatment have been shown to be effective with a number of other disorders of brain function involving the sensorimotor system. The treatment they have developed is directed towards disorders of movement and disorders of tone.

The Bobaths believe that there is, in the brain-damaged patient, a derangement of the normal postural reflex mechanism, as well as abnormal tone which results from the lesion. This postural reflex mechanism comprises the automatic reactions of equilibrium, righting and protection, and requires for its normal function, normal tone. These motor responses are acquired and gradually develop during childhood.

"The righting reactions are automatic movements which serve to maintain or restore the normal position of the head in space and its normal relationship with the body, plus the normal alignment of the limbs and trunk. These are modified and gradually disappear towards the end of the child's fifth year, but they are essential in the building up of motor patterns for adult life. The patterns of these reactions are seen in such movements as rolling over, getting up from supine or prone, and standing up. The equilibrium reactions serve to maintain and restore balance during all activities, especially when we are in danger of falling.

"These postural adjustments to changes in the centre of gravity are continuous while we move, and even the smallest change in equilibrium has to be countered by a change in tone. These adaptive changes in muscle tone are protective against the force of gravity. Postural adjustments may be so small they cannot be seen — they may consist only of tonus changes." Others may be quite dramatic. Protective extension of the arm, for example, is an essential automatic reaction. It serves to protect the face from injury when we are in danger of falling, and many fractures of the arm bear witness to its effectiveness.

"All our movements are in response to sensory stimuli, which act upon the central nervous system from the outside world through the exteroceptors, vision, touch and hearing. These sensory messages are integrated at cortical level and produce a co-ordinated motor response in keeping with the environment. Movements are guided throughout their course by vision and by sensory cues from the proprioceptors of muscles, tendons and joints, that is, by a sensory feedback system. All learning is through sensation and we not so much learn a *movement* but the *sensation* of that movement." When a patient has a disorder of movement resulting from brain damage, it is recognized that he has therefore a sensorimotor disorder, rather than a purely motor disorder.

To understand the Bobaths' concept we must appreciate the automatic nature of all movement. "Motor activity" has been described as ranging from most automatic to least automatic. This suggests no fundamental difference between automatic and voluntary movement.

We consider posture to be dynamic rather than static. After all, movements are only changes of posture. The physiologist, Sherrington, commented that "posture follows movement like a shadow". Automatic postural reac-

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tions underlie all functional behaviour. They form the background to movement, and unless they are fully developed, which they can only be in an intact, mature brain, the full range of normal activities is impossible. The Bobaths' treatment is not at all static. The therapist aims at giving the patient the ability to make the postural adjustments which will enable him to move more normally, and in a less stereotyped manner.

Though patients with brain damage rarely demonstrate the same picture of sensorimotor disorder, there are certain features which they all have in common. They all show poor or absent equilibrium and righting reactions, asymmetry, lack of trunk rotation, poor selective control over the limbs, as well as abnormal patterns of posture and movement, and abnormal tone. One never finds spasticity in a single muscle, but always in groups of muscles forming a particular pattern. Increased tone is found in the hemiplegic patient, for example, in fairly predictable patterns; flexion, retraction, internal rotation of the upper limb, and extension, internal rotation and plantarflexion of the lower limb. This means that the trunk itself will be involved through the spasticity of groups of muscles attached from the pelvic and shoulder girdles to the limbs. This results in retraction and side flexion of the affected side, and adds to the inability of the trunk as a whole to rotate. This lack of rotation around the body axis is an important contributory factor in the patient's inability to move effectively.

It is difficult and sometimes impossible for the spastic patient to move, and when he does so, he can only move in these stereotyped patterns. It is the ability to vary the way in which we move which differentiates the neurologically normal person from the neurologically abnormal. In the spastic hemiplegic patient, for example, there is no longer the background of normal righting and equilibrium, so he cannot do such basic activities as rolling over, or getting up from a chair. When he falls to the affected side he cannot put out his hand to protect himself, because the flexor pattern of his spasticity prevents him from doing so. He cannot transfer weight or right his head in relation to his trunk. He cannot use even his so-called normal side normally, illustrating for us the fact that one side of the body cannot function effectively alone. Gradually the sound side begins to compensate in a manner which is itself abnormal. When he learns to sit up, to stand and to walk, he does so by compensating with the postural reactions of the sound side. He will not initiate any of these movements with the affected side. He cannot support himself on the affected arm, and he will take as little weight as possible on the affected leg.

It is not difficult to imagine what the sensory feedback from all this abnormality will be like. It will be a feedback of abnormal tone and abnormal patterns of movement. This will result eventually in a distorted body image. We feel normal even if we are not, and the spastic hemiplegic patient will eventually feel normal too.

Let us consider why spasticity interferes with movement. The excessive contraction of spastic muscles produces a relative inhibition of the potential activity of their antagonists. This makes the antagonists appear weak. An example can be seen in the apparent weakness of the triceps brachii in a patient with flexor spasticity of the arm. The movement of extension appears weak or even paralysed when the patient tries to do the movement sitting with his arm by his side. But when the flexor spasticity is inhibited, with the arm externally rotated and protracted, he may be able to straighten his elbow and take weight on the hand, indicating that the triceps are inhibited rather than weak. Similarly, when he tries to elevate his arm, he must do so against the spastic resistance of the flexors of the arm, and the retractors and depressors of the shoulder girdle. On an attempt to raise the arm, the inferior angle of the scapula is fixed by the spastic rhomboids and trapezius, and if the arm is raised passively the necessary external rotation of the scapula and therefore elevation of the acromion does not take place, the humerus is pressed against the acromion and the patient complains of pain. The patient's problems do not arise out of weakness as such, but rather from an inability to coordinate the contraction and relaxation of groups of muscles.

Tonic neck and labyrinthine activity may be demonstrated in brain-damaged patients, and is due to the release of phylogenetically

older brain stem reflexes from control of higher centres in the brain. These tonic reflexes are activated by changes in the position of the head in space and their influence produces predictable changes in the strength and distribution of spasticity throughout the body. A positive tonic labyrinthine reflex will result in an increase in extensor tone when the patient is in supine, and an increase in flexor tone when he is in prone. With a positive asymmetrical tonic neck reflex the patient will have difficulty flexing the arm and leg on the side to which the face is turned, and corresponding difficulty extending the arm and leg on the occiput side. This abnormal reflex activity must be inhibited before normal movement can be learnt.

Other forms of treatment have stressed the importance of strengthening weak muscles and building up of activity on the unaffected side in the case of the hemiplegic patient. The Bobaths have shown that effort and concentration by spastic patients will result in associated reactions, that is, an increase in tone in the spastic muscle groups. This can be seen if you ask the patient to squeeze your hand with his unaffected hand. The hemiplegic arm shows a build-up of flexor tone and the arm moves further into the abnormal flexor pattern.

Recovery and the result of treatment will depend to a large extent on the degree of sensory involvement. Apart from the problem of abnormal sensory feedback, from which all these patients will suffer, there may also be varying degrees of damage to the sensory system itself. The influence of sensory disturbances on the ability to initiate and perform normal movement is profound. Patients with a severe sensory deficit lack the urge to move, often in spite of only moderate spasticity. The hemiplegic patient may say his hand feels as though it is encased in a glove, and he is unlikely to use this hand because of lack of sensory drive. Patients with damage to the parietal lobe may have a disorder of perception which results in such a severe defect in motor planning that the patient, because he does not know how to plan his movements, may be unable to move effectively at all.

Before we start treatment of the patient with brain damage, an initial and very thorough assessment is made. We do a qualitative assessment, trying to find out not only what the patient can do in terms of movement, but *how* he does these basic movements. We compare his abnormal movement patterns with the normal movement patterns of a person with an intact nervous system. We assess the distribution of abnormal tone. This analysis gives us the information we need in order to plan treatment. It demonstrates to us what normal patterns have to be obtained and what spastic patterns have to be inhibited in order that the patient may develop more normal motor behaviour.

In treatment the Bobaths do not aim to relax and strengthen certain muscles, but rather to improve co-ordination of posture and movement, and to obtain more normal tone. They set out to change the tone — to increase it in the flaccid patient; to lower it in the spastic; to regulate it in the athetoid, who may have intermittent tonic spasms. Once tone is regulated, more normal movement is possible with less effort. Emphasis is placed on teaching the patient to *feel* a normal movement, remembering that repetition of normal patterns of movement will strengthen these patterns.

Treatment is directed not to one limb but to the whole patient, otherwise it is found that the desired response may be gained in one limb at the expense of greater spasticity in another. Treatment is directed basically in two ways. We attempt to inhibit hypertonus by influencing the central nervous system from the periphery. At the same time, we facilitate a background of more normal automatic reactions, that is, righting and equilibrium. These reactions, with their essential elements of protective extension of the arm and weight transference, are facilitated as the basic activities necessary for specific functions such as gait. Gait has been described as a constant losing and regaining of balance. Without the ability to transfer weight forwards and sideways, to rotate the trunk within the body axis, to weight-bear on a mobile controlled knee, we cannot walk effectively. Therefore it is only by inhibiting the abnormal tone which does not allow the patient to do these movements and by retraining these various aspects of gait, that we can hope to

improve the patient's gait. He must learn to adjust himself to changes in his centre of gravity.

The Bobaths stress the importance of treatment begun early, before spasticity is established and before the abnormal patterns of movement have become too much a part of the patient. In the case of the hemiplegic patient, there seems more possibility of preventing rejection of the affected side if weight transference to this side is encouraged early. So often we are asked to treat the patient when he has lost the urge to move and become reconciled to his status. With the hemiplegic patient, attempts to retrain the sound arm to substitute for the affected arm are discouraged in the early stages, so as much relearning as possible can take place with the affected arm. We concentrate on early weight-bearing with this arm, as this prepares the patient for reaching out to grasp objects and for stretching out automatically for protection.

This method of treatment would be best carried out if the patient were nursed in a total care situation; where the nursing staff as well as the medical officers and therapists understand all the problems involved; where the nursing staff are trained in positioning the patient to minimize the development of spasticity, and in handling the patient with speech and feeding problems; where the speech, occupational and physical therapists are all trained in this neuro-developmental method of treatment. The patient should be nursed in a unit where he can stay until relatives can be trained to take over some of the treatment, or until he can look after himself. So often, especially with the older hemiplegic patient, treatment may start early and be effective, but the patient is discharged prematurely to a nursing home, where no further treatment is given and where he spends the rest of his life in front of a television screen.

Any approach to treatment must necessarily be limited by the patient's potential — by the capacity of his injured brain to be re-organised to produce more normal function. At the moment many approaches to treatment still do not make the most of the apparent adaptability of the brain, an adaptability particularly evident in the immature brain, and many patients are rehabilitated by methods which aim at strengthening muscles which may not be weak, and at mobilising joints which may not be stiff, and are discharged without ever having a chance to develop what is left of their damaged brains.