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The Institute for Advanced Neuromotor Rehabilitation

Registered Charity: 1036049 - formerly the Hyperbaric Oxygen Trust

18 May 2004.

TO WHOM IT MAY CONCERN

I am at present in the final stages of writing up a PhD thesis supported by University College London Psychology Department in which evidence is provided suggesting that the metabolic assault of brain injury weakens the diaphragm muscle and so reduces the development of the strength and responsiveness of the respiratory system.

I am also the Clinical Director of the Charity Advance. In this capacity I was treating Conor Mitchell by a home exercise programme taught in stages to his mother and based upon the body of knowledge acquired over in the course of my research. The treatment approach is also being independently evaluated as the subject of a separate PhD by Bradford University Health Science Department.

- The strategy called Neuro Respiratory Therapy (NRT) focuses on developing core muscle strength and reducing superficial muscle strength. In this way it pararells the changes seen in the developing infant over its first year of life which the thesis maintains are brought about by the steady increases in the infant's respiratory strength.

- Progress is achieved by working directly on identifiably weak respiratory muscles, most importantly the diaphragm, the weakness of which is evident from the CP child's structure.

- The approach has involved making close observations of the children attending the Charity's centre taking their trunkal measurements and the identification of variations in regional blood flow through the brain, lungs and liver with Reography. (This is a non-invasive method of investigating organ blood flow supply based on observations of tissue electrical resistance).

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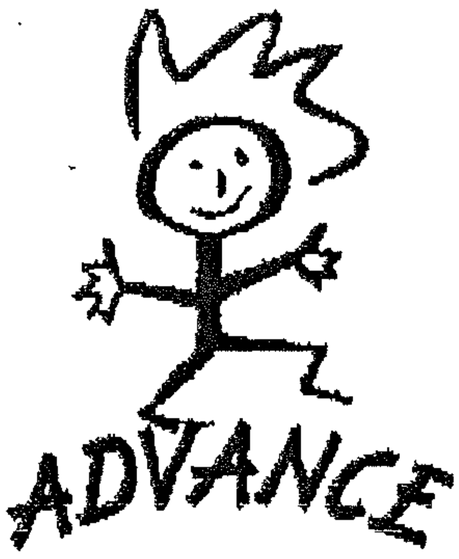

- As a result it appears that the existence of respiratory weakness may cause children with CP to respond rather differently from normal children when they are exposed to both external and internal influences causing metabolic stresses. These indications may throw more light on Conor's reactions in the last 2 days of his life.

To proceed adequately I need also to explain a little more of the probable causes of respiratory weakness in CP and then the possible metabolic consequences.

1. It is known that the metabolic stress of cerebral assault directs blood supply away from the muscles to the cerebral metabolism. This is particularly concerning for diaphragm muscle development since the diaphragm is the central respiratory muscle which is pivotal for the development of a normal respiratory system. There is reasonable evidence to suppose that respiratory system capability sets the level of homeostatic balance for the organism since evidence suggests it is responsible for levels of oxygen availability in the micro circulation of the body tissues.

2. During cerebral assault the diaphragm also appears to work harder to respond to increased demands by the cerebral metabolism for the period of the acute phase of after the injury.

These two factors probably combine to produce tissue fatigue by reducing diaphragm capillary blood flow thus leaving some of the diaphragm's capillary beds dysfunctional or weakened. As a result both the strength of the respiratory excursions and the diaphragm's responsiveness to nerve signals appear reduced.

Children coming to our centre all demonstrate abnormalities of trunkal structure consistent with this thesis as well as steady improvement with ongoing treatment.

From a physiological point of view it follows that diaphragm weakness would affect the muscles involved in the abdominal thoracic pump which pulls the venous blood (the de-oxygenated blood) back to the heart. Reography observations and the study of our children's metabolic responses during limited stress suggest that in the case of children with C.P. there is a back up of venous blood to the detriment of capillary blood flow availability. This has a direct effect on venous outflow from the

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cerebral metabolism. The volume of blood in the cerebral metabolism remains constant. However, arterial inflow is known to be limited by venous outflow. This would mean that the children with C.P. would be more likely in this less stable situation to experience an increase in inter cranial pressure or a reduction of available oxygen within the cerebral metabolism as a result of over-hydration or two zealous rehydration.

Reography readings of CP children also suggest that in many tissue areas capillary beds can be constricted or spastic so that a reduction in the efficiency of the venous return would also mean that the legs and body tissues could not be so available as a fluid reservoir during over hydration. It is more likely that there would be an increase in inter cranial pressure because the brain is encased in bone as a result of a backup of the fluids before tissue swelling was evident in other parts of the body.

There are a few other points to make with reference to the homeostatic balances that may be said to be dependent upon the respiratory system. We have noticed responses in our children that suggest that when tissue is active and therefore requiring blood oxygen the poor distribution of capillary blood supply tends to create imbalances in the system. Whereas the active tissues needs were very likely to be appropriately met this would be to the detriment of the other tissues. For example if a virus were to produce certain metabolic demands in the area of the affected tissues then the child with C.P. would be more likely to suffer from symptoms associated with a more severe level of the condition. For example, a reduction in blood supply to muscle tissue can produce muscle spasms; a reduction in blood supply to surface tissue can produce very pale skin; a reduction in blood supply to the digestive system can produce a poor ability to digest food and a loss of appetite. It is also likely that children's eyes would be affected and that they would suffer much more quickly from lethargy associated with illness. The reduction in capillary blood flow therefore means a more intense reduction in other metabolic demands so that focus can be on clearing the virus out of the system.

From this point of view we often see our children manifesting for a day as if they had a quite severe complaint and the next day they're remarkably recovered. At other times the symptoms of an illness can drag on for an abnormally long time until the

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child's residual respiratory strength returns to its normal levels. (This may have been the situation in Conor's case). In normal children where the blood flow through the micro circulation is more balanced and better available these kinds of severe symptoms could easily be associated with a much more severe assault. The other factor is that quite often the treatment such as rehydration or a heavy course of antibiotics can have an adverse effect on brain injured children who are less able to deal with the effects of invasive medical approach to disease. In my opinion such children would certainly be more liable to respond adversely to a rehydration process that wasn't very carefully balanced and slow. They would also be likely to respond badly to a routine course of antibiotics because the antibiotics themselves tend to weaken the cell walls and therefore reduce the efficiency of organ function. This in itself creates further instability in an already unstable system.

So far as I know there are no special guidelines for treating children with cerebral palsy. Insufficient is perhaps known by hospital staff about the differences in metabolic response to both symptoms and the intervention prescriptions to reduce those symptoms. However in my opinion Conor's thinness is typical of CP and reflects the weaknesses of the abdominal thoracic pump I have formally described. It should have been associated with cerebral palsy rather than with a condition of severe dehydration muscle. Spasming is a fairly typical symptom of C.P. and that since he, by his mother's own account had not been entirely without fluids the muscle spasms that may have been seen certainly was not likely to be a symptom of advanced dehydration.

It's always better when the patient presenting a condition on which the medical practitioner has little knowledge to consult some authorities in that particular situation. In this case the two Authorities that needed to be consulted in some depth were the patient's own family and the Hospital for Sick Children that had some experience treating children with C.P. To presuppose that a child with C.P. could be classed as an adult simply because he was 15 years old might be generally considered to be unwise under any circumstances and hopefully in the future better guidelines will be available for medical staff in this sort of situation.
Linda Scotson.

Linda Scotson May 15th 2004.

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