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Transdural Spinal Cord Herniation: Acquired or Developmental?

In this issue of the *American Journal of Neuroradiology,* Watters et al (page 1337) and Dix et al (page 1345) beautifully illustrate a highly unusual condition in two patients presenting with progressive myelopathy in whom apparent spontaneous herniation of the spinal cord through the ventral dura mater of the thoracic spine was found. Both cases were treated successfully by surgery, and the patients improved clinically. One cannot help but be intrigued by a number of issues these cases raise, especially when exploring the underlying cause for this dural defect and the subsequent spinal cord dysfunction associated with it.

In one of the patients described by Watters (Case 1) and in the case report by Dix, a congenital or developmental defect in the dura (as opposed to an acquired dural tear resulting from surgery or spine trauma as was the situation in four of Watters' five cases) is the probable cause of the cord herniation. There is no indication in either patient history that there had been significant trauma or a previous operation, nor were there imaging findings suggestive of prior injury or surgery. Nonetheless, an occult injury in the distant past resulting in a dural tear remains a remote possibility.

If it is accepted that cord herniation and, in Dix's case, leakage of CSF forms an extradural cyst through an attenuated or gradual widening dural defect, the question remains, why is this seen almost exclusively in ventral dura of the thoracic spine? It is probably unrealistic to accept the proposal some have suggested that a prior intradural disc herniation caused a dural defect through which the cord herniated. With such a mechanism, one would expect to find not only an intradural disc fragment but a higher incidence of cord herniation in the cervical area where disc herniations are more common. In the absence of such findings, another explanation is needed. Some have proposed that a posteriorly located arachnoid cyst pushes on the cord and over time causes the cord itself to erode the dura. In the cases presented here, however, no such intradural arachnoid cyst was found by imaging or at surgery. The close approximation of the anterior surface cord to the normal curvature of the thoracic spine alone does not provide us with a good explanation of why a dural defect is present in this area.

It is reasonable to believe that cord herniation of and by itself would not cause a myelopathy. Vascular compromise, adhesions, or a focal compression of the cord provide the probable explanation of the myelopathy in these two patients. In fact one could even suggest that in Figure 1F (page 1346) in Dix's article, an area of high signal in the left side of the cord might be the MR correlate of the cord dysfunction, corresponding to the patient's Brown-Séquard syndrome.

While these cases and others like it leave many of the above-mentioned issues unanswered, the images do serve to increase our awareness of this treatable condition. One wonders how many patients with a progressive or a static myelopathy there might be who have been variously diagnosed as having a cord atrophy or intradural adhesions but who in fact have this entity of spinal cord herniation. High detail MR imaging and widespread recognition of this abnormality may help numerous patients who otherwise may go untreated.

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When Seeing Double Is Not Always Bad

Although MR has greatly enhanced the study of human brain development, there is still much to learn. Significant questions remain concerning genetic and environmental influences on brain development. While we fully acknowledge the contributions of genetic make-up on the anatomic development of the brain, we are less certain about the extent of environmental influences. If we accept that structure and function depend on one another, then a more systematic evaluation may reveal clues about the dynamic interplay between genetic and environmental influences and how both affect brain function. It is well known, for example, that structural hemispheric asymmetry exists in the brain, and hemispheric dominance depends on whether the subject is left- or right-handed. What is not known is the extent to which genetic predisposition versus environmental stimulation influences this asymmetry. We often think of the surface of the brain as a neurologic "fingerprint," though we are only beginning to understand that variations in the gyral pattern may reflect not only our genealogy but our life's experiences as well.

As with any experiment, the ability to sort differences depends on the investigator's control of the variables that might exist in a study population. In order to evaluate questions related to form and function, one must control either the genetic or environmental influences within the model. Such a model readily exists in nature in the form of monozygotic twins. By definition, monozygotic twins develop from a single fertilized ovum, and thus share identical genetic structures. Application of this model may prove useful in sorting genetic similarities from environmental differences that could potentially affect brain development.

In the current issue of the *American Journal of*

Neuroradiology, Biondi et al (page 1361) take advantage of the genetic symmetry of monozygotic twins to explore genetic similarities and environmental differences that influence brain development through the use of advanced MR. Six observers evaluated cerebral cortical surface anatomy and midline callosal morphology by 3-D surface reconstruction and quantitative morphometry, respectively, in seven pairs of monozygotic twins aged 19–47 years. The purpose of this comparison was to determine if observers could match each twin correctly with his or her monozygotic sibling by observing cortical reconstructed images portraying surface gyral anatomy. Similarities in gyral patterns suggest that these patterns are determined more by genetic than by environmental influences. Conversely, differences would suggest a greater role for the environment in shaping both anatomy and brain function. Their results revealed significant variations in secondary and tertiary surface gyral anatomy, which did not, however, prevent observers from correctly matching the twin pairs in the overwhelming majority of cases. In addition, a significant correlation also existed in the midline measurements for the volume of the corpus callosum between related siblings. What the investigators were able to conclude was that both genetic and environmental factors influence and direct brain development. If we assume a relationship exists between structure and function, their results also imply a similar, mutual role for both influences in shaping brain function as well. They also revealed statistically similar relationships of callosal volumes between twin pairs indicative of genetic similarities in brain weight for the related siblings. Differences in the surface gyral pattern may be explained in part by environmental influences; however, genetic influences clearly play a role in shaping overall brain structure. Similarities were so apparent that observers had little or no trouble matching each of the twin pairs despite differences that existed in their secondary and tertiary gyral patterns.

Their results raise significant questions about what effect learning and the environment may have on the development of the brain. We should never underestimate these influences and the role they play in overall learning, experience, and development. These questions are especially pertinent in the first decade of brain development where plasticity of the brain is at its peak. As the brain rapidly matures in the first years of life, environmental influences may impact eventual outcome most. Perhaps the brain is more like a computer than we would like to admit. The more information incorporated into the central processing unit, the greater the change in the appearance of the interface. The more information we pump into the neural circuitry of the brain as it develops, the greater the effect on and change in our behavior. This prospect alone is enough to make us wake up and take notice of our environment and its potential influence on the developing brain. Greenough et al attributed environmental effect on behavior to two forms of plasticity (1). The first they referred to as *experience-expectant,* the collective experiences of the human species. These are common influences to us all, and shape common behavior. The second they referred to as *experience-dependent,* the incorporation of information unique to the individual. Dependent influences of the environment might be expected to account for structural differences between individuals. In Biondi et al's current work, much of the morphologic effect seen in the plasticity of the brain appears to relate more to experience-dependent plasticity. When the entire process of learning is taken into consideration, it becomes obvious why the entire gyral system of our brain evolved to best receive, store and assimilate massive amounts of information. While gross anatomic changes may be evident, ultrastructural changes may also be influenced by changes in the environment. Withdrawal of environmental influences through sensory deprivation in animal models reveals a direct effect on synaptic development and neuronal function. While our genetic composition may be responsible for our capacity to learn, it is ultimately the environment that is responsible for the experience of learning.

All this science leads us to one conclusion: we cannot escape our environment or its effect. No, this is not another essay influenced by or expounding on the teachings of Dr. Spock; however, the results of Biondi et al's study do give reason to pause and contemplate the quality of our surrounding environment and any effect it may have on the developing brain. Finally, such evidence for the power of the environmental influence on brain structure should remind us not to underestimate the potential for rehabilitation of the brain, programmed environmental stimulation, to maximize genetic potential. This study does fall short of answering what effect, if any, our environment has on cognitive development, despite the unquestionable link it may have on anatomic development. Our colleagues in child psychology will be quick to remind us, however, that they have recognized this link for many years. Until the day all such questions are definitively answered, perhaps we should pay as much attention to where and how we live as to our origins.

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References

1. Greenough WT, Black JE, Wallace CS. **Experience and brain development.** In: Mark H. Johnson, ed. *Brain Development and Cognition: A Reader.* Cambridge, MA: Blackwell Scientific Publications; 1993:290–319