Reviewer's comment concerning “Abnormal activation of the motor cortical network in idiopathic scoliosis demonstrated by functional MRI” (doi:10.1007/s00586-011-1776-8) by J. Domenech et al.

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Adolescent idiopathic scoliosis (AIS) is a multifactorial disorder with more questions than answers when it comes to aetiopathogenesis. Research has focussed on six major areas: genetic factors, central nervous system (CNS) abnormalities, hormones/metabolic dysfunction, skeletal growth abnormalities, biomechanical factors and environmental/life style factors [9].

Abnormalities in the CNS have long been implicated in the aetiology of AIS. Lowe et al. [6] have suggested a defect of central control or processing by the CNS that affects the growing spine, with most studies pointing to the pontine and hindbrain regions as the most likely sites of the primary pathology. Subsequent research into CNS abnormalities maybe conveniently divided into two major groups: neuroanatomical studies and neurophysiological studies.

Neuroanatomical studies have demonstrated subgroups of AIS subjects with hindbrain abnormalities including cervicothoracic syrinx [5] and low lying cerebellar tonsils with or without abnormal cerebrospinal fluid dynamics [2]. Other workers have reported abnormalities in the midbrain, pons and medulla, and the vestibular system [4]. Shi et al. [7] compared volume-based morphometry of brain magnetic resonance images in AIS subjects and control subjects, detecting the differences in regional brain volumes, white matter in corpus callosum and internal capsule, and subsequently in vestibular system morphology [8].

Neurophysiological studies in AIS subjects have demonstrated a plethora of abnormalities in postural balance, somatosensory function equilibrium, proprioceptive

function, oculovestibular function, electromyography, somatosensory evoked potentials, transcranial magnetic stimulation [9] and asymmetric motor cortex hyperexcitability [3].

The authors further developed this theme with a novel study using functional magnetic resonance imaging (fMRI) to explore cortical activation following a simple motor task in ten subjects with AIS and ten healthy age-matched controls. Compared to the controls, the AIS patients demonstrated increased activity in the contralateral supplementary motor area while performing the motor task. The authors state that these findings support the hypothesis that a sensorimotor integration disorder underlies the pathogenesis of AIS.

Whilst the ‘abnormal pattern of brain activation’ in the AIS subjects is indeed an interesting observation, a causal relationship has not been proven; e.g. the scoliosis itself may in some way lead to the secondary changes observed within the brain. At best we can say that the brain of the AIS subjects is behaving ‘differently’ to that of the control subjects. The study numbers are understandably small and essentially constitute a pilot-study. The ten AIS subjects consisted of eight females and two males, eight right-handed and two left-handed subjects. The curves varied in magnitude (Cobb angle 27°–55°), curve progression (3 of 10 subjects were reported to have rapid progression) and type (6 right thoracic, 2 thoracolumbar, 1 left thoracic and 1 lumbar) thereby representing a broad spectrum of pathology. It is difficult to understand how a seemingly homogenous alteration in the activity of the contralateral supplementary motor area can produce such a heterogeneous pattern of curves that behave so differently, unless other factors are at work. We know that AIS is a multifactorial disorder. To unravel this puzzle one would need to perform a much larger study with stratification into female and male subjects, left- and right-handed individuals, low

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and high magnitude curves, non-progressive and rapidly progressive curves, similar curve types and see if the observations were repeatable across this larger sample.

This study is provocative in that it suggests that a sensorimotor integration disorder is the cause of AIS. It certainly throws another piece of the jigsaw into an already vastly complex puzzle. However current knowledge remains fragmented; we are still not clear if the plethora of observations reported in AIS subjects are primary or secondary effects. Detailed prospective examinations of AIS subjects in tandem with serial MRI studies would allow one to ascertain whether, for example, the observed changes in the brain are linked with the cause or merely an effect of the AIS.

Current treatment of AIS is aimed at preventing curve progression and improving cosmesis, that is the effect of AIS [1]. A true paradigm shift in the way we examine the aetiopathogenesis of AIS is required, if we wish to treat the cause of this condition, a prize truly worth the effort.

Conflict of interest None of the authors has any potential conflict of interest.

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