

Mirror movements

Mirror movements in neurology

S F Farmer

There is a high prevalence of mirror movements in patients with asymmetric parkinsonism

Examination of patients for the presence or absence of mirror movements does not normally form part of a routine neurological investigation nor is this simple part of the motor examination normally taught to medical students and trainee neurologists. The examination requires the examiner only to observe both hands during voluntary fine finger movements of each hand in turn; for example, sequentially pressing each finger against the thumb of one hand whilst the other hand is relaxed. Mirroring occurs when there are visible involuntary movements of the "relaxed" hand that appear to replicate the timing and type of movement being carried out by the voluntarily activated hand.

Mirror movements occur during normal motor development and may reflect children's inability to suppress the activity of the ipsilateral motor cortex during attempted unilateral activation, possibly due to immaturity of transcallosal inhibition.¹ Mirror movements are common at the age of 4 years, but by age 11, if they are seen at all, they are usually weak and not sustained. Significant mirror movements are rare in adults and if present represent abnormalities of the central motor drive to the relaxed limb. These abnormal mirror movements are of great interest. Mirror movements are usually abnormal when they persist into adulthood, when they are particularly marked, very precise in their spatio-temporal characteristics, and cannot be suppressed (except through "trick" contractions of "antagonist" muscles, for example, lifting the whole hand to avoid a mirrored key strike during typing). In contrast to developmental mirror movements, congenital pathological mirror movements reflect clear abnormalities in corticospinal tract function. Neurophysiological experiments have demonstrated that this type of abnormal mirrored activity is precisely time locked to voluntary activity. Exploration of the fast conducting corticospinal tract by measuring

EMG evoked by focal trans-cutaneous magnetic or electrical stimulation of the motor cortex reveals in congenital mirror movement subjects abnormal ipsilateral and bilateral fast conducting corticospinal projections.² In addition, cross correlation and coherence analyses between left and right EMG or between EEG and EMG, confirm that during attempted unilateral voluntary contraction there is central motor drive that abnormally synchronises the discharges of left and right hand muscle motoneurons indicating that they share an abnormal common presynaptic input that is responsible for the mirroring.³ Congenital mirror movements occur when there is abnormal routing of corticospinal axons such as in X linked Kallmann's and Klippel-Feil syndromes. They are also seen in congenital hemiplegia in which a prenatal insult (probably before 28 weeks gestation) leads to persistence of functional ipsilateral corticospinal pathways from the undamaged hemisphere, which may help to sustain fine motor function despite significant contralateral central motor pathway damage.⁴

Mirror movements may be acquired. Weak mirroring may be observed after hemiplegic stroke. This phenomenon however is not produced (sadly) by significant and precise (and therefore useful) drive from the undamaged corticospinal pathways but rather appears to reflect an overall increased activation of the undamaged ipsilateral motor cortex.⁵ The phenomenon of mirror dystonia is an extremely useful diagnostic physical sign. Strictly speaking, these are not mirror movements but rather a dystonic movement emerges in a dystonic limb when it is relaxed and the opposite limb is activated. Thus there is overflow of central motor drive, possibly because of a failure of the normal cortical-cortical inhibitory processes whose malfunction is also one of the basic mechanisms of dystonia.

The paper by Espay *et al*⁶ in this issue of the journal helpfully extends the

clinical significance of mirror movements. Espay *et al* describe a high prevalence (24/27 subjects) of mirror movements in patients with asymmetric parkinsonism (due to idiopathic Parkinson's disease); furthermore, the degree of mirroring correlates with the degree of asymmetry of the parkinsonism. In contrast to dystonia, the mirror movements of parkinsonism emerge in the lesser affected limb and are mirror movements. The precise physiological mechanisms of mirror movements in parkinsonism are not yet understood and the mechanisms of mirror movement in extra pyramidal disorders will not be the same as those of congenital mirror movements which reflect pyramidal tract dysfunction. Mirroring in parkinsonism may be a transient phenomenon; perhaps the programs which are suppressed during motor development reemerge due to changes in the drive to cortex from basal ganglia structures. Espay *et al*⁶ demonstrate that mirror movements are a common early physical sign of Parkinson's disease and are useful in confirming that there is a problem with voluntary movement. Whatever neurophysiological mechanisms ultimately explain mirroring in parkinsonism, Espay *et al* have done neurology a real service in highlighting that this very useful physical sign occurs in extra pyramidal as well as pyramidal disorders. Mirroring is a physical sign that I shall be teaching future students and neurology trainees about.

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HIV and stroke

HIV infection and stroke: if not protein S deficiency then what explains the relationship?

A I Qureshi

The use of anti-retroviral agents in HIV infection is associated with an increased risk of cardiovascular disease

Over the last two decades, reports have suggested that cardiovascular diseases such as angina, myocardial infarction, and stroke can be observed in young patients with human immunodeficiency virus (HIV) infection.¹ As we review this issue, three questions need to be addressed: (1) is the risk of stroke higher in HIV infected patients?; (2) what is the underlying mechanism for the increased risk; and (3) how is the risk modified by the use of new anti-retroviral agents?

Engstrom *et al*² conducted a retrospective study of 1600 patients with acquired immunodeficiency syndrome (AIDS) and recorded 12 strokes (0.75%) in 5 years. Comparing this number with the annual incidence of stroke among young adults (aged 35–45 years) in the general population (0.025%), they concluded that patients with AIDS seem to be at substantially higher risk for stroke. We performed a case control study to determine the association between HIV infection and stroke among young persons.³ HIV infection was associated with stroke and ischaemic stroke after adjustment for other cerebrovascular risk factors. It

was initially postulated that the increased risk of stroke, particularly ischaemic stroke, was mediated by the increased susceptibility of HIV infected patients to meningitis and protein S deficiency. The report by Mochan *et al* in this issue of the Journal of Neurology, Neurosurgery, and Psychiatry is an extension of a previous study conducted by the investigators.⁴ In the previous report, the clinical, laboratory, and radiological characteristics of 33 heterosexual, HIV infected patients who presented with ischaemic strokes were prospectively studied. Underlying causes identified included coagulopathies, meningitis, cardioembolism, and hypertension. The most common coagulopathy was protein S deficiency observed in 11 of the 33 patients. In the present study, the investigators use a case control study to evaluate the relationship between protein S deficiency and ischaemic stroke in HIV infected men.⁵ A comparison between HIV infected men with and without stroke suggests that protein S deficiency is a relatively common occurrence in HIV infected patients and is not related to the increased risk of stroke. The next

question is: what explains the increased risk? Vasculopathies related to meningeal infections remain an important mechanism underlying the increased risk.³ Novel mechanisms associated with HIV infection such as the promotion of atherosclerosis by a proinflammatory effect on endothelial cells, or the indirect induction of lipid abnormalities,¹ such as a reduction in HDL cholesterol and an elevation in triglycerides, may contribute to the increased risk.

Large studies have suggested that use of anti-retroviral agents is associated with an increased risk of cardiovascular disease, particularly myocardial infarction.¹ Anti-retroviral drugs can lead to premature atherosclerosis by inducing elevations in cholesterol and triglyceride levels, insulin resistance, and lipodystrophy.¹ Further studies are needed to evaluate the effect of anti-retroviral agents on the risk of stroke.

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