Figure 2 Axial T2 weighted MR sections and MR angiography (lower right panel) of brain stem and vertebrobasilar arteries. The left vertebral artery is hypoplastic (upper left and lower right). The larger right vertebral artery turns left over the midline and the caudal basilar artery (arrow) lies in the left cerebellopontine angle in close proximity to cranial nerves VII and VIII. A small vessel crosses these cranial nerves in the right cerebellopontine angle (upper right). At the midpontine level, the basilar artery turns back to midline (lower left).

the rare bilateral hemifacial spasm by showing synchronous twitching of the muscles innervated by each facial nerve but asynchrony of muscle twitching between right and left facial muscles. In normal subjects and on the unaffected side of patients with unilateral hemifacial spasm R2 components of the electrical blink reflex are restricted to both orbicularis oculi muscles, but additional facial muscles are usually involved in patients with hemifacial spasm. This synkinesis of other muscles, which was found in this patient bilaterally, could be due to ephaptic transmission at the nerve entry zone or to facial nerve nucleus hyperexcitability; the immediate postoperative disappearance of synkinesis and abnormal R2 spread found in this case would thus be compatible with either suppression of ephaptic transmission at the nerve entry root or reduction of secondary facial nerve nucleus hyperexcitability. Both the asynchrony of spontaneous muscle bursts and the persistence or even increase of spasms on the less affected side after contralateral successful surgery in our case suggest independent trigger mechanisms on both sides most probably due to bilateral nerve entry zone irritation by aberrant vessels. Even with irritation of the nerve entry root being the probable primary cause, hyperexcitability of brain stem nuclei may also play a part; the reduced contralateral R2 component of the orbicularis oris muscle of the side contralateral to surgery (fig 1B) might be due to a loss of facilitatory effect of the operated side on the generation of this response.

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CORRESPONDENCE

Depression and its relation to lesion location after stroke

The documentation of a 20% prevalence of depression after stroke¹ has important therapeutic implications for patients with significant ischaemic heart disease and/or cardiac arrhythmia coexisting, either coincidentally or in an aetiopathogenetic role, with stroke illness. In such patients therapeutic choices

now need to be governed by the recognition that, despite comparable therapeutic benefit, adverse cardiac events such as sinus tachycardia, severe angina, and ventricular ectopy, are more likely to occur after tricyclic drugs such as nortriptyline, than after selective serotonin reuptake inhibitors such as paroxetine ($p < 0.03$).² Whereas nortriptyline causes a sustained increase in heart rate and a reduction in heart rate variability,² the second being a marker of increased cardiac mortality risk,³ no such sequelae occur after paroxetine.²

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MacHale replies:

We wholeheartedly agree with Jolobe's comments on the need to treat depressive illness after stroke with appropriate antidepressant drugs that have a low risk of cardiovascular side effects. Other studies using trazodone¹ and other selective serotonin reuptake inhibitors such as citalopram² have shown that these are safe and effective in the treatment of this condition. Unfortunately, the problem remains that the vast majority of patients with depressive illness after stroke remain untreated. Despite clear evidence of negative effects on recovery in functional status and cognitive performance³ it has been shown that inadequate and insufficient efforts at treatment had not influenced the prevalence of depression up to 5 years after stroke.⁴ The first important step therefore seems to be to encourage clinicians to recognise and treat depression after stroke, the second being to educate them about which are the appropriate medications to prescribe.

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BOOK REVIEWS

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