Transient ischaemic attacks mimicking focal motor seizures

U G R Schulz, P M Rothwell

CASE REPORT

A 61 year old woman developed attacks of rhythmic arm shaking. She would briefly feel dizzy and then her left arm would start to shake at a rate of about 3 Hz. This would last from a few seconds up to two minutes. Consciousness was never impaired and there were no other symptoms. On average the attacks occurred about once per week. They started a few months after the insertion of a cardiac pacemaker for second degree heart block. Therefore, a displaced pacing wire or unusual syncopal episodes due to pacemaker malfunction were considered among the possible diagnoses. However, her pacemaker was functioning well, a 48 hour electrocardiogram showed no significant arrhythmias and there were no other cardiovascular cause for the episodes was found. A few months later she was referred to a neurology clinic, where simple focal motor seizures were thought to be the most likely diagnosis. Computed tomography of the brain and interictal electroencephalography (EEG) were normal. She was given a trial of anticonvulsants, but these did not reduce the frequency of the attacks. Her past history of diabetes, ischaemic heart disease, hypertension, and peripheral vascular disease was noted. Hypoglycaemia was considered as a potential cause, but could not be confirmed. The patient continued to have attacks and to attend cardiology and neurology clinics over the ensuing two years, but no diagnosis was made. Five years later she developed a right intracranial haemorrhage and was reviewed in the ophthalmology clinic. She was noted to have very asymmetrical retinopathy and a carotid Doppler ultrasound examination was therefore arranged. This showed a right internal carotid occlusion and a 60% stenosis of the left internal carotid artery. The patient was referred to a neurovascular clinic for further management. She described the episodes of her left arm shaking again, and it became clear that they were often provoked by standing up and could be terminated by sitting or lying down. A diagnosis of low flow TIAs was made. This was supported by transcranial Doppler ultrasonography, which showed markedly reduced flow in the right middle cerebral artery (18 cm/sec compared with 55 cm/sec on the left).

DISCUSSION

Limb shaking TIAs are unusual. They occur in patients with severe carotid occlusive disease and, as in our patient, are often mistaken for focal seizures. However, EEG recordings during attacks have not shown any epileptic discharges, and anticonvulsants have generally been ineffective, making it very unlikely that they are due to seizure activity.

Moreover, limb shaking TIAs show no Jacksonian march and do not extend to the face. They are often brought on by postural change and can sometimes be relieved by sitting or lying down. Despite this postural relationship, there is usually no associated postural drop in blood pressure.

The exact mechanism of the limb movements is unclear. That the episodes occur in patients with severe carotid occlusive disease are often precipitated by standing up and improve after revascularisation procedures, strongly suggests that they are due to transient focal haemodynamic failure. As in our patient, cerebral blood flow and vasomotor reactivity are often reduced distal to the occluded artery. Why this should produce limb shaking is uncertain. In syncope, bilateral clonic jerks can occur and are thought to be due to subcortical release phenomena caused by diffuse cortical hypoxia. It has been suggested that the limb movements in low flow TIAs are a focal manifestation of the same process.

In our patient the attacks have persisted for seven years without development of cerebral infarction. As her disease has had such a favourable course, her treatment is currently conservative. Unfortunately, the prognosis is not always so benign. These patients are at high risk of suffering a stroke, and recognising episodic limb shaking as potential TIAs is therefore important. The management of low flow TIAs focuses on maintaining or improving cerebral blood flow by careful control of blood pressure and surgical revascularisation. In several cases an improvement of symptoms has been reported after raising blood pressure. However, in the presence of concomitant cardiac and renal disease, this may be

Abbreviations: EEG, electroencephalography; TIA, transient ischaemic attack
harmful. In such cases more aggressive treatment of hypertension is possible after surgical revascularisation, which is also effective in abolishing the attacks.\textsuperscript{1–5,7} In patients with an internal carotid stenosis, endarterectomy is the procedure of choice to abolish symptoms and reduce stroke risk. In patients with complete occlusion, extracranial-intracranial bypass surgery usually stops the attacks,\textsuperscript{39} although there is no evidence that it reduces the risk of stroke.

\textbf{ACKNOWLEDGEMENTS}

Dr Schulz is funded by a Wellcome Clinical Research Training Fellowship and Dr Rothwell by an MRC Senior Clinical Fellowship.

\textbf{Authors’ affiliations}

\textsc{U G R Schulz, P M Rothwell}, Stroke Prevention Research Unit, Oxford, UK

Correspondence to: Dr Ursula Schulz, Stroke Prevention Research Unit, Department of Clinical Neurology, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE, UK; ursula.schulz@clneuro.ox.ac.uk

\textbf{Submitted 20 September 2001}

\textbf{Accepted 10 December 2001}

\textbf{REFERENCES}


