Transient severe hypertension following alcohol withdrawal convulsions

P. G. E. KENNEDY
M.B., M.R.C.P.

JOHN MOXHAM
M.B., M.R.C.P.

Department of Medicine, University College Hospital, London

Summary

A case of transient severe hypertension following alcohol withdrawal convulsions is described. In the absence of other recognized causes of hypertension it was concluded that the hypertension was caused directly by the convulsions themselves.

Introduction

Severe hypertension is a well recognized cause of grand mal convulsions as in the syndrome of hypertensive encephalopathy. Convulsions may also be a feature of the alcohol withdrawal syndrome and are sometimes known as 'rum fits' (Victor, 1969). However, severe hypertension lasting several hours is not a recognized consequence of convulsions *per se*. In the present case transient severe hypertension occurred following grand mal convulsions due to alcohol withdrawal.

Case report

A 57-year-old woman presented in the casualty department with a one-day history of feeling generally unwell. There was a history of chronic alcoholism and of having stopped drinking on the day before admission. There was no past history of hypertension, epilepsy or head injury, and she had not been taking any drugs. On examination, the patient was flushed, sweating and the temperature was 37.3°C. She was mildly confused, very unsteady on her feet and had a fine tremor of both hands. There were no focal neurological signs and pupillary size and reaction were normal. The pulse was 100/min and regular, the BP was 140/95 mmHg and the fundi were normal. She was admitted for observation, and 2 hr later she was observed to have a grand mal convulsion, one hour before which her BP was 140/90 mmHg and 30 min after the convulsion the BP was 150/100 mmHg, pulse rate 95/min. During the evening she became increasingly confused and the BP steadily rose. Five hours after her first convulsion the blood pressure was 200/140 mmHg, pulse rate 100/min, and she had a second grand mal convulsion. Hydrallazine 10 mg i.m. was given, but

within 30 min the BP rose further, to 220/160 mmHg, pulse rate 105/min. Additional hydrallazine was ineffective and the BP was reduced to an acceptable level (190/100 mmHg) with diazoxide 300 mg i.v. She was also started on i.m. phenytoin and phenobarbitone. Over the next few hours the patient's mental state improved, the BP remained stable at 180/100 mmHg and there were no further convulsions.

Investigations were as follows: Hb, 12.7 g/dl; WCC, $8.7 \times 10^9/l$; ESR 15 mm in first hour (Westergren); plasma urea, 4.9 mmol/l; plasma electrolytes, normal; serum creatinine, 46 $\mu\text{mol/l}$; liver function tests, normal except for a serum γ -glutamyl transferase 160 i.u./l (normal 7-50 i.u./l), random blood sugar 8.0 mmol/l. Repeated MSU specimens showed a trace of protein and no cells or growth. Twenty-four hour urinary excretion of VMA was within normal limits. Cerebrospinal fluid—clear colourless fluid, pressure 130 mm water, protein 0.52 g/l, glucose 6.0 mmol/l, no cells or growth. Chest and skull X-rays, EEG, isotope and CT brain scans, ECG and IVP were all normal.

She made an uneventful recovery and remained normotensive for the rest of her admission. Drug treatment during this period included phenytoin 100 mg; phenobarbitone 30 mg; and hydrallazine 25 mg; all 4 times/day. The hydrallazine was stopped before she left hospital. She was seen in the outpatient department 2 weeks after her discharge. She had remained well and had suffered no further convulsions. Her BP was 140/85 mmHg without hypotensive medication, and she was taking phenytoin 100 mg and phenobarbitone 30 mg, both 4 times/day. She had been drinking heavily but had no abnormal physical signs. The patient has been seen on several occasions since discharge and at all times the BP has been normal.

Discussion

This patient had the clinical features of acute alcohol withdrawal. The convulsions were typical of 'rum fits' in that they occurred in a chronically

intoxicated patient after the cessation of drinking, were grand mal in nature, occurred in a short burst with a short interval between the first and last convulsion, and were associated with a normal EEG (Victor, 1969).

The patient was normotensive before the convulsions and investigations revealed no recognized cause for her hypertension. Raised intracranial pressure producing systemic hypertension was excluded by the presence of normal cerebrospinal fluid pressure and normal fundi. There was no evidence of renal impairment or pheochromocytoma, and non-ischaemic CNS causes of hypertension such as acute encephalitis (Krogsgaard and Quaade, 1961), head trauma (Raab, 1949) and posterior fossa tumour (Dickinson, 1965) were excluded. It was, therefore, concluded that her severe, acute hypertension was caused directly by the convulsions themselves. Transient hypertension occurring only for the duration of a grand mal convulsion has been previously described (Mosier *et al.*, 1957). However, in the present patient, the hypertension was sustained and persisted for many hours after the muscular overactivity associated with her convulsions had subsided. Van Buren (1958) reported hypertension associated with various autonomic abnormalities occurring during the phase of post-ictal automatism in patients with temporal lobe epilepsy. This latter study, however, excluded patients with grand mal convulsions and the BP elevations noted were mild (diastolic BP <110 mmHg) contrasting with the considerable hypertension found in the present case.

The authors are not aware of a previous published report of such severe prolonged hypertension following grand mal convulsions. Although the potential dangers of rapidly lowering systemic hypertension due to a neurogenic cause were fully appreciated, restoration of this patient's BP to normal levels proved entirely beneficial. The mechanism of the hypertension in this situation is unclear, but since this patient showed some clinical features of autonomic overactivity and in view of the fact that autonomic abnormalities in association with convulsions have been previously described (Van Buren, 1958; Mosier *et al.*, 1957) the authors suggest that a paroxysmal autonomic nervous system disturbance was the most likely explanation. Although the hypertension which follows convulsions is usually mild, this case illustrates that it may be severe, prolonged and require urgent treatment.

References

- DICKINSON, C.J. (1965) *Neurogenic Hypertension*, p. 20, Blackwell Scientific Publications, Oxford.
- KROGSGAARD, A.R. & QUADE, F.L. (1961) Hypertension caused by encephalitis: Review and report of a case. *Acta medica scandinavica*, **169**, 389.
- MOSIER, J.M., WHITE, P., FISHER, J.E. & TAYLOR, R. (1957) Cerebroautonomic and myographic changes accompanying induced seizures. *Neurology*, **7**, 204.
- RAAB, W. (1949) Hypertension and tachycardia due to concussion of the brain. *American Heart Journal*, **37**, 237.
- VAN BUREN, J.M. (1958) Some autonomic concomitants of ictal automatism. *Brain*, **81**, 505.
- VICTOR, M. (1969) A study of epilepsy in the alcoholic patient. In: *Modern Neurology* (Ed. Locke, S.), p. 555, J. & A. Churchill Ltd, London.