are rarely of any help.³ Transoesophageal 2-D echo could be useful for detecting retroauricular paragangliomas.

Although both MIBG and CT have improved the ability to localize phaeochromocytomas,⁴ these imaging techniques may not detect intrathoracic forms.^{1,2} During conventional CT of the thorax, cardiac hypervascularized phaeochromocytomas are barely differentiated from surrounding vessels and cardiac chambers. Although MIBG scintigraphy has been shown to be valuable in the localization of ectopic phaeochromocytoma,^{1,4} false negative (5–10%) have been reported.⁴ When CT and MIBG fail to detect a tumour, invasive procedures may be necessary.¹ Cardiac paragangliomas are vascularized from the aorta or from the coronary arteries.² They often drain to the right atrium. Therefore blood samples from superior vena cava and right atrium can be of value in assessing such phaeochromocytomas.^{1–3,5} Coronary angiography is a useful means of locating the phaeochromocytoma and assessing the vascularity of the tumour before surgery.⁶

In retrospect, this strategy can be discussed with the arrival of MR imaging which makes invasive techniques obsolete. In patients with negative CT and MIBG scans, MR imaging may be a useful and sensitive tool for the detection and delineation of phaeochromocytoma, and more precisely a cardiac phaeochromocytoma.⁶

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Hypoglycaemia masquerading as a stroke

Sir,
Cerebrovascular events account for a significant morbidity and mortality in the elderly.¹ Diabetes mellitus and

its treatment can cause similar clinical findings with an altered level of consciousness. Sulphonylurea therapy, such as glibenclamide, is a recognized cause of this when precipitating hypoglycaemia.^{2,3}

An 81 year old woman was admitted drowsy with complete aphasia but some motor response to verbal commands. Upper motor neurone signs were present in the left arm, and both legs, with bilaterally up going plantars. Her pupils reacted equally to light with loss of vertical eye movements but intact horizontal conjugate eye movements. Pulse rate was 80 beats per minute regular, supine blood pressure 190/90 mmHg with normal cardiac auscultation. A left carotid bruit was audible.

A history was later obtained that on the day of admission she had become confused with a 3 hour history of sudden onset left-sided limb weakness and complete aphasia.

Medication on admission included prednisolone 6 mg once daily, for polymyalgia rheumatica and glibenclamide 5 mg once daily for diabetes mellitus diagnosed 2 years previously.

She lived with her husband in a ground floor rented flat and, prior to this episode, walked with a stick and received no social service support. She was an ex-smoker and drank no alcohol.

Capillary blood BM Stix was 2–4 mmol/l, blood glucose was 1.6 mmol/l. Plasma urea and electrolytes, liver function tests, electrocardiogram and chest X-ray were normal.

Following an intravenous bolus of 50% dextrose she immediately regained the use of all limbs and her speech returned. Repeat neurological examination was normal except for a right extensor plantar response. Repeat blood glucose estimation was 7.3 mmol/l. Capillary blood BM Stix were monitored and remained in the 7–11 mmol/l range overnight.

This case highlights the importance of performing BM Stix and formal blood sugars on admission in all patients receiving glibenclamide therapy, particularly if there is an altered level of consciousness or focal neurology. The significant mortality of glibenclamide-associated hypoglycaemia should also be remembered.³

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