An 82 year old man was referred to the emergency room by his general practitioner for a right frontoparietal headache. The preceding day he had tripped and fallen, hitting the back of his head on the floor. Computed tomography showed a cortical contre coup haematoma. In view of ventricular standstill noted on ECG, a temporary pacing wire was inserted and a dual chamber permanent pacemaker was subsequently implanted. Intracerebral bleeding was treated conservatively and the patient made a good recovery. All patients admitted with head injury and sinus bradycardia or sinus arrest should be nursed at 15° to 30° with instructions to avoid the head up and supine positions. Furthermore, brain CT should be promptly recorded to assess for intracerebral haematoma and raised intracranial pressure and, if they are confirmed, these patients with cardiovascular compromise should benefit from close collaboration between neurosurgeon and cardiologist. Urgent pacing should be considered for all patients with head injury who experience symptomatic bradycardia or ventricular standstill.

An 82 year old man was referred to the emergency room by his general practitioner for a right frontoparietal headache. The preceding day he had tripped and fallen, hitting the back of his head on the floor. He was being treated with inhalers for chronic obstructive pulmonary disease and aspirin 75 mg once daily in addition to diltiazem 90 mg twice daily for probable angina pectoris. He was a former smoker of 55 pack-years.

On clinical examination he had superficial lacerations to the occiput. He was alert and orientated. He was in sinus rhythm with a resting heart rate of 90 beats/min. The supine blood pressure was 132/84 mm Hg. He had normal heart sounds and a normal cardiovascular examination.

On sitting forward, however, he felt extremely nauseated and there was loss of cardiac output. Indeed, ventricular standstill was noted on the ECG monitor (fig 1), and this was reproducible every time he sat upright. Sinus rhythm was restored and symptoms resolved when he returned to the lying position (20°). Carotid sinus massage had no effect on the heart rate or cardiac output. Neurological examination showed horizontal nystagmus on right lateral gaze, with exaggerated deep tendon reflexes and positive Babinski’s sign on the left side. There was no overt limb weakness.

Haemoglobin was 111 g/l, serum potassium was 4.2 mmol/l, and magnesium was 1.01 mmol/l. Serum creatinine was raised at 217 μmol/l. The resting ECG showed normal sinus rhythm with no evidence of atrioventricular block. The chest radiograph was within normal limits. Transthoracic echocardiography confirmed normal cardiac structure and function. Computed tomography showed a cortical contre coup haematoma. In view of ventricular standstill noted on ECG, a temporary pacing wire was inserted and a dual chamber permanent pacemaker was subsequently implanted. Intracerebral bleeding was treated conservatively and the patient made a good recovery. All patients admitted with head injury and sinus bradycardia or sinus arrest should be nursed at 15° to 30° with instructions to avoid the head up and supine positions. Furthermore, brain CT should be promptly recorded to assess for intracerebral haematoma and raised intracranial pressure and, if they are confirmed, these patients with cardiovascular compromise should benefit from close collaboration between neurosurgeon and cardiologist. Urgent pacing should be considered for all patients with head injury who experience symptomatic bradycardia or ventricular standstill. 

Figure 1  ECG recording showing ventricular standstill (arrow) with the patient sitting forward.
tomography (CT) of the brain showed a 2.5 cm × 2.5 cm cortical contre coup haematoma with extensive contusions located over the right frontal lobe (fig 2).

In view of the ventricular standstill a temporary pacing wire was inserted. A dual chamber permanent pacemaker was subsequently implanted. One year hence the patient is in sinus rhythm and rarely requires the pacemaker. The intracerebral bleeding was treated conservatively as advised by our local neurosurgical centre after review of the CT films. The patient has made a good recovery.

DISCUSSION
This case highlights that primary intracerebral haematoma is an unusual but important cause of loss of cardiac output and ventricular standstill. Although the initial CT was not suggestive of raised intracranial pressure at presentation, in view of the patient’s worsening symptoms, it is likely that brain swelling progressed further, and this is an accepted sequela in the context of contre-coup type head injury. We postulate, therefore, that a rise in intracranial pressure ensuing from the cerebral haemorrhage and contusions causes temporal lobe displacement through the tentorium cerebelli leading to increased pressure on the cardiac and vomiting centres that are situated in the medulla oblongata of the brainstem, resulting in loss of cerebral autoregulation. Changing the head position from the lying position (15° to 30°) to sitting upright leads to an increase in intracranial pressure aggravating the brain shift and the brainstem ischaemia.

An alternative explanation may be that ventricular standstill is a manifestation of medullary ischaemia caused by stretching of perforating branches of the basilar artery and the downward displacement of the brainstem. Vomiting is attributed to compression or ischaemia of the vomiting centre in the medulla oblongata and the bradycardia or asystole caused by ischaemia of the cardiac centre.

Our patient’s symptoms were apparent only on sitting forward and this can be explained by the observation that in patients with cerebral haematoma, the level of head elevation influences the intracranial and cerebral perfusion pressures. Resolution of the above haematoma, as this case shows, leads to restoration of normal sinus rhythm. Durward and colleagues and others have shown that in patients with intracranial hypertension, both the supine position (0°) and levels of head elevation at 60° and above are associated with detrimental effects on intracranial pressure, cerebral perfusion pressure, and cardiac output.

We suggest, therefore, that all patients admitted with head injury and sinus bradycardia or sinus arrest should be nursed at 15° to 30° with instructions to avoid the head up and supine positions. Furthermore, prompt brain CT ought to be recorded to assess for intracerebral haematoma and raised intracranial pressure and, if these are confirmed, it is vital that these patients with cardiovascular compromise should benefit from close collaboration between neurosurgeon and cardiologist. Urgent pacing should be considered for all patients with head injury who experience symptomatic bradycardia or ventricular standstill.

Authors’ affiliations
A K J Mandal, S Baltsezak, C G Missouris, Wexham Park Hospital, Slough, Berkshire, UK

All authors agree there are no conflicts of interest

Correspondence to: Dr Amit K J Mandal, Department of Cardiology, Wexham Park Hospital, Wexham Street, Slough, Berkshire SL2 4HL, UK; akjm@mac.com

Accepted 14 September 2004

REFERENCES
Intracerebral haematoma masquerading as ventricular standstill

A K J Mandal, S Baltsezak and C G Missouris

*Heart* 2005 91: e1
doi: 10.1136/hrt.2004.046318

Updated information and services can be found at:
http://heart.bmj.com/content/91/1/e1

These include:

References

This article cites 2 articles, 0 of which you can access for free at:
http://heart.bmj.com/content/91/1/e1#BIBL

Email alerting service

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/