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Subarachnoid haemorrhage

Rustam Al-Shahi, Philip M White, Richard J Davenport, Kenneth W Lindsay

Patients with spontaneous (non-traumatic) subarachnoid haemorrhage usually present first to their general practitioner. As general practitioners may see only a few cases during their career, subarachnoid haemorrhage can be a diagnostic and management challenge¹; the incidence is about 8 per 100 000 per year.² The condition accounts for 3% of patients presenting to emergency departments with headache³ and around 20 admissions per year to a general hospital covering 300 000 people. The prognosis remains poor: up to half of patients die and one third of survivors are left dependent.⁴ Early treatment can improve outcome⁵⁻⁸; therefore prompt diagnosis and referral to a neuroscience unit is important.

How subarachnoid haemorrhage presents

Patients with subarachnoid haemorrhage usually present with a characteristic combination of symptoms (box 1). Sudden severe headache is the cardinal symptom, but it may be the only symptom in up to one third of patients with aneurysmal subarachnoid haemorrhage.¹² When patients were asked how long it took for their headache to reach its maximum severity half of those with subarachnoid haemorrhage described it as instantaneous, one fifth said it developed over 1-5 minutes, and the rest said it escalated over more than five minutes.¹² The headache usually persists for several days but may occasionally be much shorter. Even in the emergency department the positive predictive value of instantaneous severe headache for aneurysmal subarachnoid haemorrhage is only 39% (95% confidence interval 29% to 50%),¹² so the speed of onset cannot be relied on to identify all cases of subarachnoid haemorrhage.

Although some believe that "sentinel bleeds" or "warning leaks" precede aneurysmal subarachnoid haemorrhage, the evidence is that headaches preceding the haemorrhage are rare and do not help in its diagnosis.¹⁴ Overestimation of the importance of sentinel bleeds arose from recall bias in hospital based studies.¹ We recommend that the terms sentinel bleeds and warning leaks should be abandoned: people either have had a subarachnoid haemorrhage or not and the important task is to recognise when they have.^{1 15}

Other causes of sudden severe headache

Only about one in four people presenting with sudden severe headache will have had a subarachnoid

Summary points

Subarachnoid haemorrhage should be suspected in someone with sudden severe headache that peaks within minutes and lasts more than an hour

Subarachnoid haemorrhage cannot be excluded by unenhanced computed tomography of the brain because the sensitivity of the procedure declines rapidly within hours of symptom onset

When an unenhanced computed tomogram of the brain is normal, specialist advice should be sought about subsequent lumbar puncture

When patients are referred to a neuroscience unit, the referring clinician should specify time from onset of symptoms, age, comorbidities, conscious level, and any neurological deficit

Oral nimodipine 60 mg every four hours reduces poor outcome, but good evidence for other medical interventions is limited

Endovascular coiling is superseding neurosurgical clipping for the occlusion of many ruptured aneurysms

Multidisciplinary follow-up clinics help manage complications of subarachnoid haemorrhage

Division of Clinical
Neurosciences,
University of
Edinburgh, Western
General Hospital,
Edinburgh
EH4 2XU

Rustam Al-Shahi
MRC clinician
scientist

Philip M White
consultant
neuroradiologist

Richard J
Davenport
consultant neurologist

Institute of
Neurological
Sciences, Southern
General Hospital,
Glasgow

Kenneth W Lindsay
consultant
neurosurgeon

Correspondence to:
R Al-Shahi
rustam.al-shahi@
ed.ac.uk

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haemorrhage,¹⁴ a further 40% will have benign thunderclap headache,¹² and the rest will have other primary and secondary headache syndromes (box 2). When sudden severe headache is the only symptom, one in 10 cases turns out to be subarachnoid haemorrhage,¹⁴ so the absence of other symptoms cannot be used to rule out the condition. Conversely, other symptoms of subarachnoid haemorrhage (box 1) may accompany other causes of sudden severe headache (box 2), so they cannot reliably distinguish subarachnoid haemorrhage.



References w1-w14 and ongoing research studies are on
bmj.com

Sources and selection criteria

We searched for relevant articles in Ovid Medline between 1966 and November 2005 using the search term subarachnoid haemorrhage. We also searched the Cochrane Database of Systematic Reviews.^{5-7 9-11} We have classified the evidence for our treatment recommendations.^{w1}

Investigations for suspected subarachnoid haemorrhage

Because no clinical feature is sufficiently reliable to make the diagnosis,¹² subarachnoid haemorrhage must be excluded in anyone presenting with sudden severe headache that is maximal within minutes, lasts for more than an hour, and has no alternative explanation.¹⁶ Box 3 lists the initial investigations for suspected subarachnoid haemorrhage.

The ideal investigation for subarachnoid haemorrhage is unenhanced computed tomography of the brain, which needs to be interpreted by an experienced radiologist and carried out as soon as possible after the onset of headache (immediately if consciousness is impaired). Delays in scanning allow subarachnoid blood time to degrade and increase the possibility of a tomogram appearing normal. Third generation computed tomography scanners miss about 2% of cases of subarachnoid haemorrhage within 12 hours^{w5} and about 7% by 24 hours.^{w6} Subarachnoid blood is almost completely reabsorbed within 10 days. Changes can be subtle and interpretation requires expertise (fig 1).

When and how to examine cerebrospinal fluid

Anyone with suspected subarachnoid haemorrhage and a normal computed tomogram requires a lumbar puncture.¹⁷ Because expertise is required in both the conduct and the timing of lumbar puncture, as well as in the interpretation of cerebrospinal fluid results,^{1 18} we recommend that cases should at least be discussed with a specialist at a neuroscience unit before the procedure.

Unless there is any suspicion of an alternative diagnosis such as meningitis, we delay lumbar puncture for at least six hours—preferably 12 hours—after headache onset. This allows sufficient time for haemoglobin to degrade into oxyhaemoglobin and bilirubin.¹⁹ Bilirubin signifies subarachnoid haemorrhage because it is only synthesised *in vivo*, unlike oxyhaemoglobin, which may result from a traumatic tap or prolonged

Box 2: Common causes of sudden severe headache¹⁴

- Spontaneous (non-traumatic) subarachnoid haemorrhage
- Other secondary headache syndromes
 - Vascular—intracranial venous thrombosis; intracerebral, intraventricular, extradural, or subdural haemorrhage; ischaemic stroke; arterial dissection; vasculitides
 - Infection—for example, meningitis, encephalitis
 - Acute hydrocephalus—for example, colloid cyst of the third ventricle)
 - Intracranial tumour—including pituitary apoplexy
 - Intracranial hypotension—spontaneous or after dural puncture
 - Metabolic—for example, pheochromocytoma, tyramine ingestion with monoamine oxidase inhibitors
- Primary headache syndromes^{w2}
 - Thunderclap headache
 - Migraine
 - Cluster headache
 - Headache associated with sexual activity or exertion

storage or agitation of bloodstained cerebrospinal fluid *in vitro*.²⁰

The opening pressure of cerebrospinal fluid must be recorded and samples analysed for protein, cells, and glucose (paired with a serum sample). To minimise the risk of oxyhaemoglobin arising *in vitro*, the last (least bloodstained) sample of cerebrospinal fluid should be transported to the laboratory by hand and centrifuged immediately.²¹ If there are delays the fluid should be protected from light to prevent degradation of bilirubin. Bilirubin causes yellow pigmentation of the supernatant after centrifugation of cerebrospinal fluid, known as xanthochromia. Clinicians should use the naked eye to compare the colour of the cerebrospinal fluid supernatant with water against a white

Box 1: Presenting symptoms

Sudden death—occurs in 10% of cases of spontaneous subarachnoid haemorrhage¹³

Sudden severe headache—maximal immediately or within minutes, and lasting an hour or more (usually days)¹⁴

Other symptoms of meningism—nausea, vomiting, photophobia, neck stiffness

Loss of consciousness—transient at onset (around 50% of cases) or persisting

Epileptic seizures—about 6% of cases

Focal neurological symptoms—dysphasia, hemisensory or motor symptoms

Box 3: Initial investigations

Full blood count—anaemia (sickle cell disease), leucocytosis (after a seizure, systemic infection)

Coagulation screen—underlying coagulopathy

Urea and electrolytes—hyponatraemia is common after subarachnoid haemorrhage due to salt wasting, not inappropriate antidiuretic hormone secretion

Serum glucose—hypoglycaemia needs correction, hyperglycaemia is associated with poor outcome after subarachnoid haemorrhage^{w3}

Serum magnesium—hypomagnesaemia is common and associated with poor outcome after subarachnoid haemorrhage^{w4}

Chest radiography—pulmonary oedema, aspiration

12 lead electrocardiography—cardiac arrhythmia, ST segment changes, myocardial “stunning”

Unenhanced computed tomography of the brain—as soon as possible after onset of headache (< 24 hours)

Lumbar puncture—if an unenhanced computed tomogram of the brain is normal

Computed tomography angiography—if subarachnoid haemorrhage is confirmed by computed tomography or lumbar puncture

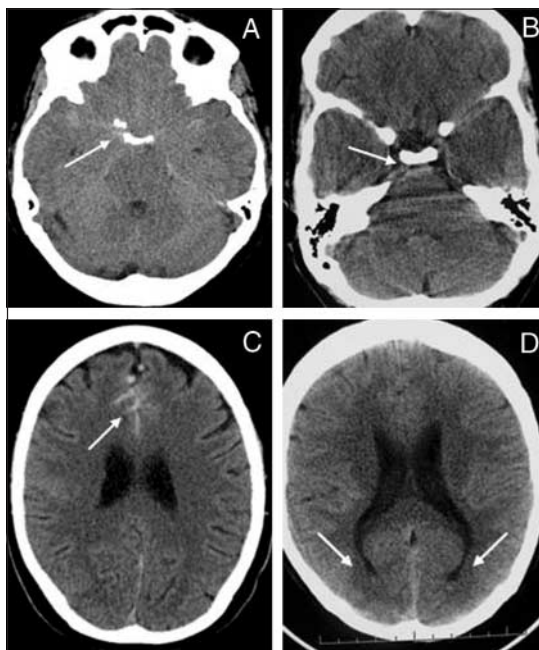


Fig 1 (A) Subarachnoid haemorrhage from right posterior communicating artery aneurysm with hyperdense or isodense blood in anterior and posterior basal cisterns (arrow). (B) Blood in prepontine cistern (arrow). (C) Blood in anterior interhemispheric fissure (arrow). (D) Blood sedimenting in occipital horns of lateral ventricles (arrows)

background,²² but every sample should be analysed for bilirubin using spectrophotometry.²¹ The estimation of erythrocyte counts in three consecutive samples of cerebrospinal fluid does not reliably distinguish subarachnoid haemorrhage from a traumatic tap.¹⁹

Box 4: Complications of aneurysmal subarachnoid haemorrhage

Rebleeding (without aneurysm occlusion²⁷)

On day 1: 15%

By one month: 40%

After six months: 3% per year

Immediate cerebral ischaemia—due to raised intracranial pressure and hence reduced cerebral perfusion pressure

Delayed cerebral ischaemia—peaks between days 4 and 14 after subarachnoid haemorrhage

Hydrocephalus

Seizures

Cardiopulmonary dysfunction—predicted by elevated cardiac troponin I²⁷

Hyponatraemia or hypomagnesaemia—caused by salt wasting

Normal computed tomograms and cerebrospinal fluid samples within days of presentation

If a computed tomogram and cerebrospinal fluid (including spectrophotometry) are normal within two weeks of a sudden severe headache, then subarachnoid haemorrhage is excluded and an alternative diagnosis must be considered (box 2).

Suspicion of subarachnoid haemorrhage more than two weeks after onset

The sensitivity of computed tomography for subarachnoid haemorrhage declines rapidly after 10 days. Xanthochromia is only detected in 70% of cases after three weeks and 40% after four weeks.²³ People who present two or more weeks after a suspected subarachnoid haemorrhage therefore should be referred urgently to a neuroscience unit for consideration of further investigations.

Investigations for the cause of subarachnoid haemorrhage

Possible causes of subarachnoid haemorrhage determine further investigations. Three quarters of spontaneous cases are due to a ruptured aneurysm, 20% have no identifiable cause (of which at least half are due to idiopathic or non-aneurysmal perimesencephalic subarachnoid haemorrhage), and the remainder are caused by a variety of rare disorders such as arteriovenous malformations of the brain or spine, arterial dissection, sympathomimetic drugs, tumours, or vasculitis.

The pattern of blood on computed tomograms may suggest the cause. Ruptured aneurysms are most commonly found on the anterior communicating artery complex (suggested by blood in the interhemispheric fissure, fig 1C), followed by the internal carotid artery, middle cerebral artery (blood in the sylvian fissure), and vertebrobasilar circulation. About 20% of people with subarachnoid haemorrhage have multiple aneurysms, so the pattern on the computed tomogram may be crucial to identify which aneurysm has ruptured. Idiopathic perimesencephalic subarachnoid haemorrhage is restricted to the perimesencephalic cistern anterior to the midbrain, but rupture of an aneurysm in the posterior circulation underlies 5% of

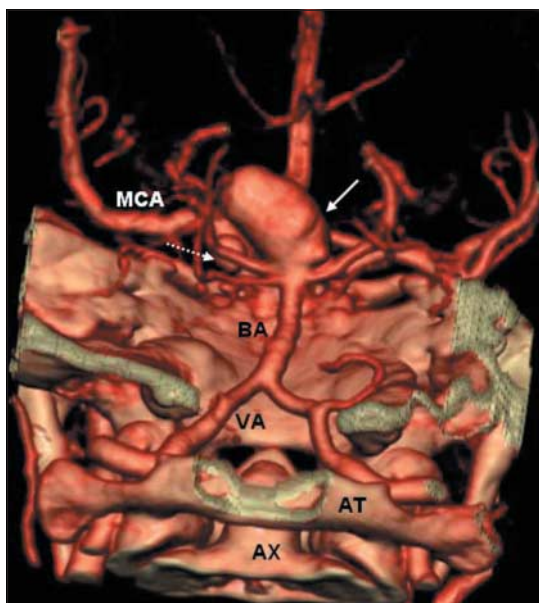


Fig 2 Volume rendered computed tomography angiogram, viewed from posterior to anterior, showing a 20 mm diameter aneurysm (solid arrow) at tip of basilar artery (BA) and a 6 mm diameter aneurysm (dashed arrow) on the left posterior communicating artery. MCA=left middle cerebral artery; VA=left vertebral artery; AT=atlas (C1); AX=odontoid process of axis (C2)

Table 1 World Federation of Neurological Surgeons grading scheme for aneurysmal subarachnoid haemorrhage¹⁸

Grade	Glasgow coma score	Motor deficit*
I	15	Absent
II	13 or 14	Absent
III	13 or 14	Present
IV	7 to 12	—
V	3 to 6	—

*Excludes cranial neuropathies but includes dysphasia.

this pattern of subarachnoid haemorrhage, so further investigation is always required.

Multislice CTA (computed tomography angiography) is our preferred investigation for underlying causes of spontaneous subarachnoid haemorrhage because of its speed, tolerability, convenience, and the ability to provide three dimensional reconstructions (fig 2). The sensitivity of the procedure for identifying aneurysms greater than 3 mm diameter is about 96% but poorer for smaller aneurysms.^{24 25} We proceed to four vessel catheter angiography if aneurysmal subarachnoid haemorrhage is strongly suspected but the computed tomography angiogram is normal. Repeated cerebral catheter angiography, spinal catheter angiography, or magnetic resonance imaging may be necessary to identify alternative causes in some cases. For people with a purely perimesencephalic distribution, a normal, good quality computed tomography angiogram allows a diagnosis of idiopathic

perimesencephalic subarachnoid haemorrhage without the need for further investigation.¹⁹

Outcome after aneurysmal subarachnoid haemorrhage

Case fatality after aneurysmal subarachnoid haemorrhage in the community is 12% and a further 10% of those who reach hospital alive die within 24 hours. In population based studies the overall case fatality was about 50%, which has reduced marginally between 1960 and the mid-1990s.⁴ Almost all deaths due to subarachnoid haemorrhage occur within the first three weeks, most due to rebleeding (box 4). Around one third of survivors are dependent,⁴ often with cognitive impairment,²⁶ and two thirds have a reduced quality of life.^{19 26}

The three strongest predictors of death or dependence are impaired conscious level on admission, increasing age, and large volume of blood on initial computed tomography of the brain. The World Federation of Neurological Surgeons grading scale standardises clinical evaluation over time and helps estimate prognosis (table 1).

Management of aneurysmal subarachnoid haemorrhage

People with confirmed subarachnoid haemorrhage should be referred to a neuroscience unit for supportive management and interventions directed at preventing complications (box 4). The specific treatment of complications such as hydrocephalus, delayed ischaemia, or rebleeding is covered elsewhere.²⁸

Best medical management

Frequent checking of the Glasgow coma score and pupillary responses as well as vigilance for focal neurological deficits is essential. A drop of just one point in the Glasgow coma score may be the first sign of a complication. Close monitoring of blood pressure, fluid balance, heart rhythm, and respiratory function are crucial because of the risk of cardiac arrhythmia and pulmonary oedema. Table 2 lists the medical treatments for aneurysmal subarachnoid haemorrhage. Several drugs could plausibly improve outcome, including magnesium, antiplatelet agents, statins, and endothelin receptor antagonists (see bmj.com).

When and how should ruptured aneurysms be secured?

The main purpose of occluding an aneurysm is to prevent rebleeding. Age, World Federation of Neurological Surgeons grade, comorbidity, time from onset of subarachnoid haemorrhage, and the aneurysm's vascular anatomy help determine whether intervention is worthwhile and possible.

Although neurosurgical "clipping" became the standard treatment of aneurysms in the 20th century, endovascular occlusion of ruptured aneurysms using detachable coils ("coiling," fig 3) is now superseding clipping (grade A, level 1+).⁶ For aneurysms suitable for either treatment, coiling confers an absolute risk reduction over clipping of about 7% (25% relative risk reduction) for dependency or death at one year, with benefit sustained to seven years³²; the number needed

Table 2 Medical interventions for aneurysmal subarachnoid haemorrhage

Treatment	Indication or benefit	Evidence ^{wt}
Standard practice:		
Nimodipine (oral) 60 mg every four hours for three weeks	Prevention—reduces risk (absolute 5%, relative 18%) of poor outcome and delayed cerebral ischaemia	Grade A, ⁵ level 1+
≥3 litres intravenous 0.9% saline daily	Prevention—sodium depletion and hypovolaemia contribute to delayed cerebral ischaemia. ²⁹ Monitor fluid balance and cardiac function	Grade C, level 2+
Analgesia (paracetamol 1 g every six hours or dihydrocodeine 30 to 60 mg every four hours, or both)	Pain relief	—
Graduated compression stockings	Prophylaxis against deep vein thrombosis	Grade B, ³⁰ level 1++
Antiemetics	As required	—
Stool softeners	As required	—
Variation in practice:		
Intermittent pneumatic compression	Prophylaxis against deep vein thrombosis	Grade B, ³¹ level 1+
Plasma volume expanders	Poor evidence for prevention or treatment of delayed cerebral ischaemia, and increases complications	Grade B, ¹⁰ level 1-
Antihypertensives	No proved benefit in preventing rebleeding, and may cause cerebral ischaemia ⁹	Grade B, ¹⁰ level 1-
Antifibrinolytics	No overall benefit because reduction of rebleeding is offset by more delayed cerebral ischaemia. Use before aneurysm occlusion seems more promising, but evidence is still lacking ¹¹	Grade A, ¹¹ level 1++
Antipyretics	Temperature >37.5°C	Fever associated with poor outcome ¹²
Insulin sliding scale or infusion	Plasma glucose >11 mmol/l	Hyperglycaemia associated with poor outcome ¹³
Magnesium supplementation	Plasma magnesium <0.7 mmol/l	Hypomagnesaemia associated with poor outcome ^{14, 13}
Antiepileptic drugs	For treatment (not prophylaxis) of seizures	—



Fig 3 Digital subtraction catheter angiography (internal carotid artery injection) showing a ruptured anterior communicating artery aneurysm (A, arrow) treated with endovascular coiling (B, arrow)

to coil to prevent one poor outcome is 14 (95% confidence interval 10 to 25).⁶

The evidence for coiling over clipping is influenced by one large trial, the international subarachnoid aneurysm trial.³² Those included in the trial were mostly young and had good World Federation of Neurological Surgeons grades and small anterior circulation aneurysms: this represents half to three quarters of people with aneurysmal subarachnoid haemorrhage³³ and reflects the lack of equipoise about most posterior circulation aneurysms that had already developed in clinical practice at the time of the trial. Despite the overall superiority of coiling, complete aneurysm occlusion was more likely to be achieved with clipping. Evidence for the durability of coiling beyond seven years is awaited from the trial.

Aneurysm occlusion tends to be attempted as soon as practicable (within 3–4 days) after onset of subarachnoid haemorrhage to prevent rebleeding. Morbidity seems to occur more often after clipping in the second week after haemorrhage when the risk of delayed cerebral ischaemia is greatest (grade B, level 1+),^{7 8} although the timing of coiling does not seem to influence morbidity related to it (grade C, level 2+).³⁴ Immediate evacuation of some intracerebral haematomas caused by aneurysm rupture, with concomitant aneurysm clipping, is supported by one small randomised trial (grade B, level 1 –).³⁵

What to do after aneurysmal subarachnoid haemorrhage

Survivors of subarachnoid haemorrhage need good advice and information on support groups. Multidisciplinary follow-up clinics involving a neurologist, neurosurgeon, neuroradiologist, and professions allied to medicine are likely to help identify and manage the physical and cognitive complications, which often delay or preclude return to work.²⁶ Tackling modifiable risk factors for aneurysmal subarachnoid haemorrhage is likely to be beneficial in preventing its recurrence: the most important are smoking, hypertension, and excessive alcohol intake (>150 g (19 units) of ethanol per week), each individually conferring a twofold to threefold increase in risk.³⁶

Only 2% of relatives of people with aneurysmal subarachnoid haemorrhage are similarly affected, but the risk is higher when two or more first degree relatives are affected.³⁷ Familial aggregation is only partly explained by classic risk factors¹⁴ and monogenic conditions such as adult polycystic kidney disease. Screening family members for the presence of

Information for patients

The Brain and Spine Foundation (www.brainandspine.org.uk/)—provides information, advice, and booklets (for example, recovering from a subarachnoid haemorrhage, a guide for patients and carers, coiling of brain aneurysms)

The Brain Aneurysm Foundation (www.bafound.org/)—provides information and support

unruptured aneurysms may be justifiable when two or more first degree relatives are affected, taking into account age, comorbidities, and other risk factors.³⁷ This approach is not supported by evidence from clinical trials.

Conclusion

People with subarachnoid haemorrhage should be managed by multidisciplinary teams in specialist neuroscience units. Future improvements in treatment of aneurysms and the management of medical complications of subarachnoid haemorrhage should be tested in randomised controlled trials. Such research should improve outcome, which should be monitored in population based studies or by comprehensive national audits.³³

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Who decides?

Tom had been an active child, enjoying school and playing football. More importantly, he was a central part of his family. He was born with an atrioventricular septal defect and trisomy 21. At that time, intervention had been thought to be too risky and, as a result, he had frequent hospital admissions and poor exercise tolerance. Outcomes and perceptions changed, and, at 7 years old, he underwent surgical repair (pulmonary vascular resistance was low). This led to four fun years of walking and playing.

We met him when he was 13 years old, and he was six months into his ordeal. He had had recurrent arterial thromboemboli, resulting in occlusion of the aorta at the bifurcation. After an embolectomy and a long stay in the paediatric intensive care unit, his medical problems included renal failure, lower limb paralysis, and severe back pain. As plans for discharge were made, with home care and equipment being organised, he became unwell again. Because of his recent traumatic experiences, Tom and his family were not keen on transfer back to intensive care. Optimal medical therapy was continued.

Tom quickly became withdrawn. He was quiet and scared. He barely ate or drank. Venepuncture had made him angry, but this changed: at first he would cry, and then he just lay still and silent. His sleep was broken. Up to this point, his mother, brothers, and certain nurses could easily bring on laughter even when he was in pain or frustrated.

Although there were no direct discussions with him, we took his cue. Like other teenagers, he had been

exposed to death, and some of his school friends had died. His understanding was limited but not poor. For example, he said that being in intensive care was being dead and that going back was dying again. From a medical standpoint, the prognosis for his condition was undefined and the chances of him surviving the current acute problem were low. His family faced these issues with us bravely.

When all his family and friends began arriving and the needles stopped, he was different. The laughter returned, as did his appetite for McDonalds. His spirit returned, and his dislike for wheelchairs did not stop him going to meet Michael Owen (he did the 30 mile round trip on a stretcher). His last few days were happy, and he was surrounded by those he loved.

We remain inspired by Tom and his family.

Jasveer Singh Mangat *specialist registrar in paediatric cardiology (jasveer_mangat@yahoo.com)*, Priya Kumar *specialist registrar in paediatrics, Guy's and St Thomas' NHS Trust, London*

We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake*, or any other piece conveying instruction, pathos, or humour. Please submit the article on <http://submit.bmj.com>. Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.