

Spontaneous intracerebral haemorrhage

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Spontaneous (non-traumatic) intracerebral haemorrhage accounts for at least 10% of all strokes in the United Kingdom,¹ but the incidence is higher in some ethnic groups.^{w1} Intracerebral haemorrhage may present with a sudden focal neurological deficit or a reduced level of consciousness, after which it kills about half of those affected within one month and leaves most survivors disabled.²

Although early case fatality after spontaneous intracerebral haemorrhage has not changed over the past two decades,^{1 2} brain imaging has illuminated the pathophysiology of intracerebral haemorrhage and its various causes,^{3 w2} such that the term primary intracerebral haemorrhage now seems antiquated. Improving prevention of intracerebral haemorrhage in primary care and its outcome in secondary care is especially important in view of trends towards a rising incidence of intracerebral haemorrhage in an ageing population.¹

How should intracerebral haemorrhage be distinguished from other causes of stroke?

No clinical scoring system has been shown to reliably differentiate intracerebral haemorrhage from ischaemic stroke.^{w3} Timely brain imaging is the key to recognising intracerebral haemorrhage. Computed tomography detects symptomatic intracerebral haemorrhage within minutes of symptom onset and up to one week thereafter; magnetic resonance imaging with gradient-recalled echo sequences reliably differentiates infarction from haemorrhage more than one week after onset of stroke.⁴ Diagnostic imaging distinguishes intracerebral haemorrhage from other types of intracranial haemorrhage (fig 1), although intracerebral haemorrhage may extend into other intracranial compartments. This distinction is important, because the causes, prognosis, and treatment vary according to the location of intracranial haemorrhage.⁵

SUMMARY POINTS

Spontaneous intracerebral haemorrhage accounts for at least 10% of strokes in the United Kingdom
Half of the patients die within the first month of onset
Stroke unit care improves outcome
Early neurosurgical haematoma evacuation can improve outcome
Secondary prevention by lowering blood pressure is effective

What are the detectable causes of intracerebral haemorrhage?

The major risk factors for spontaneous intracerebral haemorrhage are systemic arterial hypertension, excess alcohol consumption, male sex, increasing age, and smoking.^{6 w4 w5} These risk factors may lead to secondary vascular changes, such as small vessel disease and arterial aneurysms, which may eventually cause intracerebral haemorrhage. Pioneer postmortem studies from the era when non-invasive brain imaging was not widely available suggested that many intracerebral haemorrhages, especially those in deep brain locations, were caused by deep perforating artery lipohyalinosis attributable to chronic hypertension.^{w6} However, a systematic review found a much weaker association between hypertension before a stroke and deep intracerebral haemorrhage.⁷

Systematic investigation of selected patients with intracerebral haemorrhage identifies an underlying arteriovenous malformation in about 20% and an aneurysm in about 13%, so the focus should be on identifying these potentially treatable causes of recurrent intracerebral haemorrhage (table 1).⁸

As a result of the rising use of thrombolytic, antiplatelet, and anticoagulant drugs their association with intracerebral haemorrhage is also increasing,¹ such that many

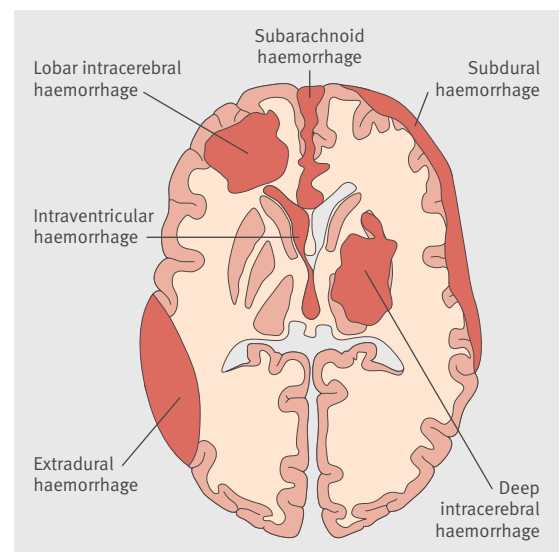


Fig 1 | Axial illustration of the brain showing the subtypes of intracranial haemorrhage

Table 1 | Commonest causes of apparently spontaneous intracerebral haemorrhage

Cause*	Clues
Small vessel disease	Associated with risk factors such as hypertension; leukoaraiosis and lacunes on brain imaging are clues, but only pathological examination is definitive
Amyloid angiopathy	Older patients, without another detected cause for lobar intracerebral haemorrhage; lobar microbleeds are clues, but only pathological examination is definitive
Brain arteriovenous malformation	Extension of intracerebral haemorrhage into other compartments (fig 1); history of intracranial haemorrhage or epileptic seizure(s); calcified or enhancing vessels on imaging
Intracranial arterial aneurysm	Extension of intracerebral haemorrhage into other compartments (fig 1), or located near Sylvian or inter-hemispheric fissures
Cavernous malformation	Personal or family history of intracerebral haemorrhage or epileptic seizure(s); usually small, intracerebral haemorrhage without extension into other compartments
Intracranial venous thrombosis	Associated with pregnancy, thrombophilia, and inflammatory diseases. Intracerebral haemorrhage or haemorrhagic infarcts close to venous sinuses and cortical veins
Dural arteriovenous fistula	Pulsatile tinnitus; haematoma close to venous sinuses and cortical veins
Haemorrhagic transformation of cerebral infarction	Recent cerebral infarction, sometimes followed by a further deterioration
Clotting factor deficiency	Haemorrhages at other sites in the body (skin, joints)
Neoplasm (primary/metastasis)	History or current evidence of a tumour; recently pregnant
Vasculitis	Evidence of systemic vasculitis; lymphocytes in cerebrospinal fluid
Infective endocarditis	Septic embolism into brain arteries, leading to formation of "mycotic" aneurysms
Hypertensive encephalopathy	Evidence of accelerated phase hypertension
Undisclosed trauma	Scalp laceration or skull fracture; widespread contusions on imaging; extension of intracerebral haemorrhage into other compartments (fig 1)

*In descending order of frequency, although the likely cause is thought to depend on the age of the patient and his or her comorbidities and treatment, and on the location of the intracerebral haemorrhage.

patients have several concurrent causes, none of which is either necessary or sufficient to have caused the intracerebral haemorrhage.^{w7}

How should we investigate intracerebral haemorrhage?

After a radiological diagnosis of intracerebral haemorrhage, some routine investigations are essential (box 1), but international guidelines reflect the lack of consensus on which patients to image further, and how and when to do so.^{9,10} Doctors are most likely to further investigate younger patients with intracerebral haemorrhage.⁸ But patient age, comorbidities, and location of intracerebral haemorrhage are unreliable means of predicting cause with certainty,^{7,8} so we recommend further imaging in all patients who can tolerate it and whose prognosis is not bleak (box 1).

Early computed tomography angiography is a quick and widely available first line investigation for an underlying aneurysm or arteriovenous malformation or fistula

when these diagnoses are suspected (table 1), but only a few small studies have investigated its sensitivity (88-100%) and specificity (95-100%) compared with catheter angiography.⁸ Arteriovenous malformations are likely to be under-ascertained in clinical practice because catheter angiography is not used systematically and needs to be repeated to show some arteriovenous malformations.⁸ Magnetic resonance imaging is useful for detecting venous thrombosis acutely and for detecting underlying tumours and cavernous malformations at least two months after the intracerebral haemorrhage.¹¹ Magnetic resonance imaging may also detect some foci of haemosiderin, known as microbleeds, but the diagnostic importance of their detection and distribution is still under investigation.^{12,13}

What is the outcome after intracerebral haemorrhage?

The main predictors of death within one month are older age, low score on the Glasgow coma scale on admission, increasing volume of intracerebral haemorrhage, infratentorial intracerebral haemorrhage location, and intraventricular extension.¹⁴ These five prognostic factors may help to assess the risk of death within one month for individual patients using the total intracerebral haemorrhage score (table 2),¹⁴ which has been externally validated although it may not be as accurate as other scales.^{w8} Intracerebral haemorrhage volume can be estimated easily using the "ABC/2" method. This method entails identifying the axial computed tomography slice with the largest area of intracerebral haemorrhage, and halving the product of its maximum width (A in figure 2), the width perpendicular to A (B in figure 2), and the depth (C, which is determined by multiplying the number of slices on which intracerebral haemorrhage was visible by the slice thickness of the relevant part(s) of the brain computed tomogram).^{w9}

Early neurological deterioration is explained by various mechanisms, including perihematoma oedema and haematoma expansion, which affects

Box 1 | Tests to investigate intracerebral haemorrhage*

Essential

- Full blood count
- Coagulation screen: prothrombin time, activated partial thromboplastin time, d-dimers
- Electrolytes, urea, creatinine, liver function tests
- Glucose
- Inflammatory markers (C reactive protein, erythrocyte sedimentation rate)
- Toxicology screen
- Electrocardiography
- Chest radiography
- Pregnancy test

Dependent on patient characteristics, prognosis, and characteristics of intracerebral haemorrhage

- Computed tomography angiography or venography
- Magnetic resonance imaging
- Catheter angiography

*Adapted from the American Heart Association's guidelines⁹

Table 2 | Scoring system to assess 30 day case fatality* after intracerebral haemorrhage (adapted from Hemphill et al¹⁴)

Component	Score
Glasgow coma scale (at initial presentation or after resuscitation)	
3-4	2
5-12	1
13-15	0
Intracerebral haemorrhage volume (ml) (on initial computed tomography, using the ABC/2 method—see main text for definition)	
≥30	1
<30	0
Any intraventricular haemorrhage on initial computed tomography?	
Yes	1
No	0
Infratentorial origin of intracerebral haemorrhage?	
Yes	1
No	0
Patient's age (years)	
≥80	1
<80	0

*30 day case fatality as percentages (95% CI) as indicated by scores:

Score 1: 13 (5 to 28)

Score 2: 26 (13 to 45)

Score 3: 72 (55 to 84)

Score 4: 97 (83 to 99)

Score 5: 100 (61 to 100)

There were no patients with a score of 6.

about a third of patients within the first 24 hours of onset (figure 2, C and D).^{w10} However, withdrawal of care and “do not resuscitate” orders may have an equally powerful influence.^{w11}

The location and underlying cause of an intracerebral haemorrhage partly determine the long term risk of recurrent haemorrhage, dependence in daily activities,

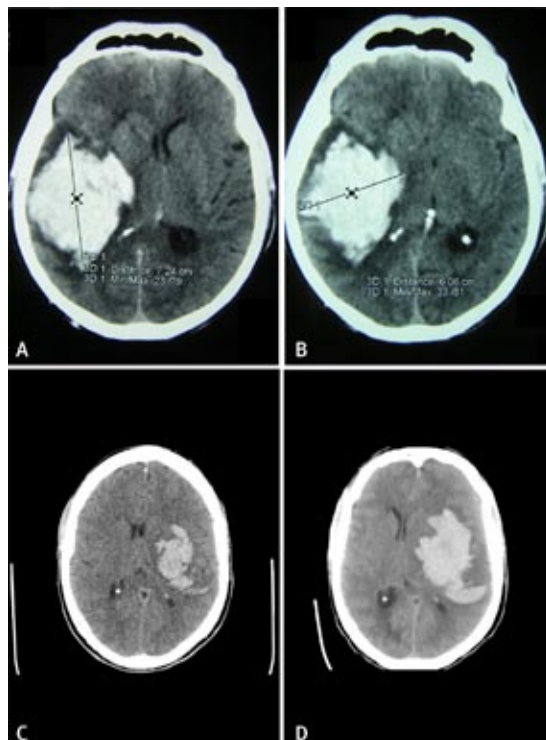


Fig 2 | Location and growth of intracerebral haemorrhage. Lobar right temporoparietal haematoma (A and B, with diameter measurements); deep left basal ganglionic haematoma (C), which expanded in size 24 hours after onset (D)

and death.³ The annual risk of recurrent intracerebral haemorrhage is about 2% for deep intracerebral haemorrhage without an identified cause and about 10% for lobar intracerebral haemorrhage.^{w12} However, the annual risk of recurrent intracerebral haemorrhage from a ruptured arteriovenous malformation varies from about 4% to about 34% according to its vascular anatomy, which is another reason for angiographic investigation of intracerebral haemorrhage.^{3 15} People with intracerebral haemorrhage are also at risk of subsequent ischaemic stroke, at a rate of about 1% a year.^{w13}

How should we treat intracerebral haemorrhage?

General management of stroke

Guidelines and systematic reviews recommend that patients with spontaneous intracerebral haemorrhage should be managed either in a stroke unit, or in an intensive care unit if they need ventilation or intracranial pressure monitoring.^{9 10 16 w14} International guidelines are available for the management of hydration, nutrition, hyperglycaemia, and hyperthermia, prevention of complications, and early rehabilitation.^{9 10} Although the risk of epileptic seizure(s) is higher within the first week of lobar than deep intracerebral haemorrhage (about 14% v about 4%),^{w15} no evidence exists to support the use of prophylactic antiepileptic drugs after intracerebral haemorrhage.^{9 10}

Because of the shortage of high quality evidence on how blood pressure should be managed after acute intracerebral haemorrhage, clinical guidelines recommend various blood pressure reduction regimens.^{9 10} A recent, randomised pilot trial of adults who had a systolic blood pressure 150-220 mm Hg within six hours of onset of intracerebral haemorrhage found intensive blood pressure reduction to be feasible, well tolerated, and associated with a reduction in haematoma growth,^{w16} although clinical benefit remains to be established in ongoing clinical trials (see web extra table on bmj.com). For now, the use of antihypertensive agents seems necessary if there is end organ damage (though the desirable parameters are uncertain),¹⁰ but randomisation in relevant clinical trials is recommended if there is uncertainty.

Small randomised controlled trials have not found significant beneficial or harmful effects from the acute administration of corticosteroids,^{w17} mannitol,^{w18} glycerol,^{w19} or a free radical-trapping neuroprotectant.^{w20}

Haemostatic drugs

Because volume of intracerebral haemorrhage influences outcome and about a third of acute intracerebral haemorrhages enlarge within 24 hours of onset,^{w10} early treatment with a haemostatic drug might improve outcome by limiting expansion of the haematoma. Phase II trials of intravenous recombinant activated factor VII were initially promising (fig 3), although their sample sizes were small and the outcomes for the placebo group were surprisingly poor. Recombinant activated factor VII did not improve clinical outcome in a larger phase III trial; the broader inclusion criteria,

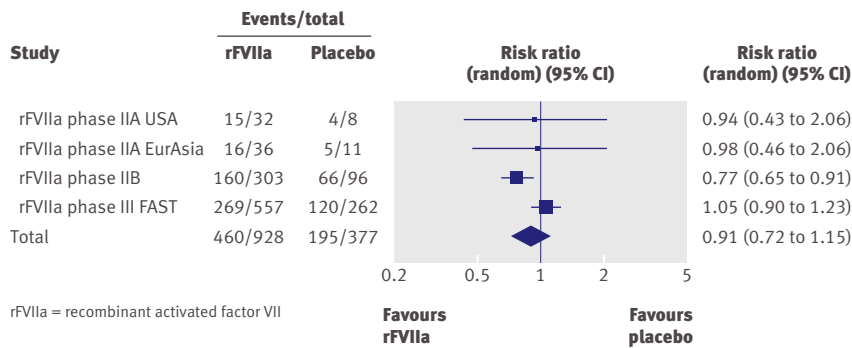


Fig 3 | Forest plot of the effect of recombinant activated factor VII on death or dependence at 90 days after acute spontaneous intracerebral haemorrhage (dependence defined as score 4-5 on modified Rankin scale). Adapted with permission from a Cochrane review¹⁷

problems with randomisation, and preponderance of arterial thromboembolism after recombinant activated factor VII could all explain why this treatment shows no overall clinical benefit in a meta-analysis (risk ratio 0.91, 95% confidence interval 0.72 to 1.15; fig 3).¹⁷

Neurosurgical haematoma evacuation

Evacuation of the haematoma may show the underlying cause in the cavity or lead to the identification of amyloid angiopathy if cortical biopsy is performed, but the dilemma is whether surgery improves outcome.

Infratentorial intracerebral haemorrhage

Guidelines recommend that neurosurgical intervention should be considered immediately for people with a cerebellar haemorrhage if it is causing deterioration in consciousness, brainstem compression, or hydrocephalus as a result of obstruction of the drainage pathways for cerebrospinal fluid (fig 4).^{9 10} Ventricular drainage may be sufficient to alleviate hydrocephalus, but further neurological deterioration requires evacuation of the haematoma.^{9 10} These recommendations are based on case series, in which outcome has been so good that

randomised trials are unlikely to be undertaken.^{w21}

Supratentorial intracerebral haemorrhage

One systematic review found that evacuation of spontaneous supratentorial intracerebral haemorrhage improves outcome (odds ratio 0.71, 95% confidence interval 0.58 to 0.88; fig 5),¹⁸ although another did not.^{w22} However, about 14 patients with supratentorial intracerebral haemorrhage would need to have neurosurgical evacuation for one to avoid death or dependence,¹⁸ and these estimates are not robust because of the modest quality of most of the trials, methodological differences between them, and losses to follow up in the largest trial. A subgroup of patients with superficial lobar intracerebral haemorrhage within 1 cm of the cortical surface seemed to benefit in the STICH trial^{w23} and is being studied further in the STICH 2 trial. Thrombolytic treatment of intraventricular extension from a spontaneous intracerebral haemorrhage is also the subject of ongoing randomised trials (see web extra table on bmj.com).

Treatments for specific causes

Some causes of intracerebral haemorrhage should not be missed because their treatment may improve outcome.

Aneurysms and arteriovenous malformations

One small randomised trial supports immediate evacuation of some intracerebral haematomas caused by aneurysm rupture, with concomitant clipping of the aneurysm.¹⁹ A large randomised controlled trial of coiling versus clipping for ruptured arterial aneurysms that could be occluded by either of these treatments has shown that coiling is less likely to result in death at five years despite a higher risk of rebleeding after coiling.²⁰ Although a primary prevention trial for unruptured arteriovenous malformations is under way (www.arubastudy.org), there are no randomised trials of intervention for ruptured arteriovenous malformations^{w24} or cavernous malformations.

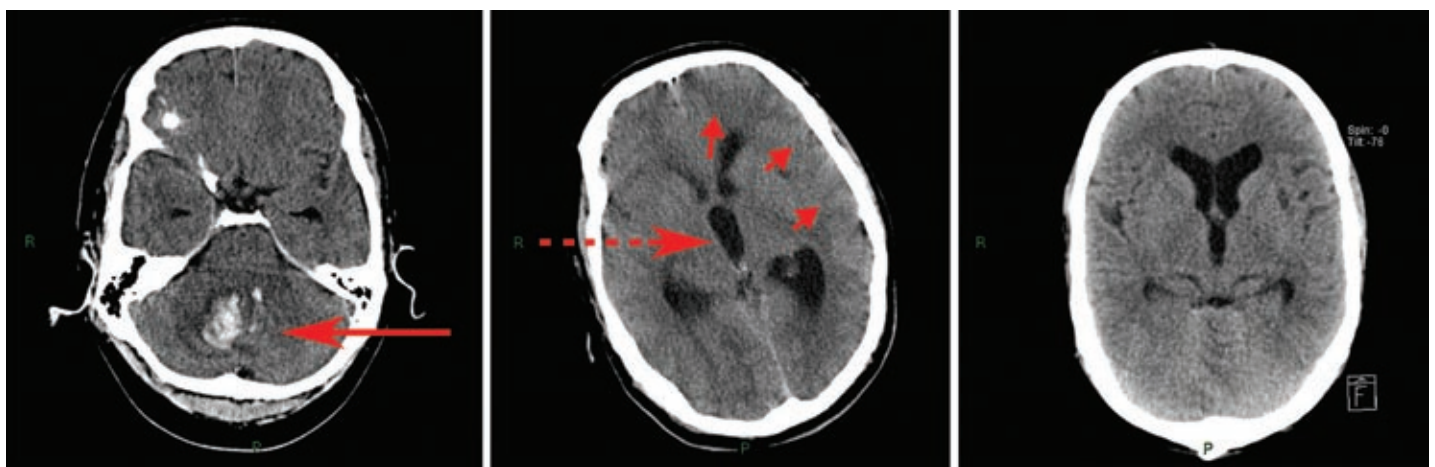


Fig 4 | Infratentorial intracerebral haemorrhage. A 40 year old man presented with sudden headache, vomiting, and unsteadiness. On examination his score on the Glasgow coma scale was 15 and he had nystagmus, limb ataxia, bilateral retinal haemorrhages and papilloedema, a blood pressure of 315/180 mm Hg, and proteinuria. Blood tests showed acute renal failure, and he had immediate brain computed tomography, which showed a cerebellar haematoma adjacent to the fourth ventricle (left, arrow). Three days later, his consciousness level fell rapidly, and repeat brain computed tomography showed an increase in size of the third ventricle (centre, dashed arrow) and effacement of cortical sulci (centre, arrowheads) owing to obstructive hydrocephalus. His consciousness level improved rapidly after ventricular drainage and consequent resolution of hydrocephalus (right)

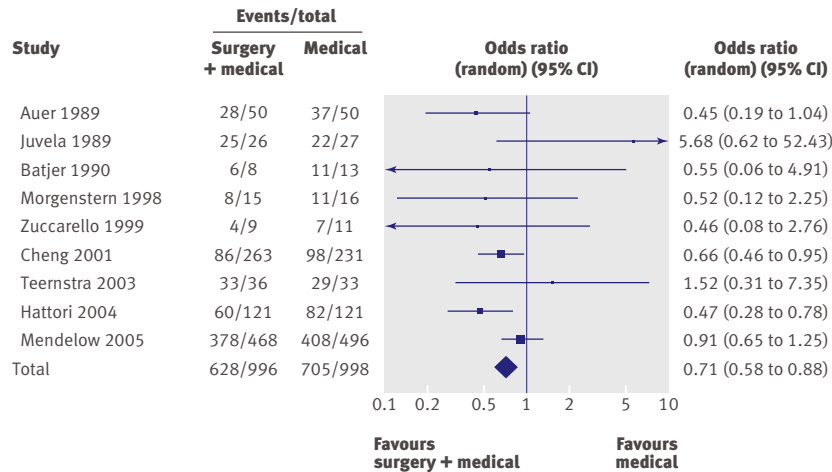


Fig 5 | Forest plot of the effect of neurosurgical evacuation of acute spontaneous intracerebral haemorrhage on death or dependence at the end of follow up (dependence defined as Barthel index <60, score 3-5 on the Rankin scale, or 1-3 on the Glasgow outcome scale. Adapted with permission from a Cochrane review¹⁸

Intracranial venous thrombosis

Data from two randomised controlled trials show a reduction in the risk of death or severe disability after anticoagulation for cortical vein or venous sinus thrombosis.²¹ Although the benefit of anticoagulation was based on relatively small trials, expert opinions favour immediate anticoagulation,²² which does not seem to precipitate or worsen clinically important intracerebral haemorrhage.

Haemorrhage associated with antithrombotic drugs

Guidelines state that when intracerebral haemorrhage occurs in patients taking oral anticoagulants, these drugs should be stopped and their effects urgently reversed, although surprisingly little evidence exists about the best method of doing so.^{9,10} Although intravenous vitamin K is given in most circumstances, it is slow to act, so either prothrombin complex concentrate or fresh frozen plasma are given to immediately replenish vitamin K dependent coagulation factors.¹⁰ The benefits of antiplatelet or even anticoagulant drugs may outweigh their risks after intracerebral haemorrhage for patients at very high risk of myocardial infarction or ischaemic stroke,^{w11 w25} but for now, whether to restart these drugs at 7-10 days after an intracerebral haemorrhage should be decided on a patient by patient basis.

Infective endocarditis

Septic emboli may cause cerebral mycotic aneurysms, which may in turn lead to intracerebral haemorrhage if left untreated.

Other medical treatments

The enthusiasm for medical treatment of acute intracerebral haemorrhage has resulted in several ongoing trials to reduce haematoma expansion (blood pressure lowering, recombinant activated factor VII in subgroups, and platelet infusions for antiplatelet associated intracerebral haemorrhage) or to reduce adverse

consequences of intracerebral haemorrhage (thrombolysis for intraventricular extension of intracerebral haemorrhage, anti-inflammatory drugs, statins, free radical scavengers, and iron chelators).

What about secondary prevention?

Guidelines recommend that survivors of intracerebral haemorrhage should stop smoking and limit their alcohol consumption.^{9,10} A large randomised controlled trial found that after the acute phase of intracerebral haemorrhage a reduction in blood pressure (with an angiotension converting enzyme and a diuretic, if tolerated) was beneficial in preventing future vascular events.^{w26} For an average systolic blood pressure reduction of 12 mm Hg, the risk of recurrent intracerebral haemorrhage may fall by up to 76%.²³

ADDITIONAL EDUCATIONAL RESOURCES (FOR PATIENTS)*

Europe

- Stroke Alliance For Europe (www.safestroke.org)
- Stroke Association (www.stroke.org.uk)
- Chest Heart and Stroke Scotland (www.chss.org.uk)
- Brain and Spine Foundation (www.brainandspine.org.uk)
- German Stroke Foundation (www.schlaganfall-hilfe.de)
- France AVC (www.franceavc.com)

North America

- National Stroke Association, USA (www.stroke.org)
- American Stroke Association (www.strokeassociation.org)
- Heart and Stroke Foundation, Canada (<http://ww2.heartandstroke.ca/splash/>)

Africa

- Heart and Stroke Foundation South Africa (www.heartfoundation.co.za)

Australasia

- National Stroke Foundation, Australia (www.strokefoundation.com.au)
- Stroke Foundation of New Zealand (www.stroke.org.nz)

Asia

- Japan Stroke Society (www.jsts.gr.jp)

*The organisations offer a range of services including support for patients, families, and carers; information leaflets and booklets; welfare grants; telephone and online advice lines; discussion groups

TIPS FOR NON-SPECIALISTS

- Computed tomography reliably distinguishes cerebral infarction from haemorrhage within minutes and for up to seven days after onset of symptoms
- Magnetic resonance imaging (including gradient-recalled echo sequences) is usually required to reliably detect haemorrhage more than one week after onset of stroke
- The early prognosis is poor, so seek specialist advice quickly
- Infratentorial haemorrhage causing a declining level of consciousness, brainstem compression, or hydrocephalus requires immediate neurosurgical referral
- Underlying causes meriting immediate consideration of specific treatment include anticoagulant drugs, uncontrolled hypertension, arterial aneurysms, and venous thrombosis

SOURCES AND SELECTION CRITERIA

We referred to the Cochrane database of systematic reviews and the published guidelines in September 2008, and we used our personal reference collections.

Conclusion

Randomised trials, systematic reviews, and international guidelines find that stroke units and secondary prevention with blood pressure reduction benefit people with intracerebral haemorrhage. Unfortunately, randomised trials of acute medical and surgical interventions do not conclusively support their routine use in clinical practice. Because the outcome after intracerebral haemorrhage is still extremely poor, ongoing trials are reason for optimism (see web extra table on bmj.com), and should be advocated in clinical practice.^{24 25}

Contributors: RA-SS searched the literature and drafted the article, and every author revised it critically for important intellectual content. All authors gave final approval of the final manuscript. RA-SS is the guarantor.

Competing interests: None declared.

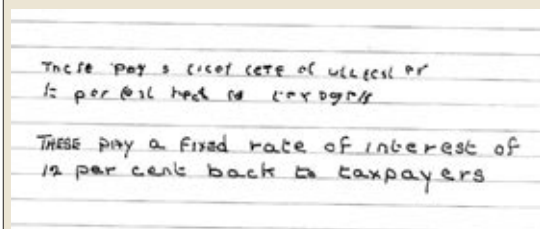
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Patient consent obtained.

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A clear vision of our finances

A 74 year old diabetic patient of mine came to see me yesterday for her annual ophthalmic review. She said her right eye had become blurry again over the past two months. She has mature onset diabetes of 15



Sentence as copied by patient using only her right eye (top) and her left eye (bottom)

years standing and had macular oedema in her right eye last year with vision down to 6/60. I had given her intravitreal triamcinolone at that time as I couldn't see any obvious areas to laser and she had surprisingly had an improvement to 6/9 for about nine months. Now her vision in this eye was blurry again due to macular oedema.

To help me, she had copied a sentence out of the *Daily Mail* as she saw it with her right eye and her left eye (figure).

I told her that neither of these statements made any sense and have sent her for a psychiatric consultation. She agreed with me and quietly left.

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