Why there's no point telling me to lose weight

In the first of a new series in which patients and carers set the learning outcomes for readers, **Emma Lewis** tells her story. For more information about the series, contact Rosamund Snow, patient editor, rsnow@bmj.com

I am one of over 97% of people for whom dieting does not lead to sustained weight loss.[1-3]

I've experienced health benefits from increased exercise, and from switching to a wholemeal vegetarian diet. My blood pressure's normal, as are my fasting glucose and my lung function—as far as I can tell, my health is great. But my body mass index (BMI) has been above 30 my entire adult life.

When I worry that I might be unwell, I often try to avoid visiting a general practitioner. Almost every consultation I've ever had—about glandular fever, contraception, a sprained ankle—has included a conversation about my weight; and that's inevitably the conversation that destroys any rapport or trust that might have existed between me and my doctor.

Fighting "the obesity epidemic" is supposed to be about making me—as a "severely obese" person—more healthy; but the impact of obesity rhetoric on my life has been quite the opposite.

I've been out dancing in some slightly inadvisable shoes. On the walk home, I step awkwardly in a gutter and hurt my ankle. The next morning, the swelling is pretty severe, so I decide I ought to get it checked out.

The doctor tells me that I should be exercising more. I say: I know that increased circulation boosts healing, but as it currently hurts to stand I'm not sure what it's best to do for exercise. He says: he's not talking about healing up the ankle, he means, in general.

He hasn't asked me how much exercise I already do. He doesn't know that just last night I danced energetically for four hours then walked several miles home. I assume that he tells all his fat patients the same thing, without bothering to find out about their individual situations. This doesn't exactly fill me with trust that I'm receiving responsible medical advice. I don't visit this practice again.

I have been fat my whole life. So when healthcare professionals ask me—in the middle of a consultation about something completely unrelated—whether I know that my BMI is too high and whether I'm engaged in any weight management, I'm always a little surprised when they act like they might be the first to have ever brought it up. As if I might have made it through my 30 years without ever once noticing that I was fat and that some people think that fat is bad.

It's just a little reminder that my GP—like many other people in the world—sees me as a fat person first, and an individual second. It makes me feel like a problem to be solved—something unpleasant that needs to be eliminated.

I recently took up weightlifting. I'm happier in myself now—my stamina has increased, as has my strength; I can cycle up hills that used to defeat me.

Unfortunately, building up enough muscle mass to squat a 100 kg barbell has tipped my BMI over from "obese" to "severely obese." I haven't been back to a GP since, but I'm dreading it more than ever.

When health professionals bring up my weight in a consultation, I don't feel like they're looking out for my health. They give me the impression that my weight is the most important thing about me—more important than, say, my penchant for body piercing and platform shoes, both of which have caused me more infection and injury than my adipose tissue has. They put me right back to where I was when I was a binging-fasting teenager: full of shame.

My childhood contained so many diets, so many humiliations in school PE

They tell me that my body type is a "risk factor" for all kinds of diseases, and that statistically I'm more likely to be healthy if I lose weight. I might query the science behind that supposition—citing the "obesity paradox," which indicates that fat people have better survival rates than thin people for all sorts of diseases,[4-6] but I do accept that it's orthodox medical opinion.

Even if I did want to change my body type to be less of a "risk factor"—it's not that easy. I'm already physically active well beyond the recommendations of the chief medical officer,[7] and I don't rate my chances of being one of those seemingly mythical people who manage to maintain weight loss through dietary intervention.[1-3]

My childhood contained so many diets, so many humiliations in school PE (physical education). No attempts to make me lose weight have ever had any long term effects. All they did was give me a constant sense of shame and of not being good enough. This led to unhealthy eating habits that would have been labelled "disordered" in someone with a lower BMI. It has taken me years to unlearn those habits. And it's only recently that I've really discovered the joys of physical exertion, having spent most of my life thinking of exercise as "that punishment I get given for being fat"—impact based activities like running are physically painful for someone with my body type.

I've opted out of the weight loss game. If that makes me a non-compliant patient, then so be it. I'm healthier and happier than I was when I hated myself. I just wish that my healthcare providers would work with me on that.

KEY MESSAGES:

1. Focus on what the patient has come to see you about today. If you only do that, you've done a good job. Think twice before offering unsolicited advice in the guise of "education," particularly when your patient is consulting you about something unrelated.
2. It is appropriate to give diet or exercise advice when somebody asks you directly, but try to focus on the other benefits of eating well and getting regular exercise, rather than treating weight loss as an end in itself.[8] That way your patients won't get discouraged from healthy behaviours even when they do not result in permanent weight loss.
3. Fat people know that they are fat. You don't need to tell us; society's been making us feel bad about it our whole life.

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CPD/CME QUESTIONS

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- This author directly questions the role of the healthcare professional with respect to conventional health promotion. What is your reaction to that?
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PRACTICE POINTER

Is it a stroke?

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CPD/CME

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Stroke is increasingly common and often fatal or disabling.[1] The absence of a definitive diagnostic test for stroke and the potential for emergency interventions to restore brain perfusion,[2] improve survival free of handicap, and minimise early recurrent stroke[3] mean that doctors need to be able to diagnose acute stroke rapidly and accurately.

What is a stroke?

Stroke is not consistently defined in clinical practice, clinical research, and public health. Traditionally, stroke has been defined clinically by the abrupt onset of symptoms of focal neurological dysfunction that last more than 24 hours (or lead to earlier death) and are caused by acute vascular injury to part of the brain.[4] The vascular causes include inadequate blood supply to part of the brain or spinal cord (ischaemic stroke, arterial or venous) and spontaneous haemorrhage into part of the brain (primary intracerebral haemorrhage) or over the surface of the brain (subarachnoid haemorrhage).

Advances in technology have prompted an updated definition of stroke as an acute episode of focal dysfunction of the brain, retina, or spinal cord of any duration in which imaging (computed tomography or magnetic resonance imaging) or autopsy show focal infarction or haemorrhage relevant to the symptoms.[5] This definition awaits endorsement, particularly for regions without access to magnetic resonance imaging.[4 6]

How do patients with acute stroke present?

Stroke presents in a variety of ways.

Typical presentations: focal neurological symptoms of sudden onset

Typically, stroke presents spontaneously with the sudden or rapid onset of loss of function of a particular part(s) of the body due to loss of function of a particular part of the brain, retina, or spinal cord.

Common focal, anatomically-localising neurologic symptoms include unilateral weakness (corticospinal tract), unilateral sensory loss (spinothalamic tract), monocular blindness (retina or optic nerve), hemianopic visual field loss (optic radiation), double vision (oculomotor pathways), speech disturbance (dominant hemisphere), visual-spatial-perceptual dysfunction (non-dominant hemisphere), clumsiness or ataxia (cerebellum or its connections), and vertigo (vestibulocerebellum), which may appear in isolation or combination.

Associated symptoms vary and usually reflect the cause or a consequence of the stroke. For example, headache occurs in about a quarter of patients with acute ischaemic stroke, half of patients with intracerebral haemorrhage, and nearly all patients with subarachnoid haemorrhage, and may reflect the underlying cause of the stroke (such as cervical artery dissection, giant cell arteritis) or a consequence of the stroke (such as cortical ischaemia, intracranial haemorrhage).[4]

Atypical presentations: stroke "chameleons"

Less commonly, patients have atypical stroke symptoms (stroke "chameleons") that imitate other neurological diseases. This is because the symptoms

- Are not anatomically localising (such as neuropsychiatric, confusion, depressed consciousness)
- Are positive (such as abnormal movements, rather than paralysis, due to seizure, alien hand syndrome, or hemiballismus)
- Seem to be peripheral nerve in origin (vestibular syndrome, other cranial nerve palsy, cortical hand syndrome, monoparesis)
- Are isolated (isolated vertigo, binocular blindness, amnesia, anosognosia, dysarthria, dysphagia, stridor, foreign accent, or headache).[7 8]

Such atypical symptoms are more likely to be due to a stroke if the patient has known cardiovascular disease or risk factors.

Subarachnoid haemorrhage

This typically presents with a sudden, severe diffuse headache in nearly all patients,[4] usually without focal neurological symptoms or signs. Neck stiffness is not invariable and may not occur for hours. Other features include vomiting (75%), depressed consciousness (67%), focal neurological dysfunction (15%), intraocular subhyaloid haemorrhages (14%), epileptic seizures (7%), delirium (1%), radicular or precordial pain (spinal subarachnoid haemorrhage), severe hypertension, and electrocardiographic changes that may mimic acute myocardial infarction.[9]

A headache may not be present in patients with subarachnoid haemorrhage and depressed consciousness. Also, other symptoms may prevail over headache in patients with subarachnoid haemorrhage who have a confusional state, epileptic seizure, or associated head trauma.

Cerebral vein thrombosis

Cerebral vein and venous sinus thrombosis typically presents with gradual onset headache followed by focal neurological

THE BOTTOM LINE

- Suspect the diagnosis of stroke in all patients with abrupt onset of neurological symptoms, particularly in those with risk factors for stroke. Early and accurate diagnosis of stroke enables early interventions targeted to the cause, which may improve survival and functional recovery and minimise early recurrent stroke.
- Some stroke patients will present with atypical stroke symptoms in which the symptom onset is not sudden or the loss of neurological function is not clearly anatomically-localising.
- The FAST (Facial drooping, Arm weakness, Speech difficulties and Test (or Time)) score is a useful screening test in the community, while emergency department doctors may use the FAST or ROSIER scales, and stroke physicians will undertake a more complete and systematic neurovascular assessment.
- Stroke lacks a perfect diagnostic test, and current diagnosis relies on clinical history and examination, supported by brain imaging (such as computed tomography and magnetic resonance imaging, which can be normal).
- Seizures, syncope, and sepsis account for 20-25% of suspected strokes.

Box 1 | Face Arm and Speech Test (FAST)

FAST is used as a mnemonic to help detect stroke and facilitate an appropriate response. It stands for Facial drooping, Arm weakness, Speech difficulties, and Time.

Face—Ask the person to smile. Does one side of the face droop?

Arms—Ask the person to raise both arms. Does one arm drift downward?

Speech—Ask the person to repeat a simple phrase. Is the speech slurred or strange?

Time—If you observe any of these signs, call for an ambulance immediately

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- Acute nasal injury (*BMJ* 2014;349:g6537)
- Complex regional pain syndrome (*BMJ* 2014;348:g3683)
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- Serotonin syndrome (*BMJ* 2014;348:g1626)
- Assessing risk of suicide or self harm in adults (*BMJ* 2013;347:f4572)

deficits, epileptic seizures, or impairment of consciousness, in different combinations and degrees of severity.[10] In up to 15% of patients however, the onset of the headache is sudden ("thunderclap headache").

How is stroke diagnosed?

Early recognition

The Face Arm and Speech Test (FAST) is a simple, three item recognition tool to aid screening for stroke in the community (box 1). The presence of acute facial paresis, arm drift, or abnormal speech increases the likelihood of stroke (likelihood ratio of >1 finding = 5.5 (95% confidence interval 3.3 to 9.1)), while the absence of all three decreases the likelihood (likelihood ratio of 0 findings = 0.39 (0.25 to 0.61)).[11] Paramedics using FAST achieve high levels of detection and diagnostic accuracy of stroke[12] but may miss posterior circulation strokes[13] and treatable stroke mimics such as hypoglycaemia.

Emergency department clinicians may opt for the Recognition of Stroke in the Emergency Room (ROSIER) score, which comprises the three FAST items plus visual field defect, leg weakness, loss of consciousness or syncope and seizure activity (see online data supplement).[14] An initial study among emergency department physicians showed increased diagnostic sensitivity compared with FAST,[14] although a later study showed similar sensitivities and specificities for both.[15]

Clinical diagnosis

Stroke is diagnosed clinically on the basis of a description of sudden onset of loss of focal neurological function that is thought to be due to disturbed blood supply to the relevant part of the brain, retina, or spinal cord. If these criteria are met, the likelihood of a stroke is high because stroke is common (that is, high prevalence or pre-test probability), and even higher if the "milieu" is appropriate (such as an elderly patient with a history of prolonged exposure to vascular risk factors).

Symptoms associated with a high agreement between observers for the diagnosis of stroke versus no vascular event are sudden paralysis or weakness, numbness or tingling, change in speech, visual loss, diplopia, and non-orthostatic dizziness (kappa = 0.60 (95% confidence interval 0.52 to 0.68)).[11]

An observational study of 350 presentations with suspected stroke found that eight items in the clinical bedside assessment independently predicted the diagnosis of stroke (see table).[16-18] Many of these predictors have been validated in other studies.[19]

If the patient is seen within 24 hours of symptom onset, and if the neurological symptoms are still present at the time of the assessment and thought to be vascular in origin, the patient should be managed as if he or she has had a stroke, rather than diagnosing a transient ischaemic attack and predicting that the symptoms will resolve within 24 hours.

Independent predictors of the diagnosis of stroke among 350 presentations by 336 patients with suspected stroke to an urban teaching hospital in the UK, of which 241 (69%) were given a final diagnosis of stroke and 109 (31%) a diagnosis of stroke mimic[16]

Variable	Odds ratio (95% CI)
Higher odds of stroke	
An exact onset could be determined	2.59 (1.30 to 5.15)
Definite history of focal neurological symptoms	7.21 (2.48 to 20.93)
Any abnormal vascular findings*	2.54 (1.28 to 5.07)
NIHSS†:	
0	1.0 (reference)
1–4	1.92 (0.70 to 5.23)
5–10	3.14 (1.03 to 9.65)
>10	7.23 (2.18 to 24.05)
Signs could be lateralized to left or right side of brain	2.03 (0.92 to 4.46)
OCSP classification was possible‡	5.09 (2.42 to 10.70)
Lower odds of stroke	
Known cognitive impairment	0.33 (0.14 to 0.76)
Abnormal findings in any other system§	0.44 (0.23 to 0.85)

*Systolic blood pressure >150 mm Hg, atrial fibrillation, valvular heart disease, or absent peripheral pulses.
†The NIHSS (National Institute of Health Stroke Scale) is a scale of neurological impairments such as level of consciousness, ocular gaze, visual fields, speech and language function, inattention, motor and sensory impairments, and ataxia that is used to grade stroke severity (not diagnose stroke or its mimics); a higher score reflects a greater number and severity of neurological impairments.[17]
‡The OCSP (Oxfordshire Community Stroke Project) classification comprises four clinical stroke syndromes (total anterior circulation syndrome, partial anterior circulation syndrome, lacunar syndrome, posterior circulation syndrome) that are based on clinical features of the stroke.[18]
§Respiratory, abdominal, or other abnormal signs.

Investigations

The diagnosis of stroke is confirmed by computed tomography or magnetic resonance imaging of the brain or cerebrospinal fluid examination for subarachnoid blood.[5]

Non-contrast computed tomography (CT) of the head

This is the initial diagnostic standard because it is widely and rapidly available and has near perfect sensitivity for acute intracranial haemorrhage. Ischaemic stroke may not be apparent on the initial CT scan, although subtle signs of early ischaemia are usually evident. The sensitivity of CT for diagnosing acute ischaemic stroke is limited if the focal ischaemia is recent (minutes to hours), small, or in the posterior fossa. The sensitivity of CT for diagnosing subarachnoid haemorrhage is limited if it is small or the CT scan is delayed, which allows time for the subarachnoid blood to degrade. The sensitivity of CT is 98% for subarachnoid haemorrhage within 12 hours, 93% at 24 hours, and declines rapidly after 10 days.

Lumbar puncture and cerebrospinal fluid examination (CSF) examination for subarachnoid blood

This is required if SAH is being considered and the CT scan is non-diagnostic.[9] However, the prevalence of xanthochromia in the CSF also declines with time and is detected in only 70% of cases of subarachnoid haemorrhage after three weeks and 40% after four weeks.

Magnetic resonance imaging (MRI)

Diffusion weighted MRI is more sensitive than CT scanning for detecting acute cerebral ischaemia, particularly in patients presenting within 12 hours of symptom onset.[20 21] A meta-analysis of eight studies involving 308 participants reported that the sensitivity of diffusion weighted MRI for diagnosing acute ischaemic stroke was 0.99 (95% confidence interval 0.23 to 1.00) and the specificity was 0.92 (0.83 to 0.97); the summary estimates for CT were sensitivity 0.39 (0.16 to 0.69) and specificity 1.00 (0.94 to 1.00).[21] A caveat of this meta-analysis is that the reported high

Diffusion weighted MRI is more sensitive than CT scanning for detecting acute cerebral ischaemia, particularly in patients presenting within 12 hours of symptom onset

sensitivity of MRI was obtained in patients with a high pre-test probability of stroke (such as major stroke).[21] Subsequent studies in patients with minor ischaemic stroke (who tend to have a lower pre-test probability of stroke) have reported the sensitivity of diffusion weighted MRI to be only 76% (71 to 81%).[22] Therefore, a negative diffusion weighted MRI result does not exclude the diagnosis of stroke, particularly in patients with minor ischaemic stroke.[22] Further, a positive diffusion weighted MRI result does not confirm the diagnosis of stroke[23 24]; it may be due to non-stroke conditions such as seizures, migraine, hypoglycaemia, tumour, encephalitis, abscess, and multiple sclerosis.

Gradient echo T2-weighted susceptibility MRI is as sensitive as CT for acute haemorrhage and is more sensitive for previous haemorrhage. MRI venography or CT venography is usually required to confirm the diagnosis of cerebral vein and venous sinus thrombosis.

Box 2 | The 20 most common conditions that may mimic stroke, identified in a systematic review and meta-analysis of cases series[8 25]

Seizure (20%)
- Positive symptoms (limb jerking, head turning, posturing, lip smacking)
- Brief (‹2 minutes) usually
- Loss of awareness and amnesia for event unless simple partial seizure
- Post-ictal negative symptoms (such as Todd's paresis) may persist for 1-2 days
- History of epilepsy, recurrent similar seizures, or previous cortical injury

Syncope (15%)
- Faint or light headed before syncope; vision may darken or hearing muffle
- Brief (‹1 minute) loss of awareness with rapid recovery to full alertness
- Associated nausea, pallor, sweating
- Sepsis (12%)
- Systemic sepsis may exacerbate neurological deficits from a prior stroke
- Associated fever and raised inflammatory markers
- Sepsis may also cause stroke: hypercoagulability, infective endocarditis

Functional (9%)
- Usually a trigger (such as panic attack, dissociative episode, emotional or psychosocial stressors and anxiety)
- Isolated non-anatomical sensory symptoms common
- Positive features of functional disease more important than lack of features of organic disease: for example, inconsistency (task dependent weakness, can't move leg but can walk) and positive Hoover's sign
- Tend to be recurrent and stereotyped

Primary headache disorder (such as migraine) (9%)
- Positive symptoms that build up and spread (such as visual to somatosensory)
- Duration usually 20-30 minutes but may last hours
- Associated nausea, vomiting, photophobia, phonophobia with or without headache
- Family history of migraine

Brain tumour (7%)
- 5% present rapidly (haemorrhage, seizure, oedema, hydrocephalus)

Metabolic (6%)
- Hypoglycaemia (due to insulin treatment, sulphonylureas, alcohol, Addison's disease, or insulinoma) can present with focal neurological symptoms and signs alone

Peripheral vestibular disorder (4%)

Neuropathy (4%)

Dementia (3%)

Extradural or subdural haemorrhage (2%)

Drugs and alcohol (2%)

Transient global amnesia (2%)

Other diagnosis (6%)

Investigation sequence

Thus when assessing a patient with suspected acute stroke, an urgent plain CT brain scan often identifies an area of acute focal brain ischaemia and almost always identifies an area of acute focal brain or subarachnoid haemorrhage. If the CT does not show either, then undertake an MRI to identify early infarction (there is no need to specify MRI sequences such as diffusion weighted or gradient echo), or lumbar puncture and CSF examination if subarachnoid haemorrhage is suspected.

To seek the cause of the stroke, imaging of the relevant carotid or vertebral arterial system by carotid ultrasound or CT or MRI angiography, and heart and aortic arch by echocardiography, may be required.

If the patient's symptoms have resolved quickly because of a probable transient ischaemic attack and the CT scan is unremarkable, subsequent tests are still needed to find the underlying cardiovascular cause of the transient ischaemia (such as carotid stenosis or atrial fibrillation) and to treat that urgently to prevent recurrent transient ischaemic attack or fatal or disabling stroke.

What other conditions may mimic a stroke and lead to "overdiagnosis" of stroke?

Among patients presenting with a typical stroke syndrome, up to 20-25% will have a stroke mimic (box 2).[8 25] Unfortunately, brain imaging alone does not always distinguish stroke from its mimics, and other investigations may be required to exclude the differential diagnoses shown in box 2.

Among patients presenting with other neurological syndromes, such as isolated vertigo, stroke may be the underlying cause. A common example is the acute vestibular syndrome (isolated vertigo with secondary ataxia and nausea), which may have a peripheral cause (such as vestibular neuritis) or a central cause (such as vestibulo-cerebellar stroke). A focused history and physical examination are often required to diagnose acute vestibular syndrome due to a vertebrobasilar stroke because the lesion is often too small to be seen on brain imaging.[26] A three-step bedside oculomotor examination (HINTS: Head Impulse test, Nystagmus, Test of Skew) to elicit a normal horizontal head impulse test, direction-changing nystagmus in eccentric gaze, or skew deviation (vertical ocular misalignment) identifies stroke with high sensitivity (100%) and specificity (96%) in patients with acute vestibular syndrome and is more sensitive than early MRI.[27]

References and competing interests are on thebmj.com.

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