REVIEW

Silent witnesses diagnosis of epilepsy

Epilepsy is primarily a clinical diagnosis that depends on the patient's account and – importantly – an accurate witness description of the attacks in the event of loss of awareness, consciousness or recall of the events. Unfortunately, not all 'blackouts' (loss of consciousness) are witnessed. So are there any useful clues – silent witnesses – that can tell us about the nature of a blackout under these circumstances?

CONSIDER FOUR PATIENTS

Patient A: a 44-year-old man wakes in the morning feeling non-specifically unwell and notices that he has bitten the side of his tongue.

Patient B: a 36-year-old woman wakes in the morning feeling groggy and achy with mid-thoracic back pain, having gone to bed completely well the night before. Because of her back pain she at-

tends her family doctor who arranges a plain X-ray, which shows a thoracic vertebral compression fracture (Fig. 1).

Patient C: a 64-year-old man wakes in the morning with right shoulder pain on movement, and some limitation in movement. He also has a mild generalized headache. He had gone to bed late but had felt perfectly well. He had not fallen. A plain X-ray reveals a posterior fracture dislocation of his shoulder (Fig. 2).

Patient D: a 33-year-old woman remembers nothing after standing alone in the kitchen until she regained consciousness on the floor, unsure how long she had been unconscious for, but she found she had wet herself.

Do any of these 'silent witnesses' (bitten tongue, spinal compression fracture, posterior shoulder fracture dislocation, and urinary incontinence) tell us what happened? In particular, do they indicate whether the patient had had an epileptic seizure?

Figure 1 Lateral and AP xray showing compression fracture of a thoracic vertebra (arrows).

Seizures in the epilepsy

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To answer these questions we need to consider how often these phenomena occur during seizures, which will tell us how sensitive they are in making a diagnosis of epilepsy; and how often they occur at times other than during a seizure, and if so in what situations, which will tell us something about specificity.

TONGUE BITING

“As the muscle spasms become more shock like... the stage of clonic spasm is reached, in which the limbs, head, face and trunk are jerked with violence, and through similar spasms in the tongue and the muscles of the jaw the former is often bitten.” (Gowers 1893)

Gowers recognized that tongue biting was a complication of a convulsive seizure. But how sensitive and specific a sign is it in

practice (Table 1)? One study of consecutive patients assessed for ‘blackout’ found that of those ultimately thought to have had a seizure 41% had bitten their tongue compared with only 6% of those who were thought to have had blackouts of other types (Hoefnagels *et al.* 1991). The authors did not specify what part of the tongue was bitten. In another study (Benbadis *et al.* 1995), of patients admitted to an epilepsy monitoring unit, 63 had episodes of loss of consciousness – 34 with epileptic seizures, and 29 had exclusively non-epileptic episodes. Eight patients suffered oral laceration, all involving the *side of the tongue* and in all these cases the patients had documented epileptic seizures. Of 45 patients with syncope, only one lacerated the tongue and this was at the tip.

These studies indicate that tongue biting is not a sensitive feature of epileptic seizures, but that biting the *side* of the tongue is a very specific one – at least as far as these small studies can tell us.

VERTEBRAL FRACTURE

Seizures have long been recognized as a cause of vertebral fracture. Electroconvulsive therapy, introduced in the 1930s, is also a recognized cause (Mattson & Gidal 2004). Vasconselos (1973) studied X-rays of 15 of 1487 patients with epilepsy complaining of back pain and all 15 had thoracic compression fractures. Most of the fractures had occurred during sleep, making trauma an unlikely cause. In one very large study that found a doubling of the

Figure 2 Xray to show posterior fracture dislocation of shoulder.

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Table 1 The sensitivity and specificity of tongue biting in epileptic seizures

STUDY	SEIZURE	NOT SEIZURE	SENSITIVITY	SPECIFICITY
Hoefnagels <i>et al.</i> (1991)	17/41 (41%)	3/53 (6%)	41%	94%
Benbadis <i>et al.</i> (1995)	8/34 (24%)	Syncopal: 1/45 (2%) Non-epileptic attack: 0/29	24%	98%

rates of all types of fracture in patients with epilepsy, the specific rate for vertebral fractures was not given (Souverein *et al.* 2005).

However, non-traumatic vertebral fractures occur commonly in the normal population over the age of 50 years, increasing with age, related to osteoporosis. As such these fractures occur more commonly in women than men: age 50–54, women 4% per year, men 1%; age 70–74 women 18% per year, men 9% per year (Cummings & Melton 2002). Fractures in younger patients are less common.

Thoracic vertebral fractures have been reported as a presenting feature of previously undiagnosed epilepsy (Aboukasm & Smith 1997). Traumatic fracture often affects the lumbar spine rather than thoracic spine, therefore thoracic compression fractures may be an indicator of an underlying epileptic seizure disorder. However, epilepsy is very clearly not the only cause of fractures.

Whilst there is evidence that epileptic seizures can produce vertebral fractures, the specificity of this finding is unknown. It is so common in normal people that the specificity cannot be very high. However, the specificity could be studied within the particular context of a patient who has had a ‘blackout’ – and determine how specific a vertebral fracture in that situation is for a seizure. This may validate vertebral fracture as another silent witness.

However, you do need to be aware that if a patient with known epilepsy wakes with mid-back pain, imaging should be considered because he or she may have sustained a vertebral compression fracture.

SHOULDER DISLOCATION

Posterior dislocation of the shoulder is rare, representing less than 3% of all shoulder dislocations (Robinson & Aderinto 2005). Posterior fracture-dislocations are even rarer. Typically the shoulder is painful and held in internal rotation, with the most consistent finding being mechanical block to external rotation (Robinson & Aderinto 2005). The diagnosis is difficult; 50% of posterior dislocations are missed when the patient is first assessed (Gosens *et al.* 2000; Robinson & Aderinto 2005). In one series, 20 of 41 posterior dislocations were not seen on standard anterior-posterior and lateral Xray views (Hawkins *et al.* 1987). Imaging requires

modified views, axillary if the patient can tolerate it or the 20/20 oblique view, or other modalities such as CT (Buhler & Gerber 2002; Robinson & Aderinto 2005).

Posterior dislocation occurs in three situations: during an epileptic seizure, as a result of high-energy trauma, and following electrocution – the last two readily identified in the history (Gosens *et al.* 2000; Brakestone *et al.* 2001; Robinson & Aderinto 2005). About 15% of posterior dislocations are bilateral (Robinson & Aderinto 2005), an injury that has been said to be pathognomonic of a seizure (Shaw 1971). In a review, 31 of 35 reported patients with bilateral posterior dislocations were related to seizures, two extreme trauma and two domestic electrocution (Brakestone 2001); this triad was termed the ‘Triple E’ syndrome – epilepsy, electrocution and extreme trauma. Posterior dislocation in the absence of significant trauma or electrocution is specific, and bilateral posterior dislocation highly specific for an epileptic seizure. It is difficult to calculate sensitivity for posterior shoulder dislocation – it is a rare complication and so inevitably an insensitive feature for seizures.

The mechanism of posterior dislocation/fracture during a seizure was described by Shaw (1971). The power of the infraspinatus, teres minor, deltoid, latissimus dorsi and teres major muscles is sufficient to dislocate the shoulder posteriorly. After the seizure, the head of the humerus lies behind the glenoid fossa. If the seizure is prolonged, the anatomical neck of the humerus can become impacted against the rim of the glenoid fossa and the fragments drawn apart by the subscapular and infraspinatus muscles. Posterior dislocation occurs more commonly in men than in woman, perhaps as a result of their more powerful musculature (Robinson & Aderinto 2005).

The diagnosis of posterior shoulder dislocation may be difficult because of the patient’s postictal confusion and generalized aching after a seizure. As shoulder dislocation can cause considerable morbidity, in addition to being considered a possible silent witness to an underlying seizure disorder, you need to be aware of the possibility of posterior dislocation in patients known to have epilepsy. Imaging should be considered in patients with epilepsy who complain of pain or restricted range of movement of the shoulder.

Whilst posterior dislocation is a specific indicator of seizures in the absence of trauma or electric shock, anterior dislo-

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Table 2 Sensitivity and specificity for incontinence as a feature of an epileptic seizure (Hoefnagels *et al.* 1991)

	SEIZURE	NOT SEIZURES
Incontinent (21)	7 (33%)	14 (67%)
Non incontinent (63)	34 (54%)	29 (46%)

Sensitivity = 17%. Specificity = 67%.

cation, which is much more common in other circumstances, can also occur. Buhler & Gerber (2002) retrospectively studied 34 unstable shoulders in 26 patients in whom the initial dislocation had been caused by an epileptic seizure. Anterior and posterior dislocation occurred with equal frequency, each occurring in 17 shoulders from 13 patients. Given the relative rarity of posterior dislocation in other settings, this further suggests a significant over-representation of this dislocation following seizures.

DeToledo & Lowe (2001) reported shoulder dislocation in 5 of 806 epilepsy inpatients in whom video recordings showed staff placing them in the lateral decubitus position during a seizure. In all cases the dislocated shoulder was on the lower side, and there is an implication that these were anterior dislocations. The authors argued that as the risk of aspiration is greater in the postictal period than during a seizure, patients should either be positioned when the seizure has ceased, or that secretions should be managed by vacuum aspiration. This recommendation has now been incorporated into UK first aid guidelines

CONCLUSIONS

- The diagnosis of epilepsy and its differentiation from other causes of blackouts (loss of consciousness) can be very difficult without a reliable witness history.
- Whilst not compensating for an accurate witness description (which should be energetically sought), 'silent witnesses' can help in deciding whether an event was an epileptic seizure.
- Bilateral posterior dislocation of the shoulder (unless there is a history of high energy trauma or electrocution), is pathognomonic for an epileptic seizure.
- Unilateral posterior shoulder dislocation and biting the side of the tongue are highly specific for an epileptic seizure.
- Vertebral fracture can occur during a seizure but is quite common in other situations making it less specific for an epileptic seizure.
- Urinary incontinence does not distinguish between epileptic seizure and syncope.
- Do not cause a shoulder dislocation by attempting to put the patient into the recovery position during a tonic-clonic seizure – wait until the convulsion has stopped.
- And finally – remember posterior shoulder dislocation occurs in seizures and is difficult to diagnose both clinically and radiologically, so you have to look hard for it.

about how to look after patients during an epileptic seizure (Epilepsy Action 2005)

URINARY INCONTINENCE

We often ask about incontinence when someone has a blackout, indeed patients are often referred to neurologists rather than other specialists when they have been incontinent on the assumption this suggests they have had an epileptic seizure rather than syncope. However, Hoefnagels (1991) found that incontinence was common both in those with seizures (17%) and those with other blackouts (33%) and did not distinguish between them (Table 2). Incontinence is neither a sensitive nor specific feature for a seizure – and yet this is probably the most asked about of all our silent witnesses! Incontinence clearly does not contribute to the differential diagnosis between seizure and syncope.

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REFERENCES

- Aboukasm A & Smith BJ (1997) Nocturnal vertebral compression fracture. A presenting feature of unrecognised epileptic seizures. *Archives Fam Med*, **6**, 185–7.
- Benbadis S, Wolgamuth BR, Goren H, Brenner S & Fouad-Tarazi F (1995) Value of tongue biting in the diagnosis of seizures. *Archives of International Medicine*, **155**, 2346–9.
- Brakestone M, Patterson SD & Kertesz A (2001) Triple 'E' syndrome: bilateral locked posterior fracture dislocation of the shoulders. *Neurology*, **56**, 1043–4.
- Buhler M & Gerber C (2002) Shoulder instability related to epileptic seizures. *Journal of Shoulder and Elbow Surgery*, **11**, 339–44.
- Cummings SR & Melton LJ (2002) Epidemiology and outcomes of osteoporotic fractures. *Lancet*, **359**, 1761–7.
- DeToledo J & Lowe MR (2001) Seizures, lateral decubitus, aspiration and shoulder dislocation. Time to change the guidelines? *Neurology*, **56**, 290–1.
- Epilepsy Action (2005) Epilepsy information and First Aid for seizures, accessed at <http://www.epilepsy.org.uk/info/firstaid.html>.
- Gosens T, Poels PJE & Rhondius JJ (2000) Posterior dislocation fractures of the shoulder in seizure disorders—two reports and a review of the literature. *Seizure*, **9**, 446–8.
- Gowers WR (1893) *A Manual of the Diseases of the Nervous System*, 2nd edn. J and A Churchill, London.
- Hawkins RJ, Neer CS, Pianta RM *et al.* (1987) Locked posterior dislocation of the shoulder. *Journal of Bone and Joint Surgery America*, **69**, 9–18.
- Hoefnagels WAI, Padberg GW, Overweg J, Van der Velde EA & Roos RAC (1991) Transient loss of consciousness: the value of the history for distinguishing seizure from syncope. *Journal of Neurology*, **238**, 39–43.
- Mattson R & Gidal BE (2004) Fractures, epilepsy and antiepileptic drugs. *Epilepsy and Behaviour*, **5**, S36–40.
- Robinson CM & Aderinto J (2005) Posterior shoulder dislocations and fracture dislocations. *Journal of Bone and Joint Surgery*, **87**, 639–50.
- Shaw J (1971) Bilateral posterior fracture dislocation of the shoulder and other trauma caused by convulsive seizures. *Journal of Bone and Joint Surgery*, **53A**, 1437–40.
- Souverein P, Webb DJ, Petri H, Weil J, Van Staa TP & Egberts T (2005) Incidence of fractures among epilepsy patients: a population based retrospective cohort study in the general practice research database. *Epilepsia*, **46**, 304–10.
- Vasconcelos D (1973) Compression fractures of the vertebrae during major epileptic seizures. *Epilepsia*, **14**, 323–8.