The Journal of Laryngology & Otology
October 2001, Vol. **115,** pp. 782–787

Vertigo syndromes and mechanisms in migraine

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Abstract

This paper attempts to define and categorize the vertigo associated with migraine. A retrospective chart review of 344 cases of vertigo identified 19 cases with headaches characteristic of migraine as per strictly defined criteria (International Headache Society, 1988).Four distinct types of vertiginous syndromes were noted. The commonest syndrome (Group I) manifested transient episodes of imbalance with additional momentary subjective rotary vertigo worsened by movement. The attacks lasted a few hours and evaluation in the inter-episode interval demonstrated no vestibular deficit. Group II manifested transient objective rotatory vertigo of from 10 minutes to a few hours but no demonstrable permanent vestibular deficit. Group III displayed symptoms and signs characteristic of benign paroxysmal positional vertigo (BPPV) and Group IV manifested a permanent unilateral labyrinthine weakness. Causation of vertigo by migraine was implied in 10 of 19 cases where the headache and vertigo occurred simultaneously and in two other cases where the vertigo improved with anti-migraine prophylactic treatment.

Four distinct and characteristic vertigo syndromes have been noted with migraine. Their spectrum ranges from a transient reversible dysfunction to a more permanent destruction, and includes involvement of both the peripheral and the central vestibular systems.

Key words: Migraine; Vertigo

Introduction

Although the association of migraine and vertigo has been frequently described,[1–3] a casual link between the two remains elusive. Some of the reasons for this are enumerated below:-

(1) The diagnoses of both migraine and vertigo are predominantly clinically based. Migraine is diagnosed entirely by the patient's history, and in most cases of vertigo, too, the history remains most important. Diagnostic features on clinical examination and laboratory testing are few. Some amount of subjectivity therefore necessarily remains with both diagnoses. This lack of objectivity in either diagnosis, makes a scientific study correlating these disorders extremely difficult.

(2) Both disorders are common in the general population. Recent population-based studies using the International Headache Society (IHS, 1988) diagnostic criteria for migraine[4] have consistently estimated the prevalence rates to be approximately six per cent in men and 15 to 18 per cent in women.[5,6] In the UK, 30 per cent of the population have consulted a doctor for giddiness by the age of 65 years,[7] and 40.8 per cent of people surveyed in the third phase of the National Study of Hearing reported that they had problems with balance, dizziness or giddiness.[8] Surveys on populations of migraine cases indicate an association with non-vertiginous dizziness in 27 to 72 per cent, and with true vertigo in 26 to 33 per cent.[1–3] However, in light of the above-mentioned high population-prevalence rates, a large proportion of this associated vertigo may well be causally unrelated to the migraine. In statistical terms, it may be easier to prove that a disorder is not associated with giddiness or vertigo, than to prove that it is associated.

(3) Migraine leads to multiple pathophysiological alterations and similarly vertigo has a multitude of pathophysiological origins. The vertigo of migraine may occur with the headache, precede the headache, follow the headache or be temporally unrelated to the headache.[2] It may occur in situations where migraine headaches have never occurred (migraine equivalent[9] or migraine aura without headache[4]). Also, many different types of vertigo syndromes have been associated with migraine.

Despite these limitations this study aims to define and categorize the vertiginous syndromes which may be associated with, or be caused by, migraine.

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Accepted for publication: 2 May 2001.

Materials and methods

A retrospective chart review of all 344 patients (Age range 12–78 yrs. (mean – 39.33yrs); Male:Female ratio 198:146) seen at the vertigo clinic of a tertiary referral centre from January 1997 to March 2000 was undertaken. Twenty-four cases where migraine had been suspected as a possible aetiology of the vertigo were identified. The diagnosis of migraine in these cases was reviewed for compliance with the criteria for migraine (Table I) as set out by the Headache Classification committee of the International Headache Society (1988).[4] Five of the 24 cases did not adhere to the criteria as specified above and were therefore excluded. One case where a migraine equivalent syndrome was strongly suspected was also excluded as it manifested no accompanying headache and therefore did not fulfil the laid-down criteria. Nineteen cases that fulfilled the criteria constituted the study group.

All cases had had a detailed neuro-otological history and examination recorded on a prescribed proforma. This included history of the vertiginous episodes, intervening vertigo-free periods, hearing loss, tinnitus, ear fullness, headache, neurological symptoms and systemic illnesses, and examination for spontaneous and induced nystagmus, vestibulospinal testing, otological examination, general physical examination, cranial nerve examination and testing for cerebellar signs. All cases had a pure-tone audiogram and all except two had an electronystagmogram (ENG) (one patient had a unilateral perforation and one other declined the procedure). The audiogram involved threshold estimations in a soundproof room at frequencies from 250 Hz to 8000 Hz and was judged as abnormal if any threshold was greater than 20 decibels. The ENG evaluation included testing for spontaneous, gaze and positional nystagmus, pendular tracking test, optokinetic nystagmus and bithermal caloric tests. The caloric tests were conducted with electronystagmographic recording in a dark room with the eyes closed and 44°C and 30°C irrigations of only 20 ml water over 30 seconds. A minimum of five minutes of a nystagmus-free period was maintained between irrigations. As per the previously determined normative data for our laboratory, the nystagmic response to caloric stimulation was judged as hypoactive when the maximum velocity of slow phase responses were less than 6° and/or a culmination frequency less than 28 beats per 30 seconds, and hyperactive when the culmination frequency was greater than 70 beats per 30 seconds. The indices of canal paresis and directional preponderance were calculated and a value of greater than 20 per cent for canal paresis and 30 per cent for directional preponderance were taken as indicative of abnormality. Additional neurological consultation, imaging studies, evoked response testing or other investigations were undertaken where necessary.

The essential characteristics of the study group are displayed in Table II. It included 15 females and four males, with a mean age of 34 years (range 18 years to 56 years). They had been symptomatic with migraine from six months to 30 years with a mean duration of 10.8 years. Six patients had migraine with aura (classical migraine) and 13 had migraine without aura (common migraine). The classical and common migraine groups did not differ significantly in the age profile or the mean duration of symptoms.

The primary symptom for which all cases sought medical attention was vertigo. They were categorized into four groups based on the vertiginous symptoms and ENG evaluation. Group I manifested transient periodic sensations of imbalance and momentary subjective rotatory vertigo especially with movement, but no demonstrable permanent vestibular deficit either on clinical examination or the ENG. Group II manifested transient rotatory vertigo with, or without, associated hearing loss of 10 minutes to few hours but no demonstrable permanent vestibular deficit either on clinical examination or the ENG. Group III displayed symptoms and

TABLE I
DIAGNOSTIC CRITERIA FOR MIGRAINE (INTERNATIONAL HEADACHE SOCIETY, 1988)[1]

Migraine without aura	Migraine with aura
At least five attacks fulfilling the following	*At least two attacks fulfilling the following:*
Headache lasting 4–72 hours (untreated or unsuccessfully treated) and having at least two of the following– – Unilateral location – Pulsatile quality – Moderate or severe intensity (inhibits/prohibits daily activities) – Aggravation by routine physical activity AND – Nausea and/or vomiting – Photophobia and phonophobia	Headaches with at least three of the following– – One or more fully reversible aura symptoms indicating focal cerebral cortical and/or brain stem dysfunction – At least one aura symptom develops gradually over more than 4 minutes, or two or more symptoms occur in succession. – No one aura symptom lasts more than 60 minutes. – Headache may begin before the aura, occur simultaneously or follow the aura with a symptom-free interval of less than 60 minutes.
AND	
– History, physical and neurological examination do not suggest head trauma, vascular disorders, non-vascular intracranial disorder, substance exposure or withdrawal, non-cephalic infection, metabolic disorder or disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structure. – If such a disorder is suspected by the history, physical or neurological examination it is either ruled out by appropriate investigations or the migraine attacks do not occur for the first time in close temporal relation to the disorder.	

TABLE II
CLINICAL PROFILE, TREATMENT ADMINISTERED AND RESPONSE TO TREATMENT IN THE STUDY GROUP

Sr no.	Age/ Sex	Systemic illness	Migraine type	Vertigo Description	Temporal association with headache	ENG	Category*	Otological manifestations	Treatment	Follow up
1	28 F	–	Common	Imbalance, motion provoked subjective vertigo	Concurrent usually	Normal	1A	Nil	Propranolol	Episodes frequency decreasd
2	32 F	–	Classical	Imbalance, motion provoked subjective vertigo	Concurrent Sometimes	Normal	1A	Nil	Propranolol, sleep hygiene, avoid ppt factors	Episodes frequency decreased
3	18 M	–	Classical	Imbalance,, motion provoked subjective vertigo	Concurrent usually	Normal	1A	Bl-tinnitus concurrent with headache	Non-specific	No follow-up
4	25 F	–	Common	Imbalance, motion provoked subjective	Concurrent Sometimes	Normal	1A	Nil	Non-specific	No follow-up
5	40 F	–	Classical	Imbalance, nausea	Concurrent usually	Hyperactive calorics	1A	Hyperacusis and occ tinnitus concurrent with headache	Propranolol	Decreased frequency
6	26 M	–	Common	Episodic imbalance, motion intolerance	Nil	Normal	1B	Nil	Non-specific	No follow-up
7	30 F	–	Classical	Imbalance, motion provoked subjective vertigo	Nil	Mildly suppressed calorics	1B	Nil	Propranolol	Asymptomatic for 6 mth, then long follow-up
8	45 F	–	Common	Imbalance, motion provoked subjective	Nil	Normal	1B	Nil	Propranolol	Improved
9	55 F	–	Classical	Imbalance, motion provoked subjective vertigo	Nil	Normal	1B	Nil	Non-specific	No follow-up
10	30 F	–	Common	Episodic objective rotatory vertigo of 10–60 minutes	Migrainous attacks and vertigo attacks started around same time (6 mths ago) but do not occur concurrently	Normal	2B	Transient rt hearing loss with vertigo, Intermittent tinnitus	Anti Ménierès	Improved
11	30 F	–	Common	Episoidic grouped attacks of 30–120 minutes	Nil	Borderline hyperactive	2B	Transient rt ear fullness with vertigo, Intermittent tinnitus	Anti-hydrops	No follow-up
12	38 F	–	Classical	Positional vertigo	First attack with migraine attack	B/L mildly hypoactive	3A	Nil	Avoidance of ppt. position/movement	Symptoms improving at presentation itself. Subsequent resolution +
13	28 F	–	Common	Classical left PSCC BPPV	BPPV onset after one typical migraine headache	Declined by patient	3A	Nil	Particle repositioning manoeuvre × 2	Initial response then recurrence. Repeat prm effective
14	56 M	DM	Common	Typical of BPPV	Nil	B/l cold Hyperactive	3B	Nil	Avoid ppt factors	Symptoms improving at presentation itself. Subsequent resolution +
15	38 F	HT	Common	Positional vertigo	Nil	Contraindicated-TM perforation	3B	Rt ear perforation	Precautions- pt. already improving	18 months follow-up asymptomatic
16	29 F	–	Common	Sudden prolonged obj. rotatory vertigo	Initial severe vertigo with migraine attack	Left canal paresis	4A	Nil	Propranolol, Vest. rehab. exercises	Both headache and unsteadiness improved
17	40 M	–	Common	Initial prolonged obj. rotatory vertigo, subsequent occ. positional vertigo	Initial severe vertigo with migraine attack	Left canal paresis	4A	Left high fr. Hearing loss 40 dB	Vest. Rehab. exercises	Symptoms improving at presentation itself. Subsequent resolution +
18	28 F	–	Common	Sudden prolonged obj. rotatory vertigo	Nil	Left canal paresis	4B	Nil	Vest. Rehab. exercises	Improved
19	39 F	–	Common	Imbalance/swaying with nausea and headache	Simultaneous	Right canal paresis	4B, 1A	2 yrs. rt. Tinnitus and hearing loss −40 db	Vest. Rehab. exercises, avoid ppt. factors.	No follow-up

*1 – periodic transient sensations of imbalance and momentary subjective rotatory vertigo especially with movement and no demonstrable permanent labyrinthine deficit; 2 – transient rotatory vertigo of 10 minutes to few hours and no demonstrable permanent labyrinthine deficit; 3 – symptoms and signs characteristics of benign paroxysmal positional vertigo; 4 – unilateral labyrinthine deficit as per either the clinical examination or the ENG.
A – definite temporal relationship between vertiginous episodes and migraine headaches; B – no temporal relationship between vertigo and headache.
ppt. = precipitating
pt. = patient

signs characteristic of benign paroxysmal positional vertigo and Group IV manifested a definite unilateral labyrinthine weakness as per either the clinical examination or the ENG findings. Cases were further classified as A or B depending on whether or not the vertigo was temporally concurrent with the headache.

Treatment was variable and depended on the individual diagnosis regarding the cause of vertigo. It included anti-migraine prophylaxis, treatment to abort a migrainous attack, treatment directed at endolymphatic hydrops, avoidance of factor precipitating vertigo, the particle repositioning manoeuvre and Cawthorne Cooksey exercises. Follow up of greater than six months was available in 13 of 19 cases.

Results

Nineteen of 344 cases evaluated (5.5 per cent) fulfilled the prescribed criteria for migraine and were diagnosed to suffer from migraine-related dizziness. The relative incidence of the diagnosis was greater in females (10.3 per cent) than in males (two per cent). The chief characteristics of these cases are reported in Table II.

The mean period between the onset of migraine headache and the onset of vertiginous symptoms was 8.4 years (range 0 to 30 years). The vertiginous symptoms reported by these patients could broadly be classified into the four categories (I–IV) as described above. The cases further sub-categorized into those wherein the vertigo attacks demonstrated a significant temporal relationship to the headache (sub-category A) and those wherein no such relationship was manifest (sub-category B). Ten of 19 cases demonstrated a clear temporal relationship between the headache and vertigo leading to the assumption that they were causally related. Nine cases had no temporal relationship between the vertigo and the headache.

Nine cases (*Cases 1–9*; symptom category I-A and B) reported episodic sensations of imbalance, associated with nausea and vomiting, and lasting from 30 minutes to a few hours. During these episodes patients also manifested motion intolerance with even minimal head movements precipitating momentary subjective rotatory vertigo. The intervening periods between episodes were entirely asymptomatic. ENG evaluation, performed in the symptom-free periods, did not reveal any significant abnormality in any case. Five of these cases (category 1A) reported concurrent vertiginous episodes and migraine headaches on at least some occasions – three of these with classical migraine also reported simultaneously occurring other neurological symptoms, and two of these same three also reported simultaneous otological symptoms (tinnitus and phonophobia). The attacks, which were concurrent with the headache, lasted the entire duration of the headache. Four cases (category IB) reported of entirely separate attacks of vertigo and migraine and no concurrent attacks.

Of the total six cases of classical migraine in the entire study group, five were in this symptom category (I-A and B). This association between symptom category 1 and classical migraine was statistically significant ($p = 0.049$ Fisher's exact one-tailed test).

Four cases (two each of category IA and IB) were judged to have attacks frequently enough to merit prophylactic anti-migraine treatment and received propranolol (80–160 mg/day). All four reported a reduction in the frequency of vertiginous symptoms with this therapy.

Two cases (*Case 10* and *11*; symptom category II) suffered episodic objective rotatory vertigo lasting from 10 minutes to a few hours. Although the vertigo and headache did not occur concurrently in either case, in one of the cases both these symptoms had started around the same time. One case reported an associated transient unilateral hearing loss, and the other transient unilateral ear fullness occurring with the vertiginous attacks. The intervening periods between attacks were entirely asymptomatic and the ENG and the audiogram performed in these periods were also normal. Although these patients had been symptomatic with vertigo for three years and for six months respectively, the complete features of Ménierè's disease were not present in either.

Four cases (*Cases 12–15*; symptom category III-A and B) presented with the characteristic syndrome of BPPV. In two of these cases the onset of vertigo had been with a migraine headache. The other two had no such temporal association with a migraine attack but had other predisposing factors for BPPV – one had long-standing diabetes and the other was hypertensive. In all cases except one the symptoms had significantly improved by the time of presentation to the clinic. One, however, had severe and persisting symptoms and these subsequently improved following the particle repositioning manoeuvre.

Four cases (*Cases 15–19*; category IV-A and B) had a unilateral persistent vestibular deficit as indicated by the clinical manifestations and the ENG. Three of these reported symptoms characteristic of a destructive vestibular lesion – i.e. a particularly severe first vertiginous attack which had taken a few weeks to recover, and subsequent occasional momentary mild vertiginous symptoms. In two cases this first vertigo attack had started with a concurrent migraine headache. One other of the four cases (*Case 19*) reported no initial severe vertiginous symptoms but reported periodic sensations of imbalance and subjective vertigo occurring simultaneously with her migraine headaches. She thus had symptoms as in cases of symptom category IA, but was nevertheless categorized as IVA as she also displayed a unilateral canal paresis on the ENG and ipsilateral non-fluctuant moderate hearing loss and tinnitus.

Discussion

Migraine-related dizziness or vertigo is infrequently diagnosed in our clinic. In not all cases with both migraine and vertigo can a linking causative mechanism be demonstrated or be reasonably supposed. In this study the diagnosis was noted in two per cent of males and 10.3 per cent of females. This is in contrast to some other reports where migraine was diagnosed as the cause in about 15 per cent of patients presenting with vertigo.[10] The selectivity exercised in making the diagnosis is the probable reason for this study having a higher proportion of cases with concurrent headache and vertigo.

The IHS 1988 criteria for the diagnosis of migraine have been strictly adhered to. The subjectivity in the diagnosis of migraine has thus been minimized. The subjectivity in vertigo diagnosis has been minimized by avoiding any reference to an implied diagnosis. Instead the vertigo syndromes have been defined on the basis of the patient's described symptoms. The categorization of the vertigo syndromes has been based on the description of the vertigo attack, the intervening period, the audiogram and the ENG.

This study identifies four distinct vertigo syndromes in patients with migraine. The commonest syndrome identified was that of a persistent sensation of imbalance (symptom category I), which when

concurrent with the headache lasted the entire duration of the headache. Patients with this symptom complex were more likely to suffer from migraine with aura than were patients manifesting the other vertiginous syndromes. Following the vertigo these patients had a complete recovery and no residual labyrinthine symptoms or deficit. In the other categories (symptom category III and IV) however the migraine attack resulted in permanent damage primarily localized to the peripheral vestibular system.

Both migraine and vertigo being common disorders,[5–8] their simultaneous occurrence in a particular patient does not necessarily imply causation. Conclusive evidence of migraine being the cause of vertigo is difficult to establish. In this study causation of vertigo by migraine was implied either by evidence of a temporal concurrence of the vertigo and the migraine headache; or by response of the vertiginous symptoms to anti-migraine treatment. Causation was implied in 10 cases by the first criteria and in an additional two by the second criteria. Overall, causation was implied in 12 of 19 cases.

No direct causation is implied in the other seven cases. In at least one (*case 15*) and possibly in another (*case 14*) the labyrinthine pathology could be potentially attributed to an origin other than migraine. No other aetiological factor was however identified in the other five. Since they all otherwise fulfilled the criteria for migraine it is possible that the vertiginous attacks were manifestations of the syndrome previously known as the migraine equivalent syndrome and currently described as 'Migraine aura without headache'.[4,9,11]

The pathophysiology of migraine is incompletely understood. Two distinct mechanisms have been proposed to be the cause of the associated neurological symptoms – a) ischaemia caused by vasospasm; and b) abnormal neurological processing of sensory inputs. Vasospasm in association with the migraine aura has been documented.[12] The vasospasm-induced aura precedes the headache and is by definition short-lasting (less than 60 minutes).[4] Current concepts however conceptualize the vasospasm and other vascular changes in migraine to be secondary to other primary neurological phenomena, which act via the trigemino-vascular reflex to cause a neurogenic sterile inflammation of the intracranial and extracranial vasculature.[13] The primary abnormality in migraine may be a disinhibition of the pathways subserving sensory input, particularly noxious sensations from the trigeminal and cervical nerve distributions.[14] Abnormal central processing leading to an increases sensitivity to sensory inputs is thought to result in the headache and also the associated neurological symptoms of photophobia and phonophobia.[15,10] Such symptoms are not part of the aura (Table I) and usually last the entire duration of the headache.

Analogous to the increased sensitivity in migraine to visual and auditory stimuli, a similar alteration in the sensitivity to vestibular inputs has been hypothesized.[16] The syndrome of episodic prolonged imbalance associated with nausea/vomiting and with momentary subjective vertigo on movement can be very accurately explained by such an alteration in the calibration, or gain, of the vestibular inputs and its associated reflexes. The vestibulo-ocular reflex and the vestibulo-spinal reflexes display an enormous physiological plasticity, that is mediated primarily by neurological mechanisms involving the cerebellum and the inferior olivary nucleus.[17–19] These mechanisms monitor the adequacy of the vestibular nucleus efferent discharge in maintaining a stable retinal image and body posture, and correct for inadequate or inappropriate activity by fine tuning the gain of the vestibulo-ocular and vestibulo-spinal reflexes. A pathological alteration of the gain of these reflexes, as is proposed to occur with migraine, would lead to abnormal perception of the sensory input from the otoconia and cupula and lead to symptoms similar to category I. Abnormal perception of otoconial input and the force of gravity would lead to symptoms of disorientation in space and of swimming and swaying sensations. Abnormal perception of the semicircular canal input and alterations in the gain of the vestibulo-ocular and vestibulo-spinal reflexes would lead to abnormal reflex eye and head movements and thus cause motion intolerance and subjective vertigo with movement.

The observation in this study that these symptoms of imbalance and motion intolerance occur with the headache rather than as a preceding aura lends further credence to the hypothesis that they result from abnormal central processing of vestibular input rather than from vasospasm.

The presentation of patients in category III and IV is typical of the well-defined syndromes of BPPV and of a sudden vestibular destructive lesion. They have been associated respectively with the pathological findings of canalolithiasis/cupulolithiasis[20,21] and of diffuse damage to the vestibular sensory epithelium and neuronal elements.[22] A wide variety of aetiologies have been implicated as the cause of these syndromes. Both have also been previously reported in temporal association with migraine headaches[2,10,23,24] and have been attributed to the associated vasospasm. The extent of pathological damage- and the corresponding clinical syndrome-probably depends on the severity of ischaemia. BPPV may result from restricted ischaemia to the ultricular otconia, while the permanent destructive vestibular lesion (with or without associated hearing loss) would result from more global damage to the vestibular (and cochlear) neuroepithelium.

The symptom complex of objective rotatory vertigo of 10 minutes to few hours duration, with or without other otological symptoms, but with no demonstrable permanent labyrinthine deficit or other neurological deficit (symptom category II) is generally believed to be the result of a reversible labyrinthine failure.[22] Although no case in this study demonstrated such a vertigo simultaneous with the migraine headache, other workers have reported such simultaneous attacks,[25,26] and the syndrome –

termed as benign recurrent vertigo – has been regarded as a migraine aura which may also manifest without the headache.[3,27] The duration of vertigo in the various reported cases has, however, often exceeded the defined 60 minute limit of aura symptoms, and the vasospastic mechanism has therefore been questioned.[10] It is, however, probably unreasonable to expect the labyrinthine failure of ischaemia to necesssarily revert immediately on reversal of the vasospasm. The alternate proposed explanation for this syndrome conceptualizes an asymmetrical alteration of the gain of the vestibulo-ocular reflex to account for the objective vertigo.[16] This explanation however fails to account for the sometimes frequently associated unilateral fluctuant hearing loss and tinnitus. Ischaemia therefore seems a plausible explanation for this symptom complex.

Basilar artery migraine, now termed basilar migraine, is also known to cause vertigo but was not encountered in this study. The IHS 1988 document however cautions that this entity has possibly been overdiagnosed in the past, especially as it can easily be confused with hyperventilation and anxiety disorder.[4]

Conclusion

Migraine is a frequent cause of vertigo and was identified as the causative factor in 5.5 per cent of cases in our clinic.

Migraine can cause a variety of vertiginous syndromes. The commonest manifestation is a transient sensation of imbalance with associated motion intolerance, which usually lasts for a few hours and when concurrent with migraine occurs with the headache. The group with this symptom complex seems to have a higher proportion of classical migraine than is the case otherwise. The symptoms are probably the result of an abnormality of central vestibular processing.

Other manifestations include a reversible labyrinthine failure lasting for a few hours; the syndrome of BPPV and the syndrome of an acute irreversible labyrinthine destructive lesion. These may be the result of varying degrees of ischaemia to the labyrinth.

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Mr A. Thakar takes responsibility for the integrity of the content of the paper.
Competing interests: None declared