Short report

Clinical features of the exploding head syndrome

J M S PEARCE

From the Department of Neurology, Hull Royal Infirmary, Hull, UK

SUMMARY Fifty patients suffering from the “exploding head syndrome” are described. This hitherto unreported syndrome is characterised by a sense of an explosive noise in the head usually in the twilight stage of sleep. The associated symptoms are varied, but the benign nature of the condition is emphasised and neither extensive investigation nor treatment are indicated.

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A short paper on this recently described syndrome1 outlined the salient features of 10 patients. This attracted attention in the national lay press in the UK, Australia and North America which in turn provoked a considerable correspondence addressed to the author, furnishing a remarkable wealth of further clinical data. This paper attempts to expand the clinical features to include anamnestic data from a further 40 patients.

The syndrome in essence consists of an hitherto unreported benign symptom characterised by a sense of explosion in the head, confined to the hours of sleep, which is harmless but very frightening for the sufferer. It receives no mention in current texts.2-4 Correspondents includes several physicians and neurologists who suffered from it, but in each case had not encountered the symptom in their own patients—“because I hadn’t bothered to enquire”, they typically remarked.

The main features are illustrated by the following case report.

Case report

A 74 year old stable and sensible woman (case 1 in ref 1) had transient attacks of common migraine with generalised severe throbbing headache, photophobia and vomiting between the ages of 41 and 55, after which they disappeared gradually in the next two years. She presented complaining of different attacks starting when aged 67 years, occurring with no recognisable antecedent cause, exclusively during sleep, at varying intervals, usually 2 to 3 per week, with remissions of up to 3 weeks. She described: “Being wakened by a sudden bang in the head, as if my head was bursting with a flash of light over both fields of vision, after which I would be dazed for a split second” and would “Come round, terrified, my heart thumping. There was no pain, just a frightening sense of explosion”. The attacks caused apprehension of a brain haemorrhage or stroke. Examination was normal. She has remained healthy 7 years later.

Patients

Clinical symptoms, evolution and natural history were obtained from 44 patients (new series) in addition to the original 10 cases. Four new patients were excluded because of inadequate historical data (two patients) or features suggestive of primary otological disease with prominent tinnitus which did not conform to the original syndrome (two patients). The other descriptions proffered conformed precisely to the original account, totalling 50 cases in all. In the interests of space 30 of the new series are summarised. Table 1 shows the sex, age distribution and the duration of symptoms where stated (since some correspondents omitted their current age and age of onset) in the whole series of 50 patients. Table 2 records the description of symptoms, the associated symptoms and observations submitted by the patients.

Clinical features

The new cases reported add new information. As originally stated the syndrome is exclusively nocturnal, and it is now clear occurs predominantly in the twilight stage as the patient is dropping off to sleep, or, less often if they waken during the night and again fall asleep. Some patients simply report it waking them from sleep but, without sleep monitoring it is not possible to know whether this occurs during stages 1 to 4 or if they have woken briefly and are falling asleep again—in

Table 1  Sex, duration and age* of patients

<table>
<thead>
<tr>
<th>Sex</th>
<th>Duration of symptom (yr)</th>
<th>Number</th>
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<tr>
<td>31 females</td>
<td>19 males</td>
<td>4-10</td>
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<tr>
<td>3</td>
<td>4</td>
<td>2</td>
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* = Exact age given by 39 of 50 patients.
another twilight stage. All sufferers report noise, not pain. The dramatic nature is evident in their words “enormous roar, so loud it could kill me”; some, however, say that it can be mild and infrequent. The terror induced is notable in every case, until some degree of acceptance is achieved after many years in which they have maintained good health. Preceding events are generally unremarkable, but three physicians noted attacks to start and to recur when they were under personal stress or tired and overworked. No consistent observations were proffered in respect of “incidental medication”.

The onset is much more varied than I had originally appreciated; some start in childhood, but no decade is spared. The commonest age of onset remains however in middle and old age: 22 of 39 reported their first attack after the age of 51 (table 1).

The pattern of episodes of explosions is also variable. Some report 2 to 4 attacks followed by prolonged or total remission, others have more frequent attacks up to 7 in one night, for several nights each week and may then remit for several months, for reasons largely unknown.

Associated symptoms are documented by some sufferers. Five (10%) report a simultaneous flash of light; three (6%) reported a curious sensation as if they had stopped breathing and had to make a deliberate effort to breathe again—“an uncomfortable gasp”. Interestingly, no patient remembers simultaneous night starts (myoclonic jerks), though many had experienced these separately. A family history was volunteered by three patients (one a man of 49 and his grandmother, but neither knew of the others’ affliction for many years). Five patients gave a history of migraine and one of epilepsy in the past, but with no evident relationship to the explosions. In almost every case (47/50) the after effects of fear, terror, palpitations or forceful heartbeat are mentioned. A physiologist reported attacks “accompanied by an obvious arrhythmia—heart rate was normal but with missed beats with compensatory pause; neither symptom recurred when I cut down my coffee drinking”. He attributed the exploding head to a stronger arterial pulse after the compensatory pause, but it seems more probably, post hoc, that the anxiety-induced catecholamine surge promoted his extrasystoles. Five subjects described tinnitus, three of whom had deafness,
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but the tinnitus was the usual daytime phenomenon and the nocturnal explosion was qualitatively different in each case.

Discussion

O'Donnell and Martin drew attention to a syndrome previously recorded as ice-pick headache or needle in the eye syndrome and renamed it cephalgia fugax. They also mentioned in the same paper earlier anecdotal report of the quite separate "exploding head syndrome". We have suggested that the syndrome is one of a variety of common but alarming cranial symptoms, quite distinct from migraine, muscle-contraction headache and other well described cranial neuralgias. We suggested that the ice-pick and needle in the eye attacks could be provisionally designated "benign paroxysmal cranial neuralgias" but, I believe the exploding head syndrome has a separate identity since it is not painful; hence the terms neuralgia and cephalgia are wholly inappropriate.

Weir Mitchell may have been describing this phenomenon when referring to "sensory shocks...a feeling of rending...a bolt driven through the head". Armstrong-Jones, in a paper I had overlooked, described a frequent complaint of a sudden crash or noise as if something had given way in the brain. It was frightening, and in many patients was the initiating symptom of melancholia or neurasthenia. No age restriction was noted and prognosis was good. Oswald described "a flash of light accompanied by a violent bang" with "a sense of alarm, together with a cold sweat, laboured breathing and tachycardia" but associated it with electric feelings ascending from the abdomen to the head as a feature of light sleep.

As a symptom it is probably fairly common. No less than fifty patients voluntarily wrote about their symptoms within 4 weeks of publication. But, as a source of complaint it is rare; many said they had been ashamed to mention it to their doctors or that their complaint had been greeted with incredulity if not frank disbelief. The patients are predominantly middle-aged or elderly, slightly more commonly women than men. There is little evidence of relevant past illness and no other CNS disease in evidence. The complaint is exclusively in sleep, but this may include daytime naps. The victim is aroused from sleep by a violent sensation of explosion in the head. It occurs abruptly with apparently great force, yet it is not a pain. Patients are so alarmed that at first they may, inaccurately, describe it as a pain, but closer questioning shows the awareness is not of a hurt but more of a noise deep in the centre or back of the head. By the time the sufferer is wide awake it has gone, but not surprisingly it leaves in its wake a sense of great consternation and sometimes momentary difficulty in breathing, tachycardia and sweating.

It may occur for a few weeks or months, then spontaneously disappear, or, may recur irregularly every few days, weeks or months for much of a lifetime, yet with no preceding cause in the habits or events of days prior to the attack. The patient's fear is usually of a cerebral haemorrhage, stroke, or brain tumour.

It is entirely benign, and I suspect quite common, but underreported. What causes the bomb-like noise remains a mystery and I know of no vascular or hydrodynamic changes in the brain, labyrinths or CSF pathways which cause comparable symptoms. A momentary (almost ictal) disinhibition of the cochlea or its central connexions in the temporal lobes might produce such a phenomenon; less likely is a sudden involuntary movement of the tympanum or the tensor tympani. Gordon has suggested rupture of the labyrinthine membrane or a springing open of the Eustachian tubes with a crack like a pistol, especially if there is a tendency to undue patency. I doubt that these tentative explanations account for the repetitive phenomenon recorded here in patients without evidence of tinnitus, vertigo or deafness.

The likeliest explanation is to class it with the other physiological phenomena such as nocturnal myoclonus, which mark the transition from wakefulness to grade 1 sleep. Normal sleep cycles of non-REM sleep alternate with REM sleep at intervals of about 90 to 100 minutes, with three to five cycles per night. However the elderly are known to have more variable and broken sleep and considerably less delta sleep (stages 3 and 4) which declines from 15 to 20% of total sleep time in youth to 0 to 5% in the elderly. There is a corresponding increase from 5% (youth) 15% (elderly) of stage 1 sleep, the onset of which is associated with twilight phenomena. This is consistent with the twilight occurrence of explosions in the head. Such postulates are speculative. We need further studies using polysomnography employing continuous recordings of EEG, EOG, mentalis EMG, surface EMG on the limbs, ECG, nasal and oral airflow measurements as well as ear oximetry. Until more is understood of its aetiology, it remains as a genuine source of distress which does not seem related to neurosis. Firm reassurance is essential but drug therapy appears unwarranted.

References

3 Pearce JMS. In: Blau JN, ed. Migraine: Clinical,


13 Fry JM. Sleep Disorders. Medical Clinics of N America, April 1987, 95-110.)