

Post-epileptic headache and migraine

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SUMMARY One hundred epileptic patients were questioned about their headaches. Post-ictal headaches occurred in 51 of these patients and most commonly lasted 6-72 hours. Major seizures were more often associated with post-epileptic headaches than minor attacks. Nine patients in this series of 100 also had migraine: in eight of these nine a typical, albeit a mild, migraine attack was provoked by fits. The post-ictal headache in the 40 epileptics who did not have migraine was accompanied by vomiting in 11 cases, photophobia in 14 cases and vomiting with photophobia in 4 cases. Furthermore, post-epileptic headache was accentuated by coughing, bending and sudden head movements and relieved by sleep. It is, therefore, clear that seizures provoke a syndrome similar to the headache phase of migraine in 50% of epileptics. It is proposed that post-epileptic headache arises intracranially and is related to the vasodilatation known to follow seizures. The relationship of post-epileptic headache to migraine is discussed in the light of current ideas on migraine pathogenesis, in particular the vasodilation which accompanies Leao's spreading cortical depression.

Little is known about post-epileptic headaches, although their occurrence is accepted by patients and doctors. Yet standard works¹⁻³ on both epilepsy and headache give the subject cursory mention only. Gowers⁴ drew attention to their duration, describing them as "severe headaches, general, lasting for several hours and often continuing during the rest of the day." In contrast, headaches occurring as a warning of impending fits, either as part of the prodrome or the aura, have been extensively studied most recently by Young and Blume⁵ in 1983. In this paper the authors have studied the incidence and characteristics of post-epileptic headache.

Method

One hundred epileptic patients (39 men and 61 women, average age 32 years) were interviewed, 70 at neurological out-patient clinics, 15 while they were on general neurological in-patient wards and 15 at a specialist residential centre for epileptics. Patients were questioned about prodromal symptoms, auras and post-epileptic phenomena, particularly those related to headache such as head tenderness, analgesic use and whether the pain was affected by manoeuvres such as coughing or sudden head movement. Other symptoms

including nausea and vomiting, photophobia, phonophobia, and visual disturbances were also noted. Patients were specifically asked whether they suffered local head trauma during their fits, and only patients who rarely experienced any head injury and clearly developed headaches without trauma were included in this study. Details of other headaches, associated neurological conditions, electroencephalographic and neuroradiological findings were recorded. The results of computed tomographic scans were present in the notes of 32 patients, of which five revealed abnormalities (two presumed gliomas, neither of which had been operated upon, one infarct, one arteriovenous malformation and one arachnoid cyst). All patients had experienced more than 10 fits and only patients who had post-ictal headaches after more than a quarter of their attacks were included in the detailed analysis of post-epileptic headaches.

Results

Incidence of headache

Post-epileptic headaches occurred in 51 patients. In 35 they were invariable, in five a usual sequel and in 11 they occurred after 25-50% of fits (fig). In contrast, 42 patients never suffered such headaches and seven others only rarely recollected headaches after fits. Headaches preceding fits affected only seven patients.

Two patients experienced prodromal headaches: one described a sensation of bifrontal pressure associated with mood depression for 2 days prior to a fit and the other right frontal headache, tiredness and urinary frequency for 3 days preceding fits. Five patients suffered a cephalic aura: two complained of a

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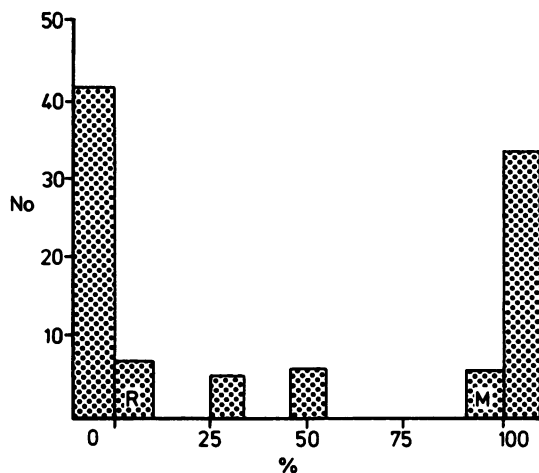


Fig Incidence of post-epileptic headache. The percentage of fits followed by headaches in the 100 patients studied. R and M indicate that patients' fits were "rarely" or "mostly" followed by headaches respectively.

vague ache in the head for 5 minutes, two had sharp stabbing retro-orbital pains lasting only a few seconds and one had a sensation of electricity passing through her head.

Features of post-epileptic headaches

Duration In 30 patients the pain lasted 6–72 hours, in 19 the pain continued for up to 6 hours and in three its duration was less than 30 minutes.

Characteristics In the 51 patients with headache, the pain was bilateral in 35 (generalised in 16, bifrontal in 15 and bioccipital in four), predominantly unilateral in 12 and four were uncertain of the distribution. The quality was described as throbbing by 16; the remainder had aching pains.

Photophobia necessitating prolonged periods in a darkened room was described by 20 patients and in most was accompanied by phonophobia. Scalp tenderness in the absence of generalised muscle aching was mentioned by 14. Coughing, sneezing, bending and sudden head movements increased the pain in 27 out of 51 patients. Three volunteered that even after the headache had worn off, these manoeuvres precipitated further pain. Most patients found that sleep helped the headache, and two people with nocturnal fits only experienced headache if woken shortly after the fit, but not if they slept several hours, usually until morning.

Analgesic requirement Twenty-five patients regularly needed analgesics for their post-epileptic headaches. Most took two or four tablets on the day of the fit, but in two cases 8–12 tablets were required.

Associated symptoms

Alimentary dysfunction Seven out of 51 patients invariably vomited repeatedly after seizures. A further five vomited only after one third of fits, and also suffered prolonged nausea. Two other patients had vomited regularly after fits as children, but the vomiting ceased when they became adults. Pronounced nausea preventing liquid or solid food intake for 24 hours, but without vomiting was noted in three other patients. Thus 17 of 51 patients with post-epileptic headaches had significant alimentary dysfunction.

Visual symptoms Nine patients experienced visual symptoms in association with their fits, which in seven cases took the form of a visual aura lasting 3 to 10 minutes, six described flashes of light and one blurring of vision. Two patients complained of post-ictal visual disturbances: one had blurred vision for 2 hours after her fits, and the other multicoloured scintillations for 5 minutes after she regained consciousness. Seven of these 9 patients, including the two with post-epileptic visual phenomena, also had regular post-epileptic headaches, but none had migraine.

Migraine Nine patients in this series of 100 had migraine in addition to epilepsy: eight of the nine had post-ictal headaches and they described both pains as having the same quality, although in three the migraine headache when it occurred by itself was of greater severity. In addition to the head pain, these eight patients also experienced other symptoms usually associated with their migraine attacks such as photophobia, nausea and vomiting (table). One patient with common migraine and a further one with childhood bilious attacks, but no migraine, did not suffer post-epileptic headaches.

Relationship between post-ictal headaches and type of seizures

Post-ictal headaches occurred after both major and minor seizures, but they were most frequent after grand mal attacks. The 49 patients who never had headaches after fits comprised 15 with major fits only, 15 with minor and major fits and 19 with minor fits alone. The 51 patients with regular post-ictal headaches included 17 with major fits only, 12 with both major and minor fits, and 13 with minor seizures only. Nine of the 12 patients with major and minor fits suffered headaches only after major seizures and never after minor attacks. Two patients felt that their headaches were worse after prolonged or repeated seizures. None of these 100 patients suffered from petit mal (3/s spike and wave) epilepsy.

There was no consistent relationship between the side of the EEG focus in patients with prolonged unilateral headaches and the side of the pain. Four patients with post-seizure headaches had other,

Table Features of post-epileptic headache (PEH)

	Total PEH patients	PEH & migraine	PEH only	Non PEH patients
No of patients	51	8	43	49
Duration:				
30 min	3	0	3	—
½-6 h	17	5	12	—
6-72	30	3	27	—
Not known	1	0	1	—
Nausea and vomiting	15	4	11	1*
Nausea alone	4	1	3	1*
Photophobia	21	7	14	1*
Vomiting and photophobia	7	3	4	—
Throbbing	18	5	13	—
Exacerbation on bending	32	8	24	—
Routine analgesia	26	6	20	—

*These three patients without post-epileptic headache had however the associated features shown.

neurological conditions, one multiple sclerosis, one presumed intracranial tumour, one previous optic nerve glioma and one chronic central nervous system degenerative condition while five of the 49 patients without post-seizure headaches had other abnormalities; one presumed tumour, one cerebral infarct, one unilateral temporal arachnoid cyst, one arteriovenous malformation and one spinocerebellar degeneration.

Other inter-ictal headaches

The incidence of tension headache did not vary between patients with post-epileptic headaches and those epileptics without post-ictal headaches. Neither of the two patients with presumed gliomas suffered the headaches of raised intracranial pressure.

Discussion

Thus epileptics could be divided into two groups, half who regularly experienced post-seizure headaches and half who never did. Within the former group, headaches were more common after major than minor fits.

Seizures provoked a migraine attack in eight of the nine migraineurs who had both conditions. In a further 40/91 patients, fits provoked a headache that has interesting similarities to the headache phase of migraine. In three patients a brief headache occurred without migrainous features and of uncertain type, possibly similar to the cephalic auras that a few epileptics experience.

The headache characteristics common to migraine and post-epileptic headaches are: throbbing quality, exacerbation by coughing and sudden head movements, nausea and vomiting, photophobia, and relief by sleep.⁶ The similarity of the two conditions is further underlined by the fact that the patients with both post-epileptic headache and migraine recognise the

pains as being essentially the same. Basser⁷ commented on a patient with hemiplegic migraine and epilepsy: "it is interesting to note that the patient considered his post-ictal headaches to be identical to his independently occurring migrainous headaches".

The acknowledged features of the headache phase of migraine are: its prolonged nature; throbbing quality; the associated gastrointestinal disturbance and the presence of photophobia. Although in this series of 51 patients with post-epileptic headaches all these four cardinal features were only present in three cases, 8 patients showed three of the four features, 17 suffered two, 11 only one and eight patients had none of these features. This does not, in the authors' opinion, detract from the similarity between these two conditions. In a survey⁸ of 750 patients only 49% of migraine sufferers described their headaches as throbbing in type. Furthermore, none of the various criteria for defining migraine demand all the cardinal features to be present.

The similarity between migraine and seizure headaches was also noted by Swainman and Frank⁹ in six children with paroxysmal headaches, epileptic EEG changes and a good response to anticonvulsant drugs. They considered the headaches as epileptic equivalents, a view shared by others^{10,11} including Halpern¹² (who used the term *epileptic cephalia*).

Vining¹³ in 1922 wrote on "bilious attacks and epilepsy" and was convinced that bilious attacks, particularly in children and adolescents were "an expression of a potentially epileptic nervous system". He described patients in whom major seizures were invariably followed by headache and vomiting and even speculated that those waking in the morning with a bilious attack may have had a nocturnal seizure.

The aetiology of post-epileptic headaches is uncertain. Physical exertion which can provoke headaches

after sexual intercourse¹⁴ and running¹⁵ seems unlikely to be the cause as minor fits involve minimal muscular activity, similarly hypoxia which is involved in the headaches of mountain sickness¹⁶ does not occur after minor seizures.

Post-epileptic headaches may be related to intracranial vascular changes known to be associated with seizures. In dogs and monkeys Plum *et al*¹⁷ described a four-fold increase in cerebral blood flow during chemically induced seizures which only persisted for 2 minutes after the cessation of the fit. Gibbs *et al*¹⁸ first demonstrated an increased cerebral blood flow in patients during spontaneous fits in 1934 using a thermoelectric flow recorder inserted into the jugular vein; within seconds of the onset of a major seizure, blood flow increased rapidly and remained at a high level for approximately one minute after the fit ceased; during the subsequent 6 minutes, flow gradually returned to the preconvulsive level. Similar changes were seen in some patients with minor seizures, but in one patient no change in blood flow occurred during a minor attack. More recent studies using both intra-arterial xenon injection¹⁹ and positron emission scanning²⁰ have confirmed the rise in blood flow during seizures.

Penfeld and Jasper²¹ studied the exposed human cerebral cortex during and after fits: during seizures visible pulsations in the pial arteries disappeared over the whole cerebral hemisphere; towards the end of the fit, these pulsations returned, often becoming more pronounced post-ictally than before the fit. When respiratory embarrassment occurred during a seizure the cerebral veins became swollen and "the brain seemed to enlarge due to the venous engorgement within the cranial cavity". In addition they frequently also noted "reactive post-ictal hyperaemia" in the region of the discharging focus. Some minutes after the end of the fit bright red blood was seen in large pial veins persisting for up to 30 minutes.

This increased blood flow probably involves more than one process. Plum *et al*¹⁷ found that most of the increased flow was abolished by cervical cord transection and argued that the blood flow changes were secondary to the loss of autoregulation due to the increased blood pressure during the fit. Penfield²¹ however thought chemical factors released from the discharging focus were responsible.

The explanation of why 50% of patients never experienced post-seizure headaches is not clear. It is unlikely that these patients forget their post-epileptic symptoms because the other 50% were able to give a surprisingly clear account of their pain. Perhaps only some patients have marked blood flow changes after seizures. This hypothesis could be further studied to see if the presence of post-seizure headache correlated with the changes in blood flow.

Although the mechanisms underlying migraine attacks are still unclear, it seems likely that they have a primary neural basis.²² Leao's spreading cortical depression has been suggested as an explanation for the progression of the visual aura.^{23 24} Spreading depression in experimental animals causes marked vascular changes in pial arteries as well as veins overlying the involved cerebral cortex.²⁵ Arterial diameter increased by 50–100% and as Leao stated "the flow in the veins is strikingly increased and these vessels promptly became as scarlet as the arteries". Following this marked vasodilatation which lasts only one and a half to three minutes, a minor, but prolonged, reduction of cerebral blood flow occurs.²⁶ The observed transient vasodilatation is very similar to that seen after seizures.

In addition to describing spreading electrical depression after mechanical stimulation of the rabbit cortex, Leao also mentioned a second associated electrical phenomenon: "often during the period of depression of a region... fast components may also appear and the activity when intense closely resembles the seizure pattern of experimental epilepsy". Furthermore, he viewed the depression and the "tonic-clonic" activity of experimental epilepsy as closely related phenomena.²³ However, blood flow studies during migraine attacks have given conflicting results, some authors reporting oligoemia²⁷ and others hyperaemia.²⁸

The features of post-epileptic headache conform to a pattern of pain assumed to be of intracranial origin, other examples being migraine, meningitis and histamine headache. Histamine injected into the external carotid artery did not cause headache, whilst injection into the common and internal carotid artery resulted in pain.²⁹ Characteristic differences between headaches of intracranial and extracranial origin have been previously proposed by Blau and Dexter.³⁰

In conclusion, post-convulsive headaches occurred regularly in 50% of epileptics and shared many features associated with the headache phase of migraine. Post-epileptic headaches may offer a migraine model worthy of further study.

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