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Short report

Asymmetry of the aura and pain in migraine

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SUMMARY Patients with classical migraine whose auras included paraesthesiae or numbness in the hands have been reviewed. In 55 of 111 patients the symptoms were on the same side of the body as the headache and in only 20 on the opposite side to the headache. In the remaining 36 patients one or other was incompletely lateralised. Five right handed patients described dysphasia at the same time as paraesthesiae in the their non-dominant hand. These findings are incompatible with the notion that the headache is due to reactive hyperaemia following localised cerebral ischaemia, and it is suggested that the ischaemic and hyperaemic processes are both the result of some more generalised vasomotor disturbance.

Unilateral headache is a common, but not diagnostic feature of migraine, and the premonitory symptoms experienced by patients with classical migraine are often asymmetrical. No clear explanation for the association of such symptoms and headache is yet available. We have surveyed consecutive patients attending the Princess Margaret Migraine Clinic to examine the relationship between the side of the aura and the side of the subsequent headache.

Results

Six hundred and fifty patients complaining of headache were seen between 1st January and 2nd October, 1980. Migraine was diagnosed in patients having two or more of the cardinal symptoms: unilateral headache, aura symptoms, nausea, or a family history.[1] Two hundred and fifty-eight of the 650 patients had migrainous headaches with an aura, and were diagnosed as classical migraine. In 223 patients there was no aura and a diagnosis of common migraine was made. Thirty-four patients had cluster headache and 13 had headaches considered of structural origin. Most of the remainder had tension headache. Two hundred and twelve of the classical migraine patients experienced visual symptoms before the headache, 111 had paraesthesiae or numbness or both, and 19 patients described transient localised limb weakness. We have studied only the patients with sensory or motor symptoms as these could be more reliably lateralised.

The side of the sensory symptoms and of the subsequent headache in the 111 patients is shown in table 1. Fifty-five patients had symptoms predominantly on the same side as the headache, whereas 20 had them on the opposite side. In 36 patients either the headache or the sensory symptoms were bilateral or inconsistently localised. The predominance of ipsilaterality over contralaterality is statistically significant (χ^2 with Yates' correction = 7·678; $p < 0·01$). In two patients, only a minority of attacks were preceded by limb paraesthesiae, and these classical migrainous headaches were on the opposite side to the patients' more usual common migrainous headaches. In one case the symptoms were ipsilateral, and in the other contralateral to the subsequent headache. One subject described paraesthesiae in the fingertips of the left hand, which was followed by right sided headache lasting 15 minutes, and then a much more severe left sided headache that lasted for 36 hours.

Transient limb weakness was described by 20 patients, of whom 16 had paraesthesiae as well. Of the 20 subjects, 11 had unilateral headache, and of these nine had both motor and sensory symptoms. In all nine the motor and sensory symptoms were on the same side; in seven cases on the same side as the headache and in two cases on the opposite side.

Many patients reported disturbances of speech

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Table 1 *Side of sensory symptoms*

	Right	*Left*	*Either side, unilateral*			*Bilateral*
			Ipsilateral	*Contralateral*	*Either side*	
Side of headaches						
Right	23	10	—	—	1	4
Left	9	28	—	—	—	5
Either side unilateral	1	—	4	1	1	10
Bilateral	4	4	—	—	—	6

during the aura phase of their attacks. Any patient with dysarthria has been excluded leaving 29 with dysphasia as well as paraesthesiae. Two of these used the left hand for writing and five described mixed handedness. Of the 22 right handed dysphasic subjects, nine had both headache and paraesthesiae on the right and five on the left. Only two had left sided pain and right sided paraesthesiae, and in the remaining seven either the headache or the limb symptoms were bilateral. Six of the 20 patients with limb weakness were also dysphasic. Two of these were left handed and each had both motor and sensory symptoms on the same side as the headache, one each on the left and on the right. One subject was of mixed handedness and described left motor and sensory limb symptoms and a right sided headache. One of the right handed subjects had right sided motor and sensory symptoms and headache, and is included in the 22 subjects above; one patient had left arm weakness without paraesthesiae and right sided headache, and another right arm weakness without paraesthesiae and bilateral headache.

Discussion

Although the classical visual aura of migraine is clearly lateralised, a high proportion of our patients described poorly localised formless "flashings" and we considered that the study would be more reliable if we included only patients with limb symptoms. Although limb paraesthesiae may be induced by hyperventilation during migraine attacks, such symptoms are generally bilateral.[2] We have demonstrated that about two-thirds of our patients have neurological symptoms that arise in one cerebral hemisphere followed by a headache overlying the opposite hemisphere; in only one-third are the two processes taking place on the same side of the head. This finding is at variance with the studies of Bradshaw and Parsons,[3] and Bruyn and Weenink[4] (table 2): in neither of these series does the predominance of contralaterality of headache and paraesthesiae become statistically significant, though if all three series are added together the numbers with ipsi and contralateral headache are exactly equal.

Bruyn[4–6] has long favoured a single anatomical site, lateral to the posterior internal capsule, for the origin of all the aura symptoms in classical migraine. Our six right handed cases with dysphasia and symptoms in the non-dominant arm suggest multiple sites of origin—a single pathogenetic mechanism with widespread, though asymmetrical, effects is just as consistent with Occam's Razor.[5] Our finding also renders unlikely the hypothesis of Welch *et al*,[7] based on blood flow studies in the monkey, that external carotid vasodilatation can be so extensive that blood is "stolen" from the internal carotid circulation and cerebral ischaemia results.

Much recent work has shown a reduction in cerebral blood flow before the headache develops, in both common and classical migraine.[8–12] In classical migraine this is greatest in the cortical regions corresponding to the symptoms being experienced.[10 12] During the headache phase there is an increase in both intracranial and extracranial blood flow, again both in common and classical migraine.[10 12] All the patients described in these reports, however, have had headaches contralateral to the "ischaemic" symptoms. This hyperaemia persists much longer than the pain, and does not subside if the headache is treated with analgesics.[12] Reactive hyperaemia may well be the explanation for the increase in intracerebral blood flow, but our findings suggest that it cannot explain

Table 2 *Published series of laterality*

	Peatfield et al 1981	*Bradshaw and Parsons* 1965[3]	*Bruyn and Weenink* 1966[4]	*Total*
Contralateral	?0	35	36	91
Ipsilateral	55	17	19	91
Total	75	52	55	182
χ^2 Yates correction	7·678	2·537	2·095	0
p	< 0·01	ns	ns	ns

the unilateral increase in extracerebral blood flow confined to the side of the pain.[13] There is abundant evidence that this is the site of the headache.[11 14 15] Any proposed mechanism for this must explain both the majority, and the significant minority of patients who *do* have cortical ischaemia and headache on the same side of the head. A direct connection between the cerebral ischaemia and the headache appears less probable than both being secondary to some other mechanism.

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