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Mechanisms of increased sensitivity to noise and light in migraine headache

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To determine whether phonophobia is a manifestation of loudness recruitment, the hearing and auditory discomfort thresholds to an 8000 Hz tone were measured during the headache-free interval and again during an attack of migraine in 16 migraine sufferers. The visual discomfort threshold was also determined. For comparison, measures were taken in 10 non-headache controls of similar age and sex distribution. Auditory and visual discomfort thresholds decreased substantially during attacks of migraine. Increases (three subjects) or decreases (three subjects) in hearing threshold during attacks of migraine were significantly greater than the variation recorded in control subjects from Session 1 to Session 2. The findings do not support the view that phonophobia in migraine is a manifestation of loudness recruitment, although cochlear disturbances might mediate hearing loss in some cases. Disruption of central sensory processing mechanisms during migraine could increase sensitivity to quiet sounds, and contribute to phono- and photophobia. € Loudness recruitment, migraine, phonophobia, photophobia

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Most migraine sufferers develop an intolerance to light (photophobia) and sound (phonophobia) during headache, preferring to wait out their attack in a quiet, dark room. Kayan and Hood (1) suggested that phonophobia in migraine might have the same pathology as loudness recruitment (an abnormally rapid increase in loudness with increase in intensity of sound), which accompanies cochlear disturbances. Patients with loudness recruitment dislike loud noise because it sounds distorted. In patients with unilateral cochlear hearing loss, loudness in the recruiting ear generally catches up with and matches loudness in the normal ear at high sound levels (2). Thus, there is a reduced dynamic range between the hearing threshold and the level at which sounds become uncomfortably loud.

If phonophobia in migraine is a manifestation of loudness recruitment, perception of quiet sounds should deteriorate during attacks. However, some migraine sufferers describe their aversion to noise as an apparent *improvement* in auditory sensitivity, as though they could "hear a pin drop in the next room". They also describe an increased sensitivity to light and, more rarely, a heightened sense of smell, suggesting that a general hyperexcitability of the special senses develops during attacks (3). If so, phonophobia could be mediated by an increase in excitability of auditory neurons, possibly through disruption of normal inhibitory mechanisms or facilitation of excitatory mechanisms in the brainstem or higher centres. Unlike the loudness recruitment hypothesis, this explanation of phonophobia would account for the apparent increase in auditory sensitivity during attacks of migraine.

The aim of the present study was to investigate auditory sensitivity in relation to phonophobia and photophobia in migraine. A decrease in sensitivity would be consistent with the loudness recruitment hypothesis, whereas an increase in sensitivity would suggest that phonophobia is mediated centrally.

Method

Subjects

The migraine sample consisted of 5 males and 11 females aged between 20 and 66 years (mean age 45 years) who satisfied the diagnostic criteria for migraine-with-aura (9 patients) or migraine-without-aura (7 patients) (4). The control sample consisted of 5 males and 11 females aged between 23 and 71 years (mean age 41 years) who reported less than 12 headaches per year with no more than two headaches being considered severe. Each subject provided informed consent for the procedures, which were approved by the Murdoch University ethics committee.

Procedures

To determine the hearing threshold, tones from a 1705 Grason-Stadler audiometer (8000 Hz, 1 sec duration, 2 sec interstimulus interval) were presented simultaneously to both ears via Danplex audiocup headphones calibrated for the audiometer. Hearing threshold was tested at 8000 Hz because higher frequencies are often lost first in hearing deficits (5); thus, a change in perception during migraine was more likely to be detected at this frequency than at lower frequencies. The experimenter increased the intensity of the tone from well below hearing threshold until the subject heard it. The experimenter then adjusted the volume of the tone around this level in 2 dB hearing level (HL) steps until a stable response was obtained for five consecutive presentations (the staircase method). The hearing threshold was defined as the level at which the subject heard the tone 50% of the time. This value was found to be reliable in control subjects (test-retest reliability, $r(15) = 0.98, p < 0.001$). To differentiate the subject's response bias from his or her actual sensitivity, a signal detection procedure was then run (two-alter-native forced-choice), using the hearing threshold established by the staircase method. However, the data from this procedure were unreliable in control subjects, and will not be reported.

To determine the auditory discomfort threshold, the experimenter increased the volume of the tone in 2 dB HL steps from the hearing threshold to a maximum of 130 dB, or until the subject expressed discomfort and requested no further increase in loudness. The following instructions were read to each subject: "In this test I want to find out the loudest noise that you can stand. The sound will be the same sort of brief tone that you heard in the other tests but it will gradually get louder. You will be able to stop the test at any stage and the sound will never be loud enough to damage your ears. I will start with the sound at a quiet level and slowly increase the volume. As it increases I want you to tell me what it sounds like. For example, when the sound is quiet you might say 'it's quiet now'. As it gets louder you might say 'it's pretty loud now but it's not uncomfortable yet'. When the sound is causing you discomfort you should say 'stop now, it's too uncomfortable'." The auditory discomfort threshold was calculated from the average of three trials, and was found to be reliable in control subjects (test-retest reliability $r(15) = 0.91, p < 0.001$).

To determine the visual discomfort threshold, the experimenter increased the brightness of a 130 watt quartz halogen globe in 35 steps from 1.5 lux (dull) to a maximum of 2900 lux (measured with a Gossen Profi-six light meter at the position of the subject's eyes in the light chamber), or until the subject expressed discomfort and requested no further increase in brightness. The globe was mounted in a light chamber 64 cm from the subject's eyes. The step size ranged from 5 lux (at the lower end of the scale) to 500 lux (at the upper end of the scale). To minimize adaptation, the light was switched on for 2 sec at each intensity, with an interstimulus interval of 2 sec in darkness. To minimize the risk of inducing headache in migraine sufferers, the visual discomfort threshold was calculated from one trial. This threshold was found to be reliable in control subjects (test-retest reliability, $r(14) = 0.96, p < 0.001$).

All subjects were free from headache in the first session. Migraine sufferers were asked to phone during an attack so that the tests could be repeated. Migraine sufferers were tested in a quiet room of their own home during and between attacks, to avoid problems of travelling with a migraine. Ambient noise was kept to a minimum on both measurement occasions. Control subjects were tested in a sound-attenuated room at the university.

Data analysis

The visual discomfort threshold was transformed to log units, because perceived brightness is an exponential function of light intensity (6). Loudness was also expressed in a logarithmic scale (dB).

The hearing and discomfort thresholds were investigated with analyses of variance (MANOVA programme, SPSSX). Each analysis had one between-groups factor (migraine versus control subjects), and one repeated measures factor (the first versus the second session, corresponding to headache-free versus headache in migraine sufferers).

Results

Thresholds of auditory and visual discomfort fell substantially during attacks of migraine (Figs. 1 and 2). For both discomfort thresholds, the interaction between group and session was statistically significant (for hearing, $F(1,30) = 18.45, p < 0.001$; for vision, $F(1,79) = 13.04, p < 0.01$). During the first session, when migraine subjects were headache-free,

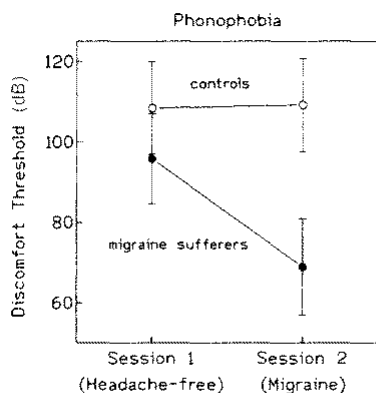


Fig. 1. Auditory discomfort threshold in migraine sufferers and control subjects. The auditory discomfort threshold fell significantly during attacks of migraine headache.

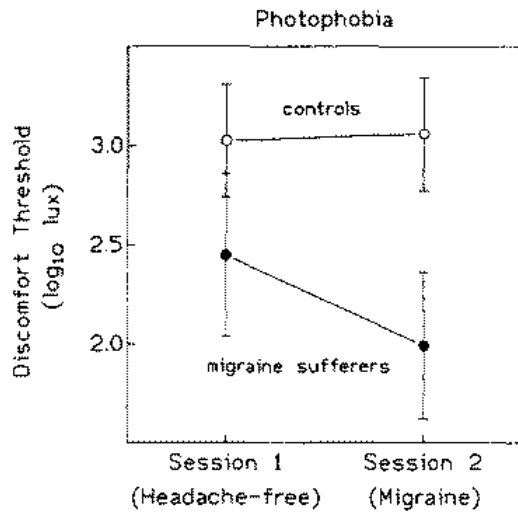


Fig. 2. Visual discomfort in migraine sufferers and control subjects. The visual discomfort threshold was lower in migraine sufferers during the headache-free interval than in controls, and fell significantly during attacks of migraine headache.

the visual discomfort threshold was lower in migraine sufferers than in controls [$t(29) = 2.45, p < 0.05$] (Fig. 2); however, the auditory discomfort threshold did not differ significantly between groups. Subjects with a low visual discomfort threshold when headache-free also had a low visual discomfort threshold during migraine [$r(15) = 0.77, p < 0.001$]. In contrast, the threshold of auditory discomfort during migraine was not related to the auditory discomfort threshold when headache-free [$r(15) = 0.46$, not statistically significant].

During the headache-free interval, the hearing threshold ranged between -2 dB and 24 dB, whereas during migraine the threshold ranged between -10 dB and 50 dB. The range in hearing threshold was similar in control subjects (-8 dB to 72 dB in Session 1, and -6 dB to 86 dB in Session 2). In comparison to the variation from the first to the second session in control subjects, the hearing threshold increased significantly during migraine in three subjects and decreased significantly in three others (Table 1). In the group as a whole, the hearing threshold did not increase or decrease consistently during attacks [mean threshold 10.2 dB HL on both measurement occasions, test-retest reliability $r(15) = 0.76, p < 0.001$]. Individual changes in the hearing threshold were unrelated to diagnosis, headache intensity, duration, association with gastrointestinal disturbances, or consumption of medication (Table 1). Correlations between the change in the hearing threshold during migraine and visual and auditory discomfort thresholds were not statistically significant. Four migraine sufferers reported that they were much more sensitive to sound (i.e. sounds were louder) during the attack than at other times (Table 1). The other 12 subjects reported that they were

Increase in hearing threshold

Table 1. Change in hearing threshold during attacks of migraine headache.

Change in hearing threshold (dB)	Features of the attack							
	Diagnosis	Aura	Nausea	Photophobia	Phonophobia	Intensity	Duration	Medication
1. 28*	aura	+	+	+	s+	severe	7 h	ergotamine
2. 24*	aura	+	+	+	+	severe	3 h	analgesics
3. 12*	no aura	-	++	+	+	severe	6 h	ergotamine
4. 4	no aura	-	+	+	-	moderate	14 h	ergotamine
5. 4	aura	-	++	+	+	moderate	12 h	ergotamine
6. 2	no aura	-	+	+	+	moderate	21 h	analgesics
<i>Decrease in hearing threshold</i>								
1. 2	aura	-	-	+	s+	moderate	2 h	nil
2. 2	no aura	-	+	+	+	moderate	38 h	analgesics
3. 4	aura	+	-	+	+	moderate	1 h	ergotamine
4. 6	no aura	-	+	-	-	severe	6 h	nil
5. 6	aura	+	+	+	+	moderate	1 h	analgesics
6. 8	aura	-	-	+	-	moderate	6 h	nil
7. 8	aura	+	+	+	s+	severe	28 h	analgesics
8. 10*	aura	-	++	+	s+	severe	2 h	analgesics
9. 14*	no aura	-	+	+	+	moderate	4 h	nil
10. 14*	no aura	-	-	-	+	moderate	11 h	analgesics

* Significant change in hearing threshold ($p < 0.08$). In 16 control subjects, the 5% and 95% confidence limits for variation in the hearing threshold from Session 1 to 2 were ± 8.8 dB.

Nausea: ++ refers to nausea with vomiting.

Photo- and phonophobia: visual or auditory discomfort threshold below the 95% confidence interval in control subjects during Session 2. For phonophobia, "s" refers to ratings of "much more sensitive to sound" during the attack than before.

somewhat more sensitive to sounds during the attack.

Discussion

The findings confirmed that most subjects were intolerant of loud noise and bright light during migraine. This study is the first to quantify phono-phobia during migraine, a prerequisite to exploring mechanisms that could be of fundamental importance in the genesis of this disorder.

Kayan and Hood (1) interviewed 200 unselected migraine sufferers about vestibulocochlear symptoms associated with migraine. They noted that five patients experienced phonophobia despite some apparent hearing loss during attacks. On the basis of these five cases, Kayan and Hood suggested that phono-phobia is a manifestation of loudness recruitment, resulting from a transient disturbance of cochlear receptors. Vestibulocochlear dysfunction was attributed to vasoconstriction and ischaemia in the territory of the vertebrobasilar artery during migraine. In the normal cochlea, an active physiological process seems to boost the neural response to low-intensity sounds. The outer hair cells of the cochlea are thought to mediate this active process by amplifying the vibration of the basilar membrane (7). If the active process is lost, quiet sounds are not amplified and the detection threshold is raised; however, the basilar membrane response to high level sounds remains relatively unchanged. Moore (2) suggested that damage to outer hair cells contributes to cochlear hearing loss and loudness recruitment. Cerebrovascular disturbances during migraine (for example, see 8) might disrupt the normal functioning of the cochlea.

The present study documents a decrease in auditory sensitivity in three subjects during an attack of migraine (preceded by a visual aura in two). Hearing loss in migraine has been noted previously (9), although in most cases hearing is normal during the headache-free interval (10). Olsson (10) attributed hearing loss and other audiovestibular symptoms in migraine to a combination of peripheral end-organ and central disturbances of neurovascular origin. He suggested that misinterpretation of sensory information by the central nervous system generates inappropriate neurovascular reflexes which, in the long term, might lead to permanent injury to the audio-vestibular system.

In contrast to patients with loudness recruitment from cochlear injury (2), the auditory discomfort threshold *decreased* substantially during attacks of migraine; in patients with unilateral cochlear injury, sounds quickly become loud in the recruiting ear, but loudness at high intensities is normal (2). Furthermore, in most cases of migraine, phonophobia was not associated with binaural hearing loss for low-intensity sounds. Because of practical problems of carrying out a full audiometric analysis during attacks of migraine, both ears were tested simultaneously and at only one frequency. Thus, partial hearing loss may not have been detected in this study. Nevertheless, the *decrease* in hearing threshold during migraine in three cases suggests that some mechanism other than loudness recruitment contributes to phonophobia.

Lance et al. (11) suggested that brainstem centres such as the locus coeruleus and raphe nuclei influence the vascular changes and pain of migraine; heightened activity of the locus coeruleus might also facilitate sensory processing. The cerebral cortex is densely innervated by noradrenergic fibres from the locus coeruleus. Locally applied noradrenaline facilitates cortical neuronal responsiveness by increasing the signal-to-noise ratio of cortical input, and prevents adaptation to trains of action potentials (12). In addition, release of noradrenaline in the thalamus inhibits rhythmic burst discharges, which increases the likelihood of accurate transfer of incoming spike trains to the cerebral cortex (12). Discharge of the locus coeruleus could therefore increase auditory sensitivity to quiet sounds, and might also increase discomfort to loud sounds through a "sensory overload" phenomenon. Alternatively, disruption of normal inhibitory influences on sensation might reduce auditory and visual discomfort thresholds.

As noted previously (13, 14), the threshold of visual discomfort was lower in migraine sufferers during the headache-free interval than in controls. In normal volunteers, irritation of the region supplied by the ophthalmic division of the trigeminal nerve facilitates photophobia (15), possibly because of interaction between trigeminal and visual inputs in the brainstem, thalamus or higher centres. Excitability of the trigeminal system might persist sub-clinically between migraine episodes. We recently reported that moderate forehead pain increased visual discomfort in migraine sufferers during the headache-free interval, but not in controls (14). Migraine sufferers might also have a low tolerance for bright light because of anxiety about light inducing a headache.

In conclusion, the present findings indicate that phonophobia in migraine is usually not accompanied by binaural hearing loss for quiet sounds. Thus, phonophobia in migraine is probably not a manifestation of loudness recruitment. Aversion to loud noise, and increased sensitivity to quiet sounds, could be mediated by disruption of central sensory processing mechanisms. An increase in latency of the fifth wave of the brainstem auditory evoked potential during migraine (16, 17) is consistent with this hypothesis.

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