Transient ischaemic attack with trigeminal autonomic symptoms

Trigeminal autonomic cephalalgias present with excruciating headaches and accompanying autonomic features such as ipsilateral lachrymation, rhinorrhoea, and eyelid ptosis. For clinical purposes, these headaches are subclassified into cluster headaches, paroxysmal hemicranias, and the SUNCT syndrome (sudden unilateral neuralgiform headache with conjunctival injection and tearing),[1] of which the paroxysmal hemicrania subtypes such as the acute and chronic hemicranias are indomethacin responsive. Neuroimaging is often normal in trigeminal autonomic cephalalgias; nevertheless MRI should be considered, as there are associations between trigeminal autonomic cephalalgia and multiple sclerosis,[2] lateral medullary infarction,[3] pontine tumours,[4] basilar aneurysms,[5] and other posterior fossa lesions. We present an unusual case of recurrent transient ischaemic attacks (TIA) with symptoms reminiscent of a trigeminal autonomic cephalalgia which ceased after a ponto-mesencephalic infarct.

Case report

A 65 year old women was admitted with a three day history of stereotyped neurological episodes. These were characterised by a burning sensation in the left side of face, congestion of the left eye, lachrymation from the left eye, watery discharge from the left nostril, slurring of speech, and difficulty in walking. These episodes were not accompanied by headache. Each episode lasted for around 30 minutes and occurred three or four times a day. On examination, there were no focal neurological deficits. In hospital, she had four more episodes. As she had a left mature cataract (fig 1), the possibility of glaucoma induced by lens rupture was considered and ruled out. Computed tomography of the brain was unremarkable. The possibilities considered were a vertebro-basilar TIA and a trigeminal autonomic cephalalgia. As she had additional neurological symptoms, a TIA was considered more likely than trigeminal autonomic cephalalgia, and she was started on aspirin and intravenous heparin. Indomethacin was begun at 75 mg/day but had to be discontinued after a single dose because of gastric distress. Two days later, her episodic symptoms disappeared; however, she now complained of persistent right sided weakness and difficulty in walking. On examination, she had a new left sided Horner's syndrome, right upper motor neurone facial palsy, and right upper limb weakness. Deep tendon reflexes were brisker on the right than on the left. Magnetic resonance imaging of the brain at this time

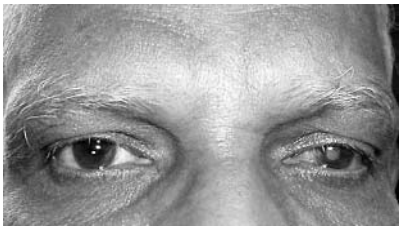

Figure 1 Congestion of the left eye during an acute attack. The patient gave written consent for this photograph to be reproduced.

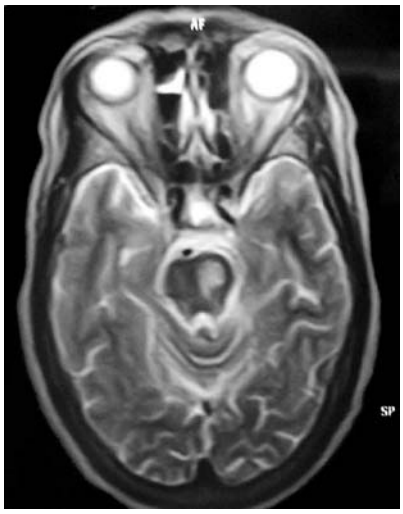

Figure 2 T2 Weighted magnetic resonance image showing an acute left ponto-mesencephalic infarct.

(one week after the onset of symptoms) showed an acute infarct at the left ponto-mesencephalic junction (fig 2). At follow up 10 days later, her gait had improved. Her episodic symptoms had disappeared.

Comment

Cranio-facial autonomic symptoms such as ipsilateral lachrymation, rhinorrhoea, conjuctival congestion, ptosis, or eyelid oedema are the sine qua non of trigeminal autonomic cephalalgia. It is thought that activation of the trigeminal afferent system generates pain, and co-activation of the VIIth nerve efferent parasympathetic pathway produces the autonomic manifestations such as ipsilateral lachrymation and rhinorrhoea. A mechanism analogous to this can be evoked to explain our patient's symptoms. She had an ischaemic stroke in the ponto-mesencephalic junction. Ischaemia in this region could involve the trigeminal main sensory and mesencephalic nuclei, resulting in facial dysaesthesia comparable to ischaemic nerve pain. In contrast to the ''boring'' intense pain often encountered with trigeminal autonomic cephalalgias, the burning dysaesthesias are more consistent with ischaemia. Ischaemia could also trigger trigemino-facial synapses in the superior salivatory nucleus and switch on the facial efferent parasympathetic pathway, resulting in ipsilateral lachrymation and rhinorrhoea.

To the best of our knowledge, this is the first report of a TIA presenting with trigeminal sensory-autonomic symptoms. This report highlights the expanding spectrum of trigeminal autonomic cephalalgias and emphasises the need to rule out a vertebrobasilar TIA in elderly patients with a new onset of trigeminal autonomic cephalalgia, especially if additional neurological symptoms are present.

B V Maramattom
Department of Neurology, Lourdes Hospital, Kochi, South India; bobvarkeys@yahoo.com

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Tympanic measurement of body temperature in stroke patients ''turned on its ear''

Body temperature is an important prognostic factor in acute stroke, and it is a marker of life threatening infections such as pneumonia.[1] In clinical practice rectal temperature is regarded as a valid and reliable indicator of body temperature, but in the past 15 years tympanic infrared thermometers have been introduced into widespread clinical use. Tympanic temperature measurement is easier, faster, and less invasive than rectal measurement. However, concerns remain about the reliability and validity of this method.[2]

The product manual of the tympanic thermometer warns against overestimation of body temperature when the patient has been lying on one ear. Many stroke patients are hemiparalytic or hemiparetic and therefore may lie on one ear. The aim of our study was to investigate the error in tympanic temperature measurements in patients who have had a stroke and have been lying on one ear in this way.

For this observational study we included patients who had had an ischaemic or haemorrhagic stroke and were admitted to the stroke unit of a university medical centre or to the stroke rehabilitation unit of an affiliated nursing home, both in an urban area. Exclusion criteria were inability to lie on one ear and absence of a rectal cavity. We used the NIH stroke scale (NIHSS) to assess stroke severity.[3] No follow up was conducted.

Tympanic temperature was measured in both ears after the patient had been lying on one ear of choice for at least 15 minutes (first measurement). The measurements were carried out in patients on waking in the morning or after an afternoon nap. The ear the patient had been lying on and the other ear will be further referred to as the lower ear and the upper ear, respectively. The second measurement was conducted by a different investigator—who was not aware of the results of the first measurement—approximately 10 minutes after the patient had no longer been lying on one ear, and consisted of tympanic and rectal temperature measurements.

To assess reliability we computed the difference between lower and upper ear temperature in consecutive tympanic measurements and analysed this difference graphically, as described by Bland and Altman.[4] Validity was assessed by comparing the mean

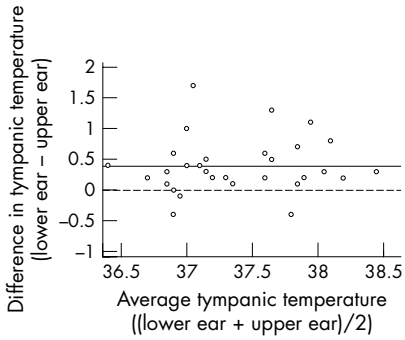

Figure 1 Difference in tympanic temperature taken from the upper and lower ear, plotted against the average tympanic temperature taken from both ears (Bland–Altman plot). The solid horizontal line in the centre indicates the mean difference between the tympanic temperatures (0.39˚C), while the dashed horizontal line indicates no difference (the null hypothesis).

tympanic temperatures with mean rectal temperature.

We studied 30 patients (nine male, 21 female). Their mean age was 69.5 years, and their median NIHSS score was 11, ranging from 0 to 38. The mean of the first tympanic temperature taken from the lower ear was 37.6˚C. The mean tympanic temperatures taken from the upper ear were both 37.2˚C and the second measurement from the lower ear had a mean of 37.3˚C. Mean rectal temperature was also 37.3˚C. The mean difference between the two ears was 0.39˚C (95% confidence interval, 0.22 to 0.56). This difference ranged from −0.4˚C to 1.7˚C (fig 1). The mean difference between the first measurement taken from the lower ear and the rectal temperature was 0.29˚C (0.13 to 0.45).

Comment

Our study showed a clinically significant difference between tympanic temperature measurements in the two ears after a stroke patient had been lying on one ear. This difference disappeared after a while when the patient was no longer lying on one ear. There was no relation between actual body temperature and the size of the measurement error. Although the manufacturer of the tympanic thermometer cautions against heating of the auditory canal when the patient has been lying on one ear, especially in children, neither this effect nor its magnitude is well known.

In our opinion, a systematic error of 0.4˚C on average is not acceptable from a clinical point of view. It may lead to unnecessary investigations and treatment with antibiotics in a considerable number of patients; moreover, an error of this size could have decreased the statistical power of clinical trials of temperature lowering treatment in acute stroke patients, if tympanic temperature was used without attention being paid to the side the patient was lying on.[5]

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A J J Rampen, E J van Breda, D W J Dippel
Department of Neurology, Erasmus Medical Centre, Rotterdam, Netherlands

Correspondence to: Dr Eric J van Breda, Department of Neurology, Erasmus Medical Centre, PO box 1738, 3000 DR Rotterdam, Netherlands; http://www.pais-study.org/

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BOOK REVIEW

Brain fiction, self-deception and the riddle of confabulation

Edited by William Hirstein. Published by Wiley, 2004, £22.95 (hardcover), pp 288. ISBN 0262083388.

The striking neuropsychological symptom of confabulation represents a prototypical form of false remembering and as such has stimulated great interest among neurologists, psychiatrists, and neuropsychologists. *Brain Fiction* by William Hirstein is the first book to place confabulation at the centre of its attention. By doing so, Hirstein is faced with the challenge of presenting and examining the various discussions surrounding the definition, the subtypes, the neural, and cognitive basis of confabulation and crucially the relation between its various forms and manifestations. *Brain Fiction*, however, has taken up further challenges. By borrowing and integrating data and notions from both neuroscience and epistemology, Hirstein puts forward an original definition and model of confabulation, as a dynamic interplay of creative and ''checking'' mental processes. More generally, Hirstein chooses confabulation as a promising template for the formulation of an interdisciplinary dialogue and interchange of ideas between neuroscience, psychology, and philosophy. In addition, the book proposes to hold a place for confabulation in a continuum of behaviours, ranging from ''normal'' other, and self-deception attempts in everyday life to deficits of theory of mind, awareness, and symptoms of sociopathy.

It should be evident from the above, that *Brain Fiction* is addressed to professionals of diverse fields and Hirstein has tried to accommodate the potential clefts in acquaintance with expert knowledge and technical terms. However, the specialised reader should keep in mind that the book does not offer an examination exhaustive in content or encyclopaedic in format. The book is of limited interest to clinicians. It mainly aims at disentangling confabulation from the strict boundaries of its hitherto neuroscientific examination and exposing it to direct philosophical enquiry. This is an endeavour that promises mutual interdisciplinary benefits. Yet the author, perhaps motivated by the existing lack of theoretical and descriptive consensus on the subject, also chooses to propose a new aetiological account of the phenomenon in neuroscientific terms. Inevitably, this analysis often entails smoothing of the hard edges of some conflicting neuroscientific findings, and partial coverage of some complex issues raised by confabulation, such as its implications for theories of consciousness, self-formation, and motivation. Such selectivity though has noteworthy benefits. The book introduces an unprecedented emphasis in the study of confabulation by placing the definition, taxonomy, and implications of the phenomenon into epistemological perspective. Thus, it sets the ground for fruitful neuro-philosophical discussions and it refreshes the way neurologists, psychiatrists, and psychologists view this and other related symptoms.

A Fotopoulou