SHORT REPORT

Measurement of pulse pressure profiles in patients with trigeminal neuralgia

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J Neurol Neurosurg Psychiatry 2003;**74**:533–535

Applanation tonometry is a non-invasive method of assessing the arterial blood pressure profiles in both the peripheral and systemic circulation. In this study the authors examined whether there were differences in these profiles in patients with trigeminal neuralgia. The carotid artery and derived aortic blood pressure waveforms were obtained using a pulse wave analysis system. The ratio of the pressure wave amplitude above the systolic shoulder to the total systolic blood pressure (augmentation index, AIx) was recorded. Thirty two patients with trigeminal neuralgia (16 male and 16 female) and 100 controls (50 male and 50 female) were recruited. Eleven patients had been treated by microvascular decompression, mean (SD) time from surgery 17 (24) months (range 3–86 months). For the patients with trigeminal neuralgia, the right and left caratid artery AIxs (mean (SD)) were 120.6 (21.7)% and 120.7 (19.1)% respectively. Corresponding values for the control group were 120.5 (19.3)% and 120.9 (19.5)%. The calculated AIx for the ascending aorta was 27.7 (10.1)% and 27.2 (10.5)% for the patients with trigeminal neuralgia and controls respectively. No significant differences were seen in either the right or left carotid artery (p=0.5 and p=0.6 respectively) or the derived ascending aorta (p=0.8). The results show that there does not seem to be a generalised increase in arterial stiffness in patients with trigeminal neuralgia.

Trigeminal neuralgia has an annual incidence of about 4.5 per 100 000.[1] Although several causes of trigeminal neuralgia have been identified, its basic pathophysiology is still not well understood. Patients with trigeminal neuralgia may have arterial tortuosity,[2] which could lead to increased arterial pulse pressure waveforms due to vascular rigidity. The most common cause of trigeminal neuralgia is focal compression of the trigeminal nerve by an aberrant loop of artery or vein, which is found in 80%–90% of cases.[1] This anomaly has been reported to occur more frequently in patients with hypertension,[3] and when coupled with the fact that trigeminal neuralgia is age related and rarely affects anyone under the age of 50, the importance of haemodynamic stress as a factor in trigeminal neuralgia is plausible.

The measurement of blood pressure does not take into account the potential importance of the pulse pressure profiles, which determine the arterial stresses within the arterial walls. Arterial stiffness is recognised as an important risk factor in vascular disease.[4] Pulse wave analysis provides a means of quantification of the vascular compliance, particularly useful in general vascular risk assessment. Applanation tonometry is a quick and comparatively simple procedure for the non-invasive recording of arterial pulse profiles, providing information on wave reflection and producing an index of arterial stiffness.[5] The method depends on the physical principle that the pressure within a viscero elastic cylinder can be estimated by partial compression of that structure permitting transmitted pressure changes to be recorded.

The pulse wave analysis technique uses applanation tonometry to record pressure waves from the peripheral circulation and then generate the corresponding central arterial waveform using transfer functions. The initial transmitted wave and the reflected waves form areas of impedance mismatch and are combined to produce a characteristic waveform. A measure of arterial stiffness, the augmentation index (AIx) can subsequently be determined. This system has been validated for accuracy against the gold standard, as determined by intra-arterial catheterisation in the ascending aorta.[6 7]

Arterial pulse pressure waveforms in patients who have suffered subarachnoid haemorrhage from an intracranial aneurysm have an altered pulse profile compared with matched controls.[8] Pulse pressures in such patients tend to be higher in the left carotid artery than the right. Patients with trigeminal neuralgia have a similar age profile to those with aneurysmal subarachnoid haemorrhage and also a female predominance. In view of these similarities we have examined arterial wave reflections and the pulse profile of the systemic circulation of patients suffering from trigeminal neuralgia to explore the possibility that haemodynamic stress factors may be of importance in trigeminal neuralgia.

METHOD

The local research ethics committees gave approval for this study (LREC 01/185 and RREC 2409). Each patient gave written informed consent.

Patient population

Thirty two patients with trigeminal neuralgia (16 male and 16 female) and 100 controls (50 male and 50 female) were recruited from the Neurosurgical Departments of Charing Cross Hospital, London and Addenbrooke's Hospital, Cambridge. Mean age was 61 years (range 38–86).

Controls were selected from two sources; volunteers composed of staff and visitors to the hospital, and a random selection of patients in the region.

Of the 32 patients with trigeminal neuralgia, six were currently taking carbamezipine, one was taking gabapentin, and seven were taking both. Eleven patients had been treated by microvascular decompression, mean (SD) time from surgery 17 (24) months (range 3–86 months). Nine suffered from symptoms on left and 23 were right sided symptoms.

Instrumentation

A blood pressure analysis system (SphygmoCor, PWV Medical, Sydney, Australia) was used to record the arterial pulse profiles. This comprises of a PWV (Millar Instruments, Houston, TX) pencil probe tonometer with high fidelity micro manometer.

Resting blood pressure was obtained for calibration by using a conventional sphygmomanometer. The monitoring procedure has been previously described.[9 10] A pressure waveform was obtained after palpation of the carotid pulse and by

Table 1 Mean augmentation index and significance of variables in all subjects

	Trigeminal neuralgia	Control	p Value
All subjects	n=32	n=100	
AIx in R CA (%)	120.6 ((21.7)	120.5 (19.3)	0.5
AIx in L CA (%)	120.7 (19.1)	120.9 (19.5)	0.6
AIx in Aorta (%)	27.7 (10.1)	27.2 (10.5)	0.8
Surgically treated	n=11	n=100	
AIx in R CA (%)	125.6 (16.7)	120.5 (19.3)	0.9
AIx in L CA (%)	124.1 (16.4)	120.9 (19.5)	0.8
AIx in Aorta (%)	28.1 (8.2)	27.2 (10.5)	0.4
Medically treated	n=21	n=100	
AIx in R CA (%)	117.9 (23.9)	120.5 (19.3)	0.5
AIx in L CA (%)	119 (20.6)	120.9 (19.5)	0.5
AIx in Aorta (%)	27.5 (11.1)	27.2 (10.5)	0.9

AIx, augmentation index; CA, carotid artery. Values expressed as mean (SD).

positioning the probe, using gentle pressure over the pulse, to flatten but not occlude the artery. The resulting pressure waveform is transmitted on line to a portable computer to assess waveform analysis.

Data analysis

Data expressed as the means (SD). We used a multivariate analysis of variance, to compare parameters, with AIx as the dependent variable and risk factors as covariates. A paired Student *t* test assessed within patient differences of the affected side versus the contralateral side with respect to AIx.

RESULTS

No significant differences were seen in heart rate (trigeminal neuralgia group 70 (12) beat/min, control group 69 (11) beat/min, p=0.53), mean blood pressure (trigeminal neuralgia group 113 (15) mm Hg, control group 108 (15) mm Hg, p=0.13) or pulse pressure (trigeminal neuralgia group 56 (18) mm Hg, control group 56 (19) mm Hg, p=0.94). No significance was reached for either height (trigeminal neuralgia group 1.7 (0.1) m, control group 1.7 (0.1) m, p=0.64) or weight (trigeminal neuralgia group 74 (15) kg, control group 75 (15kg, p=0.64). No patient in either group was reported to be suffering from diabetes mellitus.

Multivariate analysis showed no overall significant difference in the AIx between the subjects with trigeminal neuralgia and the controls (Wilks' λ test =0.95, p=0.32). Taking into account the risk factors known to potentially increase AIx (blood pressure, pulse pressure, cholesterol level, body height, age, and sex), multiple analysis of variance did not show any significant differences in the AIx, between the patients with trigeminal neuralgia and the controls (table 1).

To exclude possible factors that could be associated with a surgical procedure, multiple analysis of variance was repeated for comparison of the surgically treated and medically treated groups, (table 1). No significant differences were found in the AIx in either group after adjustment for risk factors.

Comparison of the AIx in the right and left carotid artery showed no significant intra-subject asymmetry in patients with trigeminal neuralgia when comparing the affected side with the contralateral side (mean 120.9 (21.4) and 120.3 (19.4) respectively, p=0.77).

DISCUSSION

In this study we did not identify any significant differences between the arterial pressure profiles in either the right or left carotid arteries or the systemic circulation, in patients with trigeminal neuralgia compared with controls. We have therefore not identified any association between vessel compliance and the aetiology of trigeminal neuralgia.

Fourteen of the 32 (44%) patients with trigeminal neuralgia were taking antiepileptic drugs for the condition. These drugs act on the central nervous system and are without significant influence on the cardiovascular system. The pulse pressure waveforms are therefore unlikely to have been affected by the drugs. In addition, 11 of 32 (34%) patients with trigeminal neuralgia had been treated surgically. In a subanalysis, the mean AIxs were found to be higher in the surgically treated patients, however there was a greater female: male ratio in this group (10:1 compared with 15:6 in the untreated group). Previous studies have shown that the AIx is higher in women than in men.[11] Taking all factors affecting raised AIx into account, no significant differences were seen in either subgroup.

The controls and patients with trigeminal neuralgia were matched with respect to age and sex. Risk factors such as hypertension, hypercholersterolaemia,[12] and diabetes mellitus,[13] are all known to increase arterial stiffness. This study was started as a pilot study and while blood pressure and diabetes were accounted for, fasting cholesterol readings were not obtained, a factor that can affect AIx. However, unlike neurovascular disease, trigeminal neuralgia is not known to be affected by cholesterol, and it is unlikely therefore that this would affect the results.

Although arterial tortuosity is thought to be a common cause of trigeminal neuralgia, there are many other possible causes, including vascular compression caused by arteriovenous malformations, aneurysms, neoplasm, infection, or idiopathy. It is possible that in our study the neuralgia was not related to specific vascular abnormality. We have however shown that there does not seem to be a generalised increase in arterial stiffness with trigeminal neuralgia as is the case with intracranial aneurysms.

Kerr *et al* proposed the theory of a double crush mechanism, with the contribution of the internal carotid artery pulse against the lamina that separates the artery from the Gasserian ganglion. As the right carotid artery lies in a straight line with the ascending aorta and anonymous tract, the carotid artery pulse would be stronger on the right side, and thus resulting in a higher predominance of right sided trigeminal neuralgias.[14] However our study did not find any significant asymmetry in the carotid artery augmentation indices to support this theory.

Studies have shown that various classes of antihypertensive agents have different effects on arterial haemodynamics.[15] Although our study did not identify any significant differences in the pulse pressure profiles, this does not exclude the possibility that vasoactive drugs could be considered for medically refractive trigeminal neuralgia, where vascular disease is evident.

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Competing interests: none declared.

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Received 8 August 2002
Accepted in revised form 20 November 2002

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