

AUSCULTATION OF THE SKULL

Intracranial dural arterio-venous fistula was the eventually proven cause of rapid cognitive decline affecting a 67-year-old lady. The first putative diagnosis was Creutzfeldt-Jakob disease.¹

Perhaps a bruit, having been heard upon skull auscultation, would have provided an earlier clue as to the presence of a vascular lesion?

A companion article in the same issue² is a highly persuasive paean to the clinical examination – its continued, arguably enhanced relevance diagnostically and therapeutically.

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References

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- 2 Elder A, Verghese A. Bedside matters – putting the patient at the centre of teaching and learning. *J R Coll Physicians Edinb* 2015; 45: 186–7. <http://dx.doi.org/10.4997/JRCPE.2015.301>

AUTHOR'S REPLY

I thank Dr Lowenthal for his interest in our paper.¹ The point he raises, namely whether the diagnosis of intracranial dural arteriovenous fistula (dAVF) would have been achieved, or at least considered, earlier by hearing a bruit on skull auscultation, merits response, both specific and general, relating to the importance of clinical signs.

Specifically, since the diagnosis of dAVF was not suspected clinically in our patient prior to neuroimaging, we did not undertake skull auscultation for a cranial bruit, a shortcoming or omission in which we suspect we are not unique amongst clinicians.

To my knowledge, there are no published data, other than the anecdotal,² on the diagnostic value of cranial bruit in dAVF, certainly no reviews (either narrative or systematic). It is not clear to me how easy it may or may not be in practice to distinguish a cranial bruit due to dAVF from a physiological cerebral venous hum, although the latter is admittedly more common in children, and hence unlikely to be an issue in an older patient with cognitive decline.

Hence, no definitive evidence-based comment can be made on the question. It is an empirical observation that although clinical neurology is a discipline replete with signs, the diagnostic value of few of these has been examined in a rigorous manner.³ Nevertheless, some points may be made.

Generally, one might anticipate that dAVF is a rare condition, even among patients with rapidly progressive dementia (a previous literature review identified only around 20 publications, most single case reports⁴). The frequency of a cranial bruit in these patients is unknown, but again might be anticipated to be low. (Charles Warlow and colleagues, describing skull auscultation as ‘not very rewarding’ for detecting arteriovenous malformations, once offered a free copy of their textbook to anyone who with no other clues and only by auscultation diagnosed an arteriovenous malformation in an adult,⁵ a prize which has, to my knowledge, never been claimed.) Thus, even in the absence of good empirical data some inferences may be made.

A low frequency sign is likely to have low sensitivity (the probability that subjects with dAVF will have a bruit), although specificity (the probability that subjects without dAVF will not have a bruit) may be high, meaning that there would be many false negatives. With their aversion to missing diagnoses, clinicians may not be enthused by a sign which has low sensitivity. Moreover, for a low prevalence condition like dAVF we may anticipate that, however sensitive and specific a sign is, the positive predictive value (the probability of dAVF in subjects with a bruit) will be low.

These considerations suggest that, with the current state of our knowledge, a provisional judgement might be made, namely that routinely undertaking skull auscultation for a cranial bruit in patients with cognitive decline should not be advocated as a routine part of clinical examination. Ultimately, however, a pragmatic diagnostic test accuracy study⁶ would be needed to inform clinical policy on this issue.

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