Finger Blood Pressure

To the Editor—I congratulate Gibbs and colleagues¹ for their timely evaluation of the Finapres® to measure accurately mean arterial pressure and for the form in which they chose to present the results. In addition to the bias and accuracy of the measurements, they show the frequency, magnitude, and duration of the discrepancies between the Finapres® and radial artery mean arterial pressure measurements. Had these researchers used regression analysis or calculated the correlation coefficient between the two sets of measurements, they would have reached the opposite conclusions, as have others.² However, the statement, “It is hoped that future improvements in engineering design will lead to greater reliability of FIN measurements” is overly optimistic. The idea of measuring blood pressure accurately and noninvasively and in a finger is very alluring; unfortunately, it has been known for some time that finger blood flow and pulse pressure are regulated by sympathetic vasoconstriction.³ Nijboer and Dorlas,⁴ using finger and ear plethysmograms in anesthetized patients, found that whereas the pulse pressure on the pinna of the ear was minimally affected by sympathetic stimulation (laryngoscopy, surgical stimulation, etc.), pulse pressure in the finger was influenced greatly by such events. Therefore, although the finger could be a good place to measure peripheral vascular response to sympathetic stimulation, it is not a good place to assess the general state of the circulation.

Do we need noninvasive measurement of blood pressure in dangerously sick patients? If we do, the carotid, femoral, or radial artery areas should be used. Technically, it should not be difficult to develop the proper transducer; however, its placement will require great attention to detail, a rare commodity in clinical situations.

REFERENCES


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Inconsistency of Data Linking the Ryanodine Receptor and Malignant Hyperthermia Genes

To the Editor—We read the article by MacKenzie and colleagues¹ with great interest. However, we found the rationale behind the evidence for linkage between the malignant hyperthermia (MH) and ryanodine receptor (RYR) genes tortuous, if not entirely circular.

The original paper by MacLennan et al.² suggesting the RYR gene as a candidate gene for MH produced a lod score of 4.2. This degree of linkage was obtained using in vitro contracture test (IVCT) thresholds for the phenotyping of MH susceptibility similar to those that produced no linkage with the RYR gene in the pedigree described by MacKenzie et al. If the revisions to the threshold values of the IVCT suggested by MacKenzie et al. were applied to the patients in the paper by MacLennan et al., it is difficult to imagine that linkage between the RYR gene and MH could still be demonstrated.

From our experience of testing more than 2,500 patients for MH susceptibility, we believe that no conclusions regarding the linkage of MH and the RYR gene can or should be made from the data presented by MacLennan et al. or MacKenzie et al. This view is based on their use of the combined halothane and caffeine test with its high false positive rate (11% in our unpublished series; see also the editorial by Levitt et al.²) and, in the case of MacKenzie et al., by the use of a 1% halothane test, which lacks the sensitivity of tests using 2% or 3% halothane.

Great care must be taken in selecting those families suitable for genetic studies, and the papers by these Canadian groups highlight the need for the full reporting of IVCT methods and results (i.e., the degree of muscle contraction in response to the stated concentration of caffeine and of halothane) in all studies investigating the genetics of MH. Also, because of the likelihood that MH is a heterogenous disorder,⁴ data from more than one family should not be pooled without good reason.

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