Letters to the Editor

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Limitations in Estimating Critical Closing Pressure by Noninvasive Blood Pressure Measurements

To the Editor:

We read with interest the article by Aaslid et al, describing the dynamic pressure-flow velocities relationships in the human cerebral circulation.1 The authors help to validate estimates of critical closing pressure (CCP) based on regular heartbeat data against long diastole determinations. They also suggest that filtering of the radial blood pressure data using Fourier transform techniques could produce more accurate estimates of CCP. However, they only used the average of four intermediate values out of eight values for the correlation test, which studied estimates derived from Fourier technique filtering and long diastole data. Although the Fourier transform techniques reasonably lessen the pressure waveform distortion in peripheral arteries, these techniques potentially produce a higher value of variance.2,3 We wonder whether the correlation would continue to be as good if the average of all eight values had been used.

The more interesting issue is whether CCP could be assessed with sufficient accuracy by entirely noninvasive arterial blood pressure (ABP) measurements. We believe that inaccuracy in absolute value should be an intrinsic limitation to noninvasive methods. Therefore, assessing the changes induced by hemodynamic challenges could be a more sensible approach than using a single CCP estimate to evaluate cerebral autoregulation. We have attempted to estimate CCP respectively by using finger plethysmography (Portapres, TNO-BMI) and by using tonometry over radial arteries (Colin Model 7000).

Twenty healthy young adults (ages 19 to 25; 11 female) were instructed to breathe 5% CO2 at a normal rate and to hyperventilate at a respiratory rate of 30 breaths per minute, both under continuous transcranial Doppler monitoring and ABP measuring. An analogue signal representing the peak velocity envelope of the middle cerebral artery (CBFV) and the 2 ABP signals were fed via an analog-to-digital converter (BNC2070 and 6020E, National Instruments) at a sampling rate of 200 Hz to an IBM-compatible computer and saved digitally on the hard drive for later analysis. CCP was calculated by performing the same linear regression analysis of the instantaneous ABP and CBFV waveforms as described by Aaslid et al.1 For analysis of hemodynamic responses to 5% CO2 and hyperventilation, average values over a period of a selected respiratory cycle (5 to 8 cardiac cycles) were used in order to avoid the influence of respiration. A nonparametric Wilcoxon signed rank test was used to compare the differences in CCP and mean, systolic, and diastolic ABP between different methods of ABP measurement.

No significant difference was observed for any of these parameters at baseline, during 5% CO2 respiration, and during hyperventilation. Hypercapnia produced significant decreases in CCP (from 10.4±17.0 to 4.0±16.0 mm Hg for plethysmography, and from 15.2±15.4 to 7.1±15.4 mm Hg for tonometry; P=0.001 for both methods). Hyperventilation caused significant increases in CCP (from 8.3±17.6 to 22.2±15.5 mm Hg for plethysmography, and from 8.6±20.6 to 19.3±15.7 mm Hg for tonometry; P<0.001 for both methods). A nonparametric Spearman’s rho correlation test disclosed no correlation between CCP estimates derived from the 2 different ABP measuring methods (r=0.048; P=0.84 at baseline; r=0.125; P=0.60 during 5% CO2 respiration; and r=0.161; P=0.498 during hyperventilation).

The changes in CCP induced by 5% CO2 respiration and hyperventilation showed correlation between both ABP measuring methods (r=0.666; P=0.001 for 5% CO2 respiration; and r=0.887; P<0.001 for hyperventilation).

The poor correlation between CCP values derived from plethysmography and tonometry suggested plethysmographic waveforms did not bear a constant relationship to the tonometric ABP waveforms among individuals. Several factors including site of ABP recording, method of pressure measurement, pulse wave velocity, wave reflection and dispersion contributed to the variable extent of ABP waveform distortion. Thus, the distortion of ABP waveform resulted in diverse or even negative CCP values.4

A previous meta-analysis demonstrated that the systolic ABP recorded by plethysmography may be quite different from systolic ABP recorded more proximally.5 In addition, subtle deviation from standard applications of noninvasive ABP measuring equipment also results in certain inaccuracies. However, calibration against a reliable standard is not always available. The poor correlation between CCP estimates derived from tonometry and plethysmography shown in our data reflected these inaccuracies. Simply correcting all these errors by Fourier transform technique seems inconceivable. Furthermore, the relationship between the peripheral ABP and the central pressure is not constant among individuals.6 Attempts to assess absolute CCP value with sufficient accuracy by entirely noninvasive methods or to establish a “normal range” for each noninvasive mean could be impractical.

In contrast, the changes in CCP extracted from different ABP measurements showed good correlations during hypocapnia and hypercapnia challenges. That is, the cerebrovascular regulation through alternation in CCP could be detected consistently by different noninvasive ABP measurements. Silke and colleagues demonstrated plethysmography accurately tracked the dynamic changes in pressure compared with the direct arterial record.7 Cerebrovascular regulation could be reasonably assessed by the changes in CCP during hemodynamic transience without laborious signal processing procedures and regardless of which noninvasive mean is used.

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Response: Limitations in Estimating Critical Closing Pressure by Noninvasive Blood Pressure Measurements

We appreciate the comments, raising the important issue of accuracy in critical closing pressure (CCP) determinations when these are based on noninvasive arterial blood pressure (ABP) measurements.

Using peripheral measurements of ABP, whether invasive or noninvasive, it would seem prudent to consider the effects of waveform propagation distortion. In the radial artery, the second and higher harmonics may be amplified 2 to 3 times, while the first harmonic amplification is typically less than 1.3. Using unfiltered data, the distortion of the higher harmonics would significantly influence the intercept and the slope of the regression line. Moreover, in contrast to what the letter suggests, first harmonic Fourier filtering reduces the complexity of the calculation of CCP because the need for regression analysis is eliminated. Instead the CCP can be calculated using a simple formula described in our paper. In our experience the variance in the estimates of the 2 approaches are similar. (Note also that full harmonic analysis, like in the fast-Fourier transform, is not required, just addition of the products given by simple multiplication of each of the waveform samples with 1 sine and 1 cosine function.)

We agree that the noninvasive ABP methods described in the letter are inadequate for assessment of absolute CCP. Both the finger photoplethysmographic method of Penaz (Portapress) and radial artery tonometry (Colins) may include significant absolute errors in assessing mean and pulsatile pressures. Therefore, absolute CCP determinations may not be representative, as was concluded from the study reported in the letter. Fourier filtering cannot be expected to correct such errors.

One solution to this problem, as the authors suggest, is to use the noninvasive CCP determination only as relative indices. As such, CCP can be used to determine changes in vascular tone or, possibly, intracranial pressure. This approach might be useful in assessing cerebrovascular reactivity in patients, and also in investigative studies of the mechanism of autoregulation and regulation of cerebral blood flow.

However, the better solution would be to use an accurate method of recording the ABP waveform noninvasively. It is generally acknowledged that the upper arm is the “gold standard” site for noninvasive ABP measurements. So why not use a cuff in this location for recordings of the ABP waveform? Like the Penaz method, we would just need to control the pressure in the cuff so that the artery under it is kept suspended in a semicollapsed state throughout the heart cycle. Unfortunately, photoplethysmography, essentially recording blood volume, is not suitable to generate the feedback necessary for the control of cuff pressure in this location.

A 1981 paper proposed use of the principle of restricted flow instead of blood volume for achieving an accurate ABP waveform recording in the upper arm. An animated illustration of this principle and its implementation is available on the Internet. The pressure in the cuff is controlled by feedback from a Doppler ultrasound probe so that the flow is maintained at a low reference level throughout the heart cycle.

Compared with invasive measurements in the contralateral brachial artery, the method was found to be highly accurate, within a few mm Hg in mean, systolic, and diastolic pressures. We are now using a prototype version of this ABP device, and the initial experience in achieving reproducible CCP values is encouraging.

If an accurate noninvasive recording of the ABP waveform is available, propagation distortion may still influence the estimates. We are presently investigating whether the noninvasive upper arm ABP waveform needs to be Fourier-filtered to remove the propagation distortion. Since the distance from the heart to the middle cerebral artery is about the same as to the axillary artery (where the ultrasound probe is located), one might entertain a hope that the effects of wave-propagation in the 2 systems would at least partially cancel out.

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