Does the size of intracranial aneurysms change with intracranial pressure? Observations based on color “power” transcranial Doppler ultrasound


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Object. The authors sought to determine whether the increased pulsatility of aneurysms, compared with normal intracranial arteries, on color “power” transcranial Doppler (TCD) ultrasound was due to a true change in aneurysm size and whether aneurysm dimensions change with intracranial pressure (ICP).

Methods. The authors studied nine patients who had suffered recent subarachnoid hemorrhages complicated by hydrocephalus requiring intraventricular cerebrospinal fluid drainage, in whom the presence of an aneurysm was confirmed on angiographic examination. Color “power” TCD studies of the intracranial arteries and aneurysm were obtained through the temporal bone window before and after insertion of the ventricular drain and then at different known ICPs.

Of the nine patients studied, four were examined both before and after insertion of a ventricular drain. At high ICPs, aneurysms appeared very “pulsatile” and the maximum cross-sectional area was small, whereas at low ICPs, aneurysms appeared larger and were much less pulsatile. The normal arteries did not change significantly in terms of pulsatility or maximum cross-sectional area at different levels of ICP.

Conclusions. The change in aneurysm size visualized with the aid of color power TCD is likely to be real. Aneurysm dimensions vary with ICP levels; the lesions are larger and less pulsatile at low ICPs and smaller but more pulsatile at high ICPs.

KEY WORDS • transcranial Doppler ultrasound • aneurysm • subarachnoid hemorrhage • intracranial pressure • cerebral perfusion pressure

Color “power” transcranial Doppler (TCD) ultrasound is a recent development in color Doppler imaging that, unlike conventional TCD ultrasound, is thought to be independent of the angle of insonation. Thus, power TCD is more sensitive to any movement and its proponents claim to be able to demonstrate random motion in which the net flow is zero. To test the usefulness of color power TCD, we examined patients who had suffered a recent subarachnoid hemorrhage (SAH) while we were blinded to the results of angiographic studies to determine how many intracranial aneurysms could be identified. In this study, we observed that aneurysms appeared to expand and contract (or “pulsate”) more than the adjacent normal artery with each cardiac cycle and that recently ruptured aneurysms were more “pulsatile” than unruptured ones.

This pulsatility appeared much greater than that observed by neurosurgeons during open craniotomy. We therefore wondered whether our observation was the result of an artifact caused by turbulent flow within the aneurysm. Although power TCD is supposed to be independent of the direction of flow, we were unable to find any objective evidence to confirm this. It is impossible to see the aneurysm wall in most cases in B-mode ultrasound without color, so one cannot see the true aneurysm dimensions, only the outline, in color TCD mode. Alternatively, aneurysms might really vary in size inside the intact cranium depending on the transmural pressure and therefore the intracranial pressure (ICP). There was virtually no other information in the literature on the behavior of aneurysms inside a closed cranium: for the obvious reason that it is very difficult, even with sophisticated imaging techniques, to display intracranial vascular structures rapidly enough to detect changes during the cardiac cycle.

One small study in which cine magnetic resonance (MR) angiography was used showed a change in aneurysm size between systole and diastole, but MR angiography is used to visualize the velocity of blood moving within a vascular structure, not necessarily the true size of the vascular structure and therefore is prone to the same criticism as TCD ultrasound.

At high ICP levels, the velocity waveform from the basal intracranial arteries (for example, the middle cere-
The difference in pulsatility as measured by power Doppler TCD is different from that at normal ICP. The diastolic velocity falls and the pulsatility increases at high ICP, and therefore if the color power TCD appearance was the result of a velocity-related artifact, the appearance of normal arteries on power Doppler studies, as well as that of the aneurysms, should change with ICP. The purpose of this study was to determine whether the aneurysm pulsatility observed with color power TCD was likely to reflect true aneurysm dimensions (and their change during the cardiac cycle), not just an artifact of the imaging technique.

Clinical Material and Methods

We identified patients who had been admitted to the hospital with recent SAH that was likely caused by a ruptured intracranial aneurysm and secondary hydrocephalus, observed on computerized tomography (CT) brain scanning, that was likely to require a ventricular drain. When the drain was inserted into a lateral ventricle, it was connected to a simple sterile plastic manometer so that the actual ICP could be measured.

We obtained power TCD studies through the temporal bone windows by using a Doppler device with a 2-MHz probe (model 128 XP10v; Acuson Corp., Mountain View, CA). The whole circle of Willis and the MCAs as far as the major branchpoints were examined and representative images of any aneurysms detected were recorded onto videotape for later analysis. The scan angle and probe positions were adjusted to obtain optimum images showing the maximum aneurysm dimensions and the adjacent normal arteries. Images were analyzed from the videotape recording using the cine playback function on the ultrasound machine. The maximum and minimum cross-sectional areas of any aneurysms detected and an adjacent normal artery were measured from the frozen ultrasound image by tracing around the aneurysm or artery (Fig. 1) using the trackball function on the ultrasound machine. In an attempt to keep the observer unaware of the ICP level, the images were analyzed from videotape by the following method: one operator presented the other operator with sections of tape to measure without saying what the ICP was.

We ensured that all gain and other settings on the TCD machine were kept constant between serial examinations (it is possible to activate the numerical scale describing the gain setting by using the engineering functions of the ultrasound machine). This ensured that any change in the appearance of arteries or aneurysms over time was not simply caused by technical factors. Other scan settings include high persistence (4), maximum power output (0 dB), log compression (52 dB), preprocessing (0), and postprocessing (0).

The patients’ blood pressure (monitored intraarterially if an intraarterial line had been inserted or by mercury sphygmomanometer if not) and ICP were noted at the time of the TCD examination, along with O₂ saturation and end-tidal CO₂, if known. The site of the aneurysm identified on TCD was confirmed on intraarterial angiograms obtained when the patient was well enough. The effect of altering the gain setting on the appearance of the normal arteries was assessed by recording power TCD images at different gain settings in one patient while all other parameters remained constant.

The pulsatility, or percentage change in cross-sectional area between systole and diastole, was calculated by using the following formula: maximum (systolic) − minimum (end diastolic) area/(maximum + minimum area/2) × 100%. The effect of ICP on the calculated pulsatility and maximum and minimum dimensions of the aneurysm and normal artery were analyzed by using means and confidence intervals (CIs).

In a previous study, we had tested the interobserver reproducibility of the measurements of aneurysms and normal arteries. The difference in pulsatility as measured by two observers was 6% (95% CI 0–12%) for aneurysms, and 3% (95% CI 7.5–13%) for normal arteries. Thus, we expect a reproducibility of within approximately 10%, at worst, for these measurements.

This study was approved by the local Ethics of Medical Research Committee.

Results

We obtained power TCD recordings in conjunction with ICP readings from nine patients; in four we obtained
recordings before and after insertion of a ventricular drain to treat acute hydrocephalus complicating SAH. The arterial blood pressure did not change more than 2 or 3 mm Hg between the TCD examinations, except in one patient (Fig. 2), and therefore the effect of ICP alone was examined rather than the effect of cerebral perfusion pressure.

Seven patients had solitary aneurysms (two had posterior communicating artery [PCoA] aneurysms, two had anterior communicating artery [ACoA] aneurysms, one had a basilar tip [BA] aneurysm, and two had MCA aneurysms). One patient harbored four aneurysms (one on each MCA, an ACoA, and a PCoA lesion) and two patients were never well enough to undergo angiography, but the TCD studies showed a convincing aneurysm on the ACoA in one and the PCoA in the other, and on CT scanning the subarachnoid blood was found to be concentrated around these sites. In four patients it was possible to obtain images before and after insertion of the ventricular drain, but images were obtained only after drain insertion in five.

Aneurysm pulsatility was greatest at high levels of ICP (Fig. 3, left). There was a highly significant correlation between ICP and aneurysm pulsatility (Spearman’s rank correlation coefficient 0.854, 95% CI 0.66–0.94; y = 7.04 + 1.5 x, 95% CI of the slope 0.9–2). In contrast, although the correlation between normal artery pulsatility and ICP showed a borderline significance (Spearman’s correlation coefficient 0.7, 95% CI 0.3–0.89), the slope of the line was much less steep (y = 4.4 + 0.28 x, 95% CI of the slope 0.08–0.5), indicating that normal artery pulsatility changed far less over the same range of ICPs than did aneurysm pulsatility (Fig. 3 right). Altering the gain setting on the color power TCD image did not change the apparent dimensions or pulsatility of the normal arteries significantly.

Fig. 2. a–d: Color power TCD images (reproduced in black and white) oriented similarly to Fig. 1, obtained in a patient with an ACoA aneurysm (small arrows and dotted outline). The proximal MCA is marked with arrowheads. Systolic (a) and diastolic (b) measurements (ICP 100, mean blood pressure 180 mm Hg) were taken before drain insertion, and systolic (c) and diastolic (d) measurements (ICP 17, mean blood pressure 76 mm Hg) were taken after drain insertion for high ICP complicating SAH. Note that the change in aneurysm size and pulsatility pre- and postventricular drainage is less dramatic than in Fig. 1, but the explanation may be that the blood pressure was very high before drainage, resulting in a relatively smaller change in cerebral perfusion pressure pre- and postdrainage. e: Axial CT brain scan showing SAH with a focal hematoma (arrow) in the frontal region consisting of bleeding from an ACoA aneurysm. The patient was never well enough to undergo angiography and no autopsy was performed.

Fig. 3. Scatterplots showing the relationship between ICP and the percentage of pulsatility of (left) aneurysms and (right) normal arteries. The three circles to the right of the interruption on the ICP axis designate patients in whom ICP was estimated before the insertion of the ventricular drain, when the exact value was not known but was very high. These points were placed arbitrarily on 60 cm CSF (cerebrospinal fluid). The exact ICP was known in one patient (plus to right of interruption) who was monitored by means of a Camino monitoring device before drain insertion. Circles to the left of the interruption designate the same three patients after insertion of the drain, and pluses designate patients with ventricular drains. Pulsatility was measured as the percentage change between systolic and diastolic ICP.
Effect of ICP on aneurysms

In the four patients in whom we obtained images before and after insertion of the ventricular drain, the aneurysm pulsatility fell significantly, from a mean of 135.5% before drain insertion when ICP was very high to a mean of 24% after drain insertion when ICP was near normal (mean difference 112%, 95% CI 48–176%, Fig. 4 left). (The exact value was unknown predrainage except in one patient with a frontal monitor [model 420; Camino, San Diego, CA]). The normal artery pulsatility fell slightly but not significantly, from approximately 30% before to approximately 11% after drain insertion (mean difference 19%, 95% CI 5.8–44, Fig. 4 left). The reduced aneurysm pulsatility at normal ICPs was a consequence of a significant increase in the maximum and minimum aneurysm cross-sectional area after drain insertion, with a relatively larger increase in the minimum area (mean difference 14.5%, 95% CI 8.6–20.4%, Fig. 4 right). The normal artery maximum and minimum areas fell slightly but not significantly after drain insertion, the minimum area by relatively less than the maximum (mean difference 13%, 95% CI 38–64%, Fig. 4 right). Representative images are shown in Figs. 1 and 2. It can be seen that after the drain insertion, the size of the aneurysm increased and there was relatively less change between the maximum and minimum cross-sectional areas during the cardiac cycle. In other words, as the ICP diminished, the aneurysm expanded and became less pulsatile.

Discussion

We have demonstrated that the power TCD image is likely to display the true aneurysm dimensions because if it were an artifact of flow, we would have expected to see substantial differences in the appearance of the normal arteries between high and low ICPs, and that was not the case. Therefore, given that power TCD is likely to be demonstrating true aneurysm dimensions, we have shown that aneurysms pulsate (quite markedly at high ICPs) and vary in size and pulsatility with ICP. At low ICPs after ventricular drain insertion (or during craniotony), aneurysms appear to increase in size (that is, stretch) and pulsate less, presumably because they have reached their limit and cannot stretch any more. The analogy is with an overstretched rubber balloon: if it is blown up too much it loses its elastic properties and becomes stiff shortly before it bursts.

This observation may explain the following unresolved matters: 1) why one of the peak rebleeding times occurs immediately after insertion of a ventricular drain; 2) why aneurysms do not appear particularly pulsatile at craniotomy; 3) why lowering the blood pressure at the time of craniotomy (pharmacologically or with a proximal arterial clip) may help to reduce the rate of rebleeding during surgery; 4) why aneurysms often appear larger at the time of craniotomy than noted on angiographic studies, although this is also partly because of the aneurysm wall thickness, which is not seen on angiographic examination and may be significant; 5) possibly why in some patients with definite aneurysmal SAH angiograms are negative if they are obtained soon after the rupture, but if repeated approximately 1 week later may show the aneurysm (raised ICP, which may have reduced filling of the aneurysm with contrast material as seen on an angiogram performed within the first few days of SAH, is likely to have lowered with time or to have been drained, allowing better filling of the lesion with contrast material and larger aneurysm size); and 6) possibly why patients with poor-grade aneurysms and high ICP are prone to rebleeding early after the initial SAH (they more often have ventricular drainage in our experience, and ICP that fluctuates and is difficult to control).

There may also be important implications for researchers attempting to evaluate the accuracy of MR angiography, dynamic CT, or color TCD studies in the identification of aneurysms. If the study population consists of patients with recent SAH, part of the failure to visualize aneurysms may be because high ICP in the acute stage causes the aneurysm to be smaller and therefore more difficult to see and more pulsatile and therefore visible mainly in systole. Thus, the results of such studies cannot be extrapolated easily to the population of asymptomatic patients who should be screened for aneurysms, for example those with a family history of SAH. These noninvasive techniques might actually be more accurate when ICP is normal (or they might perform less well for other reasons), and they certainly should be tested in the population to be screened before being more widely used.

In a previous study we found that recently ruptured aneurysms were more pulsatile than asymptomatic lesions, but we did not control for ICP. However, when we reexamined the data for seven patients with multiple aneurysms (thus controlling for ICP in each patient), the
recently ruptured aneurysms still appeared to be more pulsatile than the asymptomatic ones: the mean pulsatility was 74% for ruptured and 29% for unruptured aneurysms (mean difference 45%, 95% CI 3.05–86%). Obviously this observation, which was based on small numbers, requires validation in a larger series, and we cannot tell whether the increase in pulsatility predated or was a consequence of the recent rupture. However, color power TCD is opening up new ways of visualizing intracranial vascular dynamics that may in the future, for example, help predict the likelihood of a particular aneurysm bursting. It deserves further study.

Acknowledgment

The Medical Research Council of the United Kingdom purchased the power TCD machine with which this research was performed.

References


Dr. Wardlaw and Mr. Cannon received funding for this study from the Medical Research Council of the United Kingdom as part of the Clinical Research Initiative in the Neurosciences.

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