AJNR

The risks of vertebral angiography in vertebrobasilar disease.

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AJNR Am J Neuroradiol 1992, 13 (1) 415-416
http://www.ajnr.org/content/13/1/415.citation

LETTERS

Imaging Cerebral Blood Flow in Interventional Neuroradiology

The critical review (1) of the study of altered cerebral vasoreactivity after embolization of arteriovenous malformations (2) published in the May/June 1991 issue of the AJNR is greatly appreciated by the authors, but there are many who feel some of Dr Purdy's criticisms were overstated. If we read Dr Purdy's commentary correctly, his criticisms fall into three categories: 1) statistical significance of the study, 2) legitimacy of xenon/CT-derived flow values, and 3) excessive radiation dose.

We agree with the first criticism. Only eight patients were studied but we observed interesting trends we felt worth reporting because of their clinical implications. However, his criticism of the legitimacy of xenon-derived flow values is unfounded. There is no disagreement that inhalation of 30% xenon does increase cerebral blood flow. Obrist et al (3) and Dettmers et al (4), both using the ^{133}Xenon IV injection technique in humans, have documented flow increases of 13%–28% following xenon inhalation. Using transcranial doppler, Giller et al (5) studied a group of normal volunteers who inhaled 30% xenon. They found, that after 2 minutes of xenon inhalation, a mean velocity increase of 38 ± 3.6% (SEM) with a range of 14%–69%. Furthermore, two other separate studies confirm the work of Giller and colleagues (6). Good and Gur (7) created a computer simulation to study the impact of these flow increases. They assumed a 1.5-minute delay in flow activation, linear-flow activation curve, and "noise free" enhancement. They studied wash-in, washout, and combined wash-in/washout protocols. The wash-in protocol, as used in the xenon/CT methodology, developed at the University of Pittsburgh, revealed small percent errors (4.5% or less) due to flow activation. Purdy's colleague, Dr Lindstrom, reported similar findings in a separate computer simulation study (8). The discrepancy between the flow activation studies of Obrist (3) and Dettmers (4) and the effect of flow activation on xenon CT accuracy determined by the computer simulation studies of Good and Gur (7) and Lindstrom (8) is not due to incorrect assumptions made by the computer simulations, as Purdy suggests, but rather is due to the fairly constant finding of *delayed* flow activation. The first 2–3 enhanced scans are obtained *before* any significant flow activation occurs. The xenon uptake curve for any brain tissue, regardless of its pathologic state, has already been described primarily by these early data points. The last 2–3 data points, acquired after flow activation, do not influence the uptake curve as heavily as the initial data points. Stringer's discussion (9) notes that longer inhalation times, as used in Obrist's (3) and Dettmer's (4) work, will lead to more significant errors due to flow activation. This has been confirmed by the computer model of Good and Gur (7) of a washout protocol. Granted, less is known about the potential of flow effects of 30% xenon on injured brain tissue but we believe these data should lay to rest the concerns that flow activation significantly influences computed CBF values in normal brain tissue when a short inhalation time is used.

Another concern was the variability of flows in a region of interest (ROI). Dr Purdy rightly notes (1) that an ROI usually includes a mix of gray and white matter and any CSF in an adjacent sulcus. One cannot be assured of sampling anatomically pure regions of cortical gray matter, although pure samples of deep white matter may be possible. But this problem is not unique to xenon/CT, and it will also be a problem with MR perfusion imaging. However, both of these techniques, because of their anatomic basis, have a distinct advantage over ^{133}Xenon technology, SPECT (if it ever becomes quantifiable), and PET in being able to better characterize what tissue lies within the ROI. In spite of the inherent noise in our system, ROIs of 1 cm^2 or larger yield an acceptable relative error of 12% (10). There is, however, an exponential rise in relative error approaching 100% as the ROI size approaches pixel size, which precludes using smaller ROIs to sample more homogeneous gray matter.

Xenon-induced euphoria or dysphoria may induce motion of the head which we minimize by the use of velcro body wraps and form-fitting head holders with head and gas mask straps. Even so, there must be an objective measurement of the presence or absence of motion, in order to declare a study acceptable. On the GE system, the computer fits a curve to the 3–6 data points in each pixel. The measurement of the error in the curve fit can be depicted graphically as an "error" map. The lower the error, the darker the shading in each pixel. From this error map, we know when motion degrades a particular study. We report only those studies where there is little or no motion. Furthermore, end-expiratory xenon concentration, monitored throughout the study, is graphically depicted as a rising exponential curve and a constant or intermittent leak will be reflected as a low maximal xenon saturation or a fall-off after the normal exponential rise. We are aware if a gas leak has occurred. If our objective measurement of motion and gas delivery indicates no motion and no gas leak, then we feel those potential variables are optimized and the CBF values should be reliable. We manage to obtain good double studies, obtained at 20-minute intervals, in 80% of awake outpatients, and the rate rises to 90% when single studies are obtained. Furthermore, in the sickest group of patients, those that are comatose, intubated, and paralyzed, 100% yield excellent studies.

We are quite confident of the ability of xenon CT to accurately quantify cerebral blood flow, and there is agreement with PET literature upon the critical minimum of flow for tissue viability (11). A correlative study of ^{133}Xenon and stable xenon by Yonas et al. (12) in 17 normal volunteers shows strong agreement between the two techniques, except for a slight but statistically significant elevation of all

flows measured by stable xenon over ^{133}Xenon of about 10%. This is reflective of the slight flow activation we know will be observed by the end of the wash-in phase.

As Dr Purdy indicates (1) it is much easier to execute a SPECT study than a xenon/CT study, particularly during temporary carotid occlusion testing. For this reason, we are currently undergoing a study to correlate results between these two methods. Perhaps it is not necessary to know whether regional flows drop to 30 or to 15 mL/100 g·min. Perhaps it is also not necessary to know when there are symmetric flow decreases to 25 mL/100 g·min. Currently, our neurosurgeons feel that quantitative information impacts clamp time and the decision to bypass. Furthermore, we want to assure Dr Purdy that the carotid occlusion balloon is inflated only during 10 minutes of clinical testing on the angiogram table and during the 4–5 minutes of xenon testing. During patient transport from the angiogram table to the CT scanner, the balloon is deflated.

Not every clinical indication for CBF evaluation requires quantification. The semi-quantitative nature of SPECT is indeed useful, but we doubt its usefulness in cross-validating xenon/CT. We think xenon CT can stand the scrutiny of cross-correlation with quantitative data from ^{133}Xenon and PET.

Finally, we wish to address the concerns over radiation dose. A xenon/CT CBF study, depending upon the number of scans and exposure factors, will deliver 8–28 cGy (rad) to each level studied. The dose to the lens of the eyes should not exceed 0.25 cGy (0.25 rad) including the exposure due to the scout view (10). Radiation to lower structures is lower than to the lens. Best estimate of whole body dose is 0.14 rad (P. Faturos and N. Perdikaris, unpublished data). According to Swanson (13) and information from the drug circulars supplied with Spectamine and ceretec, we estimate the total dose to the lens using the dual isotope technique to be anywhere from 1 to 8 rad, brain about 1 rad, thyroid 2–3 rad, lungs 2 rad, liver 2 rad, urinary bladder wall 2–3 rad, testes 0.1 rad, ovaries 0.5 rad, and whole body 0.5 rad. Assuming a quality factor of 1, all units convert directly to rems. While the brain receives a larger dose with xenon CT imaging, it still is very radiation-resistant and doses to the lens and other radiation sensitive organs is lower than with dual isotope imaging.

We agree with our colleague that the acceptance of the benefits of CBF imaging (in the appropriate clinical settings, of course) is more important than the debate over the various technologies. There is, however, an irresistible push to accurately quantify flow. PET does it, ^{133}Xenon has done it for years, and so does xenon CT.

Acknowledgments

The authors gratefully acknowledge the contributions of Drs Walter Obrist, David Gur, Walter Good, and Warren Stringer in the preparation and proofreading of this article.

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Reply

I wish to thank Drs Johnson, et al, for their letter in reply to my commentary regarding xenon CT. Clearly, reasonable people have differences of opinion regarding the numerical reliability of flow values that are generated

for regions of interest using xenon CT scanning. In my article, I stated the case for caution in interpreting and applying statistical tests to the numbers that are generated in the performance of xenon CT scans. I still have serious reservations about the legitimacy of using correlational statistics to validate a test which is asserted to be "quantitative". The images with xenon CT should be compared against other qualitative images and a decision as to which technology to utilize should be made on the basis of a broad set of parameters, including the ease of using the technology, side effects, and radiation dosimetry. All of these were discussed in my commentary and need no further elaboration. I agree with my colleagues that this is an interesting area about which there is disagreement. I believe the specifics of my thoughts regarding xenon CT were made clear in the article and I thank Johnson et al for their considerate review of same.

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The Ventriculofugal Arteries

In two recent articles (1, 2) considerable attention is given to my publications on the periventricular blood supply. I believe some comments on these papers are needed.

In the article by Nelson et al, (1) a remark must be made concerning Figure 1, taken from a publication by Yasargil (3). Yasargil gives a schematic representation of the cerebral circulation, based on a drawing in one of my papers in collaboration with H. Vander Eecken (4), cited in his references. Unfortunately, an essential detail in this drawing has been overlooked by Yasargil or by his artist, and this error has been copied by Nelson et al. Indeed, in Figure 1B, some of the centripetal arteries, originating from the basal part of the pericerebral vascular ring, have to reach the ventricular wall, there giving rise to the periventricular vascular ring and to the centrifugal arteries. This important and essential detail can clearly be seen in the schematic representation in my papers (4, 5). In the figure in the *AJNR*, all radial vessels appear interrupted. This makes it difficult to understand where the centrifugal arteries come from.

In the article by Mayer and Kier (2), an essential error has been made in reproducing a figure from one of my publications (6). In Figure 1A, their legend reads: "One of the original *photographs* of an *ink* injected specimen, on which concept of ventriculofugal arteries was based." In reality, this is a *radiograph* of a thick (10-mm) slice after intraarterial injection of *barium suspension*. This is clearly mentioned in my original publication, Figure 8 (6).

Personally, I remain convinced of the existence of ventriculofugal arteries. I do, however, agree that the importance of these arteries has been overstated by later authors. In my own paper of 1969, I have only written about "centrifugal *elements* in the vascular pattern of the deep intracerebral blood supply" (5). Later, it became clear that the ventriculofugal arteries are quite numerous but variable. The existence of the centrifugal or ventriculofugal arteries is proved by radiographs of thick brain slices after intraarterial injection of contrast agents that do not pass through the capillaries (Barium and Schlesinger suspension). In Figure 11 of my paper in collaboration with Vander Eecken (4) it can be seen without any doubt that there are ventriculofugal arteries, which, at the level of the ventricle, branch away from ventriculopetal striate arteries.

The ventriculofugal arteries at the trigonum ventriculi are another issue. In contrast to what is suggested by other authors in later publications, I have never stated that these arteries leave the choroid plexus to run through the ventricle and reach the periventricular white matter after perforating the ependyma. This is, of course, impossible. In my articles (4–8), I state that these ventriculofugal arteries originate from subependymal branches of the choroidal arteries (not of choroid plexus arteries!) which reach the periventricular area from the medial side, by the way of the fissura telodiencephalica.

The issue of the periventricular blood supply is not solved yet, but the so-called refutation of the ventriculofugal arteries is based on wrong interpretations and cannot be maintained. These arteries do exist, but their number and their importance may be variable. Moreover, their visualization is difficult and inconstant.

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Reply

We were delighted to hear from Professor Van den Bergh and appreciate the opportunity to clarify our position on ventriculofugal arteries.

We find difficulty with some of Professor Van den Bergh's interpretation of injected brain specimens for the following reasons:

1. He assumes but does not prove his injection of barium does not pass through capillaries, and, therefore, assumes all vessels in his specimens are arteries.

2. No histologic confirmation was provided to prove his contention that ventriculofugal vessels are either arteries or arterioles.

3. He assumes his injection completely fills the arterial bed to the capillary level. To us, his "terminal" vessels appear to be incompletely filled.

4. His method of studying his specimens with two-dimensional photographs and microradiographs did not enable him to separate overlapping vessels.

We found no striatal arteries that coursed superior and dorsolateral to the caudate to end in the "periventricular" white matter in our 60 specimens. However, with our arterial injection of Microfil, we were able to reproduce the appearance of Professor Van den Bergh's specimens, but when examined with the stereomicroscope, we found single to multiple overlapping vessels. The spray of vessels at the corner of the lateral ventricle were continuous with the subependymal veins, and, when histologically examined, were veins.

The models of cerebral circulation were taken from Yasargil (1). These simplified models were used to show the difference between the "end arterial" type of cerebral circulation proposed by Cohnheim (2), and the ventriculopetal-ventriculofugal vascular borderzone in the periventricular white matter proposed by Van den Bergh (3) and DeReuck (4). The point of the model was not the origin of the ventriculofugal arteries. However, Professor Van den Bergh is correct that his original model had several solid lines connecting the outer ring with the inner ring.

The three types of ventriculofugal arteries were the invention of DeReuck, not Professor Van den Bergh. Our Figure 2 was taken from DeReuck's paper (4) in which he states

> "These ventriculofugal branches penetrate into the brain substance from the choroid plexus and at a distance of 3–10 mm, from the ventricular walls, meet the ventriculopetal medullary or perforating branches, thereby forming a border zone that we shall call type II.'

DeReuck's text and illustration clearly indicate the origin of these vessels to be the choroid plexus, not the leptomeningeal choroidal arteries. This illustration, unfortunately, has been repeatedly reproduced in many books and journals.

We remain convinced ventriculofugal arteries are an artifact of the method of studying the cerebral vasculature, brought on by the inability to resolve superimposed and small crossing vessels, and the failure to properly identify histologically, arteries and veins.

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Reply

We wish to apologize for our incorrect labeling of Figure 1A in Dr Van den Bergh's publication (1) as an ink-injected photograph. In the legend that accompanies the figure, there is no indication of the technique used except the letters "RX" which we did not recognize to mean "radiograph." Furthermore, there is no indication of the injectate.

We would like to comment briefly on the other points mentioned by Dr Van den Bergh in his letter. Throughout Dr Van den Bergh's publications, reference is made to two regions of ventriculofugal arteries: at the pars centralis ventriculi lateralis and at the level of the trigonum ventriculi. In the former type, the ventriculofugal arteries are apparently branches of lateral lenticulostriate arteries that irrigate the striatal regions. In the latter type, the ventriculofugal arteries are apparently branches of choroidal arteries. As pointed out by Dr Van den Bergh, the latter type of ventriculofugal arteries have been misrepresented in diagrams of later authors as leaving the choroid plexus, penetrating the ependyma, and entering the brain substance. We believe that the reason for this confusion is that, although Dr Van den Bergh does state that these ventriculofugal arteries are subependymal branches of choroidal arteries and drew them as such in some diagrams (eg, Fig. 12 in his 1968 article (2) and Fig. 6 in his 1969 article (3)), in other of his diagrams these arteries appear to show the anatomy that was incorrectly redrawn by subsequent authors (eg, Fig. 9 in his 1961 article (1), Fig. 11 in his 1968 article (2), and Fig. 3 in his 1969 article (3)).

As for Dr Van den Bergh's conviction regarding the existence of ventriculofugal arteries, we would reiterate that many authors have noted that branches of medullary arterioles do curve back toward the cortical surface, conforming to a "ventriculofugal" shape, as discussed in our review (4). These branches appear to occur throughout the cerebral white matter, not just in the subependymal region, and do not appear to form vascular border zones with other arterioles. However, the important point of several papers presented in recent years (4–7) is that the concept of a *ventriculopetal/ventriculofugal vascular border zone* is, at best, equivocal. This concept, popularized greatly by other authors, was advanced by Dr Van den Bergh in a series of

papers in which he clearly discussed the role of ventriculofugal *arteries* in the formation of the border zone, and suggested that the border zone model might be the basis of certain types of cerebral infarction and hemorrhage (1–3). *The existence of "ventriculofugal" arteries is certain, though the significance of this ubiquitous morphology of intraparenchymal cerebral arterioles is speculative.*

We would like to make two more points regarding the understanding of the cerebral angioarchitecture. As the brain develops in early embryogenesis, the choroidal arteries are at first located on the surface of the brain (8). Later in development, the telencephalon expands massively. This expansion results in a change in cerebral morphology best described as a "folding" of the cerebrum in various areas. This folding forms the insula, basal arterial perforation zones, and choroidal fissures (9, 10). Thus, the choroidal arteries are no different than any other arteries of the subarachnoid space: their initial segments are extraaxial and their branches penetrate the brain to supply portions of it (hippocampal formation, amygdala, diencephalon, internal capsule, midbrain, choroid plexus, etc). Because the choroidal arteries are no different than those arteries that penetrate the brain from regions more conventionally thought of an "the surface," we would not expect that the intraparenchymal branches of the choroidal arteries would form a vascular border zone with intraparenchymal branches of any other arteries.

Similarly, the early embryonic striate arteries are represented by small vessels penetrating the surface of the brain in the region of the striatal primordium. As the brain develops, the striatal region thickens and the striate arteries enlarge. As the telencephalon expands over the striatum and diencephalon, the striate arteries are gathered together at the base of the brain. This region becomes the anterior basal perforating zone or anterior perforated substance. The thickness and muscularity of the striate arteries as compared to other penetrating vessels is a reflection of the extent and vascular requirements of their territory of supply. Thus, striate arteries are also no different than any other penetrating cerebral arterioles, except that they are grouped together and are relatively large. Again, we would not expect that the striate arteries would form a vascular border zone with intraparenchymal branches of any other arteries.

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The Risks of Vertebral Angiography in Vertebrobasilar Disease

I enjoyed reading the myths, rules, and apposite aphorisms of angiography in patients with occlusive cerebrovascular disease by Drs Caplan and Wolpert (1).

My paper (2) and a paper by Faught (3) are quoted to show that there are no data to support the inference that vertebral angiography is more risky than carotid angiography in vertebrobasilar disease.

Faught had one complication in 21 patients with vertebrobasilar disease, and 15 complications in 91 patients with carotid disease (3). When one remembers that the complication rate for patients with cerebrovascular disease and subarachnoid hemorrhage in 2316 cases reported by Mani was 1.7% (4), the overall complication rate of 14% described by Faught is exceedingly high (3). Furthermore, one cannot draw conclusions of any statistical significance with such a small number of 21 cases of vertebrobasilar disease.

My overall complication rate in 1520 patients was 2.2%. There were 0.9% transient minor complications lasting less than 20 minutes, 1.1% transient major ones, and 0.2% permanent ones. There were no deaths. Fifteen of the 20 transient major or permanent complications were in the posterior circulation. Seven (4%) of 167 patients presenting with vertebrobasilar disease developed postangiographic transient global amnesia, but it only occurred in 1 (0.1%) of 1200 patients without vertebrobasilar disease!

I believe that vertebral angiography in patients with vertebrobasilar disease is more dangerous than carotid angiography for carotid atheroma. I now prefer to study the posterior circulation with a subclavian injection and DSA in vertebrobasilar disease.

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Reply

Dr Pexman may have a point. He found that 7/167 (4%) patients presenting with vertebrobasilar disease developed postangiographic transient global amnesia, but only 1/1200 (0.1%) patients without vertebrobasilar disease developed the complication. An examination of Dr Pexman's patient population and technique is in order to explain his relatively high complication rate. All seven patients had a previous history of transient global amnesia, making it difficult to separate out the underlying symptoms justifying angiography from the complications. Dr Pexman favors using a 6-French Kifa catheter for selective vertebral angiography (approximately half his complications occurred after selective vertebral angiography and half occurred after subclavian flush angiography using 7.3-French catheters). We believe that the large diameter catheter used in selective vertebral studies in Dr Pexman's series may be a significant factor in his complication rate. As described in our paper, we advocate 5-French catheters for vertebral angiography, with immediate withdrawal of the catheter from the artery after contrast injections. Dr Pexman also states that selective vertebral angiography is safer with a 5-French catheter than with a 6-French catheter. Immediate withdrawal of the catheter has a threefold benefit: 1) oxygenated blood is delivered to the posterior circulation immediately following the contrast injection; 2) any temporary vasospasm that may occur following the injection is immediately relieved; and 3) the oxygenated blood provides a bolus effect, maximizing opacification of the vertebrobasilar arteries. We believe that immediate catheter withdrawal after selective vertebral injection is mandatory in atherosclerotic patients and reduces the complication rate. Others have also confirmed that the use of softer, smaller catheters is associated with a decreased rate of neurologic complications (1). The latter authors also found no statistically significant difference in the incidence of complications when they examined the vessel studied; by inference, therefore, complications were not more frequent after vertebral angiography than after carotid angiography. We stand by our original comment about the safety of vertebral angiography, if it is carried out according to our technique.

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Early Trainees in Neuroradiology: An Omission

I am writing in order to correct some important omissions in the list of early trainees that I submitted with the paper on Development of the First Fellowship Training Program in Neuroradiology in North America (*AJNR* 12:587–590, July/August 1991).

These individuals are as follows:

Dr Ray A. Brinker, now Professor and Chairman of the Department of Radiology at the Medical School Ohio State University, Toledo, Ohio

Dr Joseph H. Allen, Professor of Radiology at Vanderbilt University, Nashville, Tennessee

Dr Harvey I. Wilner, Director of Neuroradiology at the Harper Hospital, Detroit, Michigan—Professor of Radiology, Wayne State University Medical School

Dr Carlos E. Parera (a non-citizen trainee without NIH support), now Director of Neuroradiology at the Clinica Puerta de Hierro, Madrid, Spain.

Having separated myself from my records, unfortunately, I may have forgotten other early trainees, and I wish to apologize to them.

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