ANOTHER MEANS OF DIAGNOSING SYNOVIAL CYST DISSECTION

In reference to the article by Pozderac and Good (Arthroscintigraphy in acute synovial rupture of the knee, *J Nucl Med* 15: 7–9, 1974) we would like to call attention to another technique by which the nuclear medicine physician or radiologist may make this difficult differential diagnosis.

Diagnostic ultrasonic B scanning of the calf has been used successfully to distinguish dissection of synovial fluid into the calf from thrombophlebitis, and would appear, on the basis of simplicity and rapidity, to be the method of choice, when available.

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THE AUTHORS' REPLY

We are interested in Dr. Arkless's experience with "posterior leakage" after exercise in knees of normal subjects. One may observe the entry of contrast material posteriorly into the popliteal bursa but extension well into the calf is not normally observed in our experience unless associated with synovial rupture or cyst.

We agree with Dr. Arkless that venography is a useful tool for evaluating patients suspected of having deep-vein thrombophlebitis of the calf. However, acute synovial rupture of the knee (abrupt decrease in the size of a knee involved with synovitis plus signs simulating thrombophlebitis) is an established syndrome in rheumatology that does not require venography for confirmation but may require a procedure to demonstrate the rupture (*1,2*).

We also agree that positive contrast arthrography does have advantages over gas arthrography. Either of these methods for performing arthrography can be used to confirm a diagnosis of synovial rupture. We reported the efficacy and advantages of a newer method (arthroscintigraphy) in establishing this diagnosis.

The authors do not have any experience with ultrasound B scanning; however, we do appreciate that Dr. Winston and Dr. Pritchard have called attention to yet another possible use for this noninvasive diagnostic tool.

We thank Drs. Arkless, Winston, and Pritchard for their comments and interest in the article.

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ANGIOSCINTIGRAPHY VERSUS PORTABLE-PROBE TECHNIQUE AS A ROUTINE DIAGNOSTIC AID FOR CEREBRAL DEATH

We are very interested in a recent case report (*1*) which demonstrated the cerebral passage of an intravenously injected bolus of $^{99m}TcO_4$ by means of sequential gamma camera imaging in a patient in deep drug-induced coma, who subsequently recovered. We have addressed ourselves to this problem (*2,3*) over the last few years using portable-probe equipment and have completed 142 bolus transit studies in 80 deeply comatose patients who met all the recognized clinical criteria for cerebral death. The causes of coma in these patients included transient cardiac arrest, head injury, intracerebral bleeding, and drug intoxication. Since Nordlander, et al have questioned the use of probe equipment for the study of cerebral death we offer the following comments.

For possible routine clinical application in the diagnosis of brain death any ancillary technique must meet certain prerequisites: (A) accuracy, a mistaken diagnosis of brain death is not tolerable; (B) re-

peatability at short intervals in case of doubt on any single test and more fundamentally because cerebral circulatory deficit over a single period of a few seconds is not necessarily incompatible with recovery; (C) freedom from significant risk; and (D) portability for convenient bedside use. Portability is a basic prerequisite from the point of view of safety and widespread application. These are desperately ill patients who may or may not be cerebrally dead and are connected up to multiple life-support and monitoring systems. It is neither practicable nor safe to move patients routinely and repeatedly in such a precarious situation, away from their controlled and supervised location.

The portable-probe technique was developed with these requirements in mind. A control was incorporated for reliability (*3*). Unlike a gamma camera, the probes are readily portable for easy bedside use. Only 2 mCi or less of $^{99m}TcO_4$ are used per study, which can be repeated within minutes if necessary because the background from the previous study can be easily suppressed.

We disagree with the authors' claim that with this probe method "it is impossible to decide whether the detected activity comes from vessels in or outside the brain." We also are aware that some investigators have encountered difficulty in demonstrating the deficit in cerebral circulation associated with brain death using nonimaging types of radioisotopic methods due to the presence of the extracerebral circulation (*4*). These methods involved blood or blood-flow quantitation and did not make use of the dynamic differences between the cerebral and extracerebral circulations. The very rapid, low-reservoir type of cerebral circulation clearly displays the passage of a reasonably coherent nondiffusable radioactive bolus (e.g., $^{99m}TcO_4$) by the familiar time/activity curve (transit curve) which has been used by Oldendorf (*5*) and others to measure cerebral transit time. One would not expect the relatively sluggish (*6*) and dispersed peripheral type of extracerebral circulation to give a comparable time/activity curve. Of our 142 studies, in no case of cerebral death (34 patients) with an adequate radioisotopic bolus study were there any questions or equivocation about the absence of a recognizable transit curve. In fact, so far there has not been any question of possibly mistaking cerebral circulation for extracerebral circulation alone. In contrast, four patients with absent transit curves all showed complete lack of supratentorial entry of radiographic contrast agent on concomitant four-vessel cerebral angiography.

A significant clinical question remaining which we have not been able to answer so far is at what level of cerebral circulatory deficit the transit curve might be so depressed and prolonged as to be confused with the extracerebral curve alone? If there is such a level, is it compatible with cerebral survival? We have found unmistakable evidence of the usual transit curve with cerebral blood flow as low as 24% of normal (*3*), as measured by the Argon technique. This probably approaches the critical levels of brain viability (*7,8*).

It should be pointed out here that since cerebral angioscintigraphy also depends on the cerebral passage of the radioactive bolus, there should also be a critically depressed level of cerebral blood flow below which the gamma camera will not reliably image the passage of the bolus. With decreasing cerebral blood flow the radioactive bolus decreases in peak activity, becomes spread out and is delayed (as in the author's case report). It is therefore to be anticipated that a level of cerebral blood flow may also be reached below which the "noise" from transit of the extracerebral radioactivity will obscure reliable visualization and differentiation of the low level and spread-out activity of the delayed bolus passing through the cerebral circulation.

Several of our studies were performed on drug-intoxicated patients with severely abnormal and depressed EEGs and subsequent recovery. All these cases showed clear evidence of cerebral circulation by a transit curve study. Up to this time we have not had the opportunity to study the type of patient with drug intoxication and prolonged complete electrocortical silence who subsequently recovers. Since these are obviously cases of crucial clinical significance, we would be most interested to know if Norlander, et al have been able to perform cerebral angioscintigraphy on such a patient.

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THE AUTHORS' REPLY

We have read with keen interest the impressive studies of Braunstein, et al (*1,2*) about the problems concerning brain death. We do agree that it would be of great value to have a method for routine clinical use by which it could be possible to obtain objective criteria of impaired cerebral perfusion.

We admit that it is inconvenient to bring the patient from the ICU to the isotope department. These patients, however, are few in our hospital, and the transport has not offered any difficulties and no complications have occurred. These drawbacks, however, have not influenced our choice of method.

We have had a different intention than Braunstein, et al when deciding which type of technique is preferable in these cases. In some European countries there is a tendency to insist upon x-ray angiography as a form of legal proof of the absence of cerebral circulation at brain death. The x-ray examination is rather time consuming and requires a lot of personnel. Therefore we have looked for another method of examination which is easy to perform, not so time consuming, and yet reliable. We believe that we have found such a method in the isotope angiography we have described (*3*).

The important difference between the portable probe technique and our method is that the latter makes it possible to visualize the morphologic changes of impaired cerebral circulation by means of sequential pictures as well as by time–activity curves.

On the scintiphotos from the examination of the circulation we have been able to check that the radionuclide has been properly injected. By this method it is also possible to detect local vascular changes, for instance, arterial embolic lesions with impaired circulation or dislocation of intracranial arteries owing to expanding masses.

The question concerning the distribution of the activity in the external and the internal carotid artery is contradictory. We believe that the time–activity curves obtained by the method used by Braunstein, et al are the sum of the intra- and extracranial activity at maintained cerebral circulation even though the extracranial activity may be low compared with the intracranial. For that reason we feel entitled to state that "it is impossible to decide whether the detected activity comes from the vessels in or outside the brain." Of prime importance, however, is that this holds true in impaired intracerebral circulation, "intermediate type," according to Braunstein, et al (*2*).

The discussion about the discrimination of the depressed and prolonged transit curve and the extracerebral curve is interesting. Braunstein, et al admit that it may be difficult to separate the intracranial from the extracranial activity.

This fundamental problem concerning the level of the intracranial pressure at which the intracranial circulation is totally hindered is relevant for every kind of measuring or visualization of the cerebral circulation including x-ray angiography. Further investigation of this problem is necessary.

The last question deals with angioscintigraphy of patients in deep coma due to drug intoxication with prolonged total abolishment of the EEG with subsequent recovery. We have not examined any such patient and thus we cannot give any answer to the question. The patient in our paper (*3*) had "a highly abnormal EEG, being sometimes flat, almost isoelectrically silent for periods of 10 sec."

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