be ordering an i.v. urogram—again as a screening procedure. I find these suggestions unacceptable and quite impractical. If the dynamic and static nephroscintigram is indicated in hypertension as a screen for unilateral renal disease, then surely we need perform only one procedure in the upright position, preferably sitting. If this demonstrates a unilateral abnormality, further study is indicated.

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REFERENCE


Reply

Concerning Dr. Helliwell's critique of our paper (1) we suggest the following time-saving approach. In hypertension of unknown cause, the last picture of the IVP is obtained while the patient stands. Posture-dependent change in the length of the kidney is registered, as is any caudal shift. The finding of nephroprosis would lead to a function scintigram in upright posture, and our experience suggests that the patient should stand. Unilateral disturbances of Hippuran uptake in the ptotic kidney would indicate a repeat examination in prone position. Posture-dependent renal-artery stenosis would result in a normal renogram in the prone position. The static scintigraphs could be omitted or reserved for those doubtful cases where AP and PA scintiphotographs might help in evaluating the importance of altered geometry and tissue absorption. This sequence would eliminate unneeded examinations.

We do not like the suggested diagnostic short-cut advocated by Dr. Helliwell. It will save time but might lead to ambiguous results, since the described triad of ptosis, hypertension, and posture-dependent second-phase renogram abnormality would not be identified. In attempting to identify persons with orthostatic hypertension amenable to surgical therapy, one should remember the following. a) Not every hypertension seen in patients with nephroprosis is orthostatic. b) Not every nephroprosis leads to orthostatic hypertension. c) We commonly see the described renogram pattern in persons who are normotensive. d) Orthostatic hypertension may exist in the presence of normal function scintigrams. e) The term orthostatic hypertension does not seem to describe a homogenous hypertensive population.

It may interest Dr. Helliwell to know how often we have found the described orthostatic hypertension. We examined approximately 150 persons and found the signal renogram pattern 29 times (19%). Thus, 80% of our ptosis-hypertension population had renogram patterns different from the one previously described. A very different but relatively common renogram pattern should be mentioned in this context. The renogram in the prone position is normal. The standing function scintigram demonstrates a massive, bilateral disturbance of intrarenal Hippuran transport. These persons do not have posture-dependent divergence of single-kidney Hippuran uptake. Hippuran clearance values in the prone position are pathologic. We have never seen this renogram pattern in any person with ptosis who was normotensive.

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REFERENCE


Pontine Glioma—Positive Tc-99m Brain Scan, with Negative TCT and Angiogram

Diagnosis of neurologic disease is becoming increasingly accurate, with decreasing morbidity and mortality as newer neuroradiologic techniques become available. The high cost of these specialized tests has led some to propose a complete, or near-complete, replacement of the radionuclide (emission) brain scan by transmission computerized tomography (TCT) (1). We present here a case of posterior-fossa tumor where the emission brain scan was abnormal from the onset of the clinical study, whereas cerebral angiograms and initial TCT were normal.

A 52-year-old white man presented with a 3-wk history of increasing fatigue, staggering gait with a tendency to fall to his right, subjective numbness on the left side of the face, and inability to focus on moving objects, especially when they moved to the left. Neurologic examination revealed nystagmus and equivocal plantar reflexes. His gait was wide-based without step-pare, but with a tendency to veer to the right. A TCT head scan, with and without contrast enhancement, showed only mild cerebellar atrophy.

On the following day 1-hr delayed static emission radionuclide brain images with Tc-99m DTPA showed abnormal activity located anteriorly in the left posterior fossa (Fig. 1). This prompted additional TCT views of the posterior fossa to be made, with and without contrast enhancement, and these showed evidence of a mass lesion on the left side. For further confirmation, repeat TCT scans (Fig. 2) were performed in another hospital with coronal and axial sections, with and without contrast enhancement. This examination was normal with the exception of possible flattening of the left side of the fourth ventricle. The dense area in the left posterior fossa in the post-contrast study was thought to be an artifact caused by the petrous bone and improper positioning. The subsequent normal left carotid and vertebrobasilar angiograms (Fig. 3) strengthened this belief. Because the cerebral angiogram was normal, and on two occasions...
the TCT was inconclusive for a mass lesion, the clinicians attributed the symptoms and abnormal emission brain scan to a cerebellar infarct. The patient refused a lumbar puncture. He was discharged for outpatient followup.

Three weeks later (Nov. 25) he was readmitted with worsening of symptoms. The emission brain scan on this admission was again abnormal and showed further progression of disease. A TCT scan performed at this time showed a definite left posterior mass lesion, with the additional finding of moderate ventricleomegaly.

At surgery an intrinsic tumor involving the left lateral pontine region was biopsied. Microscopy indicated a highly malignant, undifferentiated neoplasm. The patient received radiation therapy but subsequently died and the autopsy demonstrated a Grade II astrocytoma with moderate necrosis secondary to therapy (Fig. 4).

This case is interesting in that the patient’s diagnosis was delayed because of normal cerebral angiograms and inconclusive TCT. New et al. (2) could not find a case of brain disease with negative TCT, normal cerebral angiograms, and a positive emission brain scan. Strasberg et al. (5), comparing angiograms and TCT, reported that this combination did misdiagnose a neoplasm as an infarct, but both studies were abnormal. Passalaqua et al. (4) presented two cases where the TCT and emission brain scans were falsely negative, whereas angiography detected the lesions. On the contrary, our patient had a markedly abnormal emission brain scan, a normal cerebral angiogram, and two of the three initial TCTs did not indicate a definite mass.

Mikhael and Mattar (5) reported an accuracy of 88% in the detection of tumors in the posterior fossa with emission brain scans, and 92% with TCT. According to Kazner et al. (6) TCT is the most informative diagnostic procedure for posterior-fossa tumors. They quote an accuracy of 96% with contrast enhancement, and 80% without it.

Because of its accuracy, cost effectiveness, and structural detail, we would not argue against the use of TCT as the initial diagnostic tool for brain lesions. Cerebral angiography and pneumoencephalography are invasive techniques, and in many instances they are now unnecessary for evaluation of posterior fossa lesions before surgery (6). On the other hand, emission brain scanning is an innocuous procedure. At times it alone may detect the abnormality, as in our case, and over-reliance on TCT will occasionally delay diagnosis and appropriate therapy. Neither should be considered the final arbiter in ruling out disease, and other diagnostic procedures must be used, especially when a neurologic deficit is present on clinical examination.

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