Computerized tomography, magnetic resonance imaging, and positron emission tomography in the study of brain trauma

Preliminary observations

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Results of computerized tomography (CT), magnetic resonance imaging (MRI), positron emission tomography (PET), xenon-133 measurement of cerebral blood flow (CBF), and neuropsychological assessments are described in three head-injured patients. The patients were selected because they presented with intracranial hemorrhage diagnosed by CT. Two of the patients were studied acutely and again approximately 6 months later. In the acute stage, MRI was superior to CT in identifying the precise location and extent of intracranial hemorrhage and associated edema. Small subdural hematomas diagnosed on MRI were missed with CT scanning. The extent of apparent encephalomalacia in the chronic stages of injury was also better defined with MRI. Position emission tomography showed disturbances of glucose metabolism that extended beyond the structural abnormalities demonstrated by MRI and CT; anterior temporal lobe dysfunction was particularly evident in all three patients. Regional CBF studies failed to detect a number of the abnormalities seen on MRI and CT, and even ignored the metabolic dysfunction evident on PET that should have been accompanied by changes in regional CBF. The neuropsychological studies localized frontal lesions, but did not reveal abnormalities attributable to the structural lesions and the reduced metabolism in the anterior temporal lobes.

KEY WORDS • head injury • computerized tomography • cerebral blood flow • magnetic resonance imaging • positron emission tomography • neuropsychological assessment

The contributions of computerized tomography (CT) to the diagnosis and management of head injury have been enormous. Considering the incidence of head injury, its morbidity and mortality, and the advantages of CT over plain x-ray films and cerebral angiography, one can argue that the greatest impact of CT in neurology and neurosurgery has been in the field of head injury. Magnetic resonance imaging (MRI) and positron emission tomography (PET) are of interest in the study of head injury because they are likely to provide information on the nature and location of intracranial pathology that supplements and may be superior to the information provided by CT. The purpose of the studies described in this report is to begin the process of evaluating the incremental advantages of MRI and PET over CT in the diagnosis of the brain lesions produced by head injury and in assessing the evolution of the lesions over time.

We have developed a new classification system of head injury, and have entered into it over 1100 patients from our neurosurgical unit and from other head-injury centers across the nation. The classification system is based on a combination of the patients’ neurological status (determined mainly by means of the Glasgow Coma Scale (GCS)) and the CT findings. All the patients included in the system were in coma, defined as a GCS score of 8 or less. The classification system requires that the neurosurgeon and neuroradiologist responsible for the case identify the principal lesion; that is, the pathology thought to be the most likely cause of the patient’s coma. Focal injuries (subdural, extradural, and intracerebral hematomas and large con-
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tusions) accounted for 56% of the primary diagnoses in 1107 patients; the remaining patients, those whose CT scans did not demonstrate a mass lesion of sufficient size to account for the patient’s clinical status, were classified as having diffuse injuries.

The CT scan was diagnostic in all cases with focal lesions and was used to define the category of focal lesions. In contrast, it failed to identify the cause of coma in most of the patients with diffuse lesions. Based on clinical observations and studies of a model of head injury in the primate, we believe that the most common lesion responsible for the coma in diffuse injuries is primary mechanical damage to axons, which we have termed “diffuse axonal injury.” In some patients there are tell-tale signs of diffuse axonal injury in the white matter, but in the large majority of patients the CT scan either is normal or demonstrates a small extracerebral hematoma or small contusions that are not sufficient to account for the patient’s coma. In addition to diffuse axonal injury, patients who have suffered a period of systemic hypoxemia or profound shock also present with a diffuse injury syndrome of coma and a CT scan that is not diagnostic.

We postulate three principal challenges to PET and MRI in furthering the diagnosis and management of head-injured patients. The first challenge to MRI is to define better than CT scanning the location and extent of intracranial hemorrhage in the various stages of its evolution. The second challenge to both PET and MRI is to improve on the CT definition of the limits of structural and functional abnormalities in the brain parenchyma associated with hemorrhage and edema. The third challenge is to identify lesions, especially diffuse axonal injury, that are almost entirely missed by CT. In this preliminary report, we address the second of these issues by correlating abnormalities seen with CT, MRI, and PET in three patients with posttraumatic hematomas and contusions. In two of the patients, studies were obtained acutely and again 6 months later. Data are also included on cerebral blood flow (CBF) measured by the xenon-133 (\(^{133}\text{Xe}\)) method and neuropsychological assessments. To our knowledge, these are the first observations with PET to be published in head-injured patients.

**Clinical Material and Methods**

The three patients included in this report were selected for study because they had posttraumatic hemorrhagic lesions of the brain which permitted a test of the diagnostic accuracy of CT, MRI, PET, and CBF (measured by the \(^{133}\text{Xe}\) method) in detecting the extent of the lesions in the acute stage and their evolution over time. The CT scans were performed without contrast enhancement on a General Electric (GE) 9800 scanner. Slices were 10 mm thick and the spatial resolution was 0.75 x 0.75 mm. The entire brain was encompassed from the foramen magnum to the vertex in the axial plane.

The initial MRI scans of Cases 1 and 2 were performed on a GE 0.12-Tesla resistance unit using a 9-in. diameter coil. Saturation recovery images were obtained with a 143-msec repetition time, using averages of four signals, a matrix size of 128 x 128 interpolated to 256 x 256, a slice thickness of 1.3 cm, and multislice data acquisition. The follow-up MRI scans of Cases 1 and 2 and the scan carried out at the acute stage on Case 3 were performed with a GE Signa 1.5-Tesla unit using spin-echo pulse sequences. For T1-weighted imaging, repetition times of 600 to 800 msec and echo times of 20 to 25 msec were used, and for T2-weighted imaging, repetition times were 1500 to 2500 msec and echo times were 40 to 120 msec. Although the quality of the MRI scans obtained with the resistive system was quite good and permitted comparisons with the CT scans, as noted in the individual case reports below, for illustrative purposes they do not reproduce nearly as well as the scans obtained with the high-field system. Therefore, only examples of the latter scans are included in the illustrations.

The PET studies were performed on a positron emission transverse tomograph (PETT V), which provides seven simultaneous slices with 17-mm full width half-maximum resolution. Local cerebral metabolic rates for glucose were measured using the fluorodeoxyglucose technique, as modified by Huang, et al. All three patients were studied with their eyes and ears open to low ambient room conditions and were injected intravenously with from 2.5 to 6.8 mCi of fluorodeoxyglucose. Following a 40-minute period to allow maximum uptake, the patients were positioned and scanned for 10 to 20 minutes; they were then moved one-half the slice thickness and scanned again. This technique resulted in a set of 14 images at 8.8-mm separations with 1.5 to 5.0 million counts per image. Regional metabolic data were derived using a computerized overlay system consisting of mirror-symmetrical regions of interest which were scaled to match the appropriate brain section image. This analysis was compared with the CT scans of each patient, so that any gross anatomical asymmetry could be taken into account. There were no gross asymmetries in the regions of interest of any of the patients reported here, so no adjustments were required. Metabolic rates for glucose were calculated using values for rate constants and lumped constant in a manner that we have reported previously. Visual interpretation of all scans was consistent with the metabolic results.

Regional CBF was measured by the intravenous \(^{133}\text{Xe}\)-clearance method using 16 extracranial detectors, eight placed over each hemisphere. The \(^{133}\text{Xe}\)-clearance curves were subjected to a two-compartment analysis from which several blood flow indices were derived. One of these, “CBF 15,” is illustrated in the present paper. Based on a modified height-over-area method in which the curves are integrated to 15 minutes, CBF 15 represents the mean flow of the fast- and slow-clearing
Experience with head-injured patients\textsuperscript{11} indicates that this index is stable in pathological conditions and sensitive to focal alterations in flow. Normal control data obtained from 42 healthy adults provided a statistical basis for assessing the limits of normality, with respect to both CBF level and regional variation. The CBF levels were evaluated in relation to arterial CO\textsubscript{2} tension.

The battery for neuropsychological assessment included the following tests: Wechsler Adult Intelligence Scale, Russell's Revised Wechsler Memory Scale, Trail Making, Cancellation, Reitan's Modification of the Halstead-Wepman's Aphasia Screening, Bender-Gestalt with immediate recall, Finger Oscillation, Lateral Dominance, Reitan-Klove Sensory-Perceptual Examination, and also (for Case 2 only) the Oral Controlled Word Association Test, Halstead's Neuropsychological Battery, and the Visual-Verbal Test.

Case Reports

Case 1

This 22-year-old man fell off the rear of a truck moving at high speed, striking his head on the pavement. He was admitted to our hospital 3 hours later, at which time he had a GCS score of 14 without any localizing neurological signs. His condition began to deteriorate and he had a GCS score of 10 immediately prior to surgery.

A CT scan showed bifrontal contusions and hematoma formation, greater on the right than the left, a right subdural hematoma, bilateral temporal tip contusions, and subarachnoid hemorrhage (Fig. 1). At surgery, a right frontal craniotomy was performed with evacuation of the subdural and intracerebral hematomas. Postoperatively, the patient had a GCS score of 7, again without lateralizing signs, and for 3 days intracranial pressure was in the 20 to 30 torr range, requiring moderate hyperventilation. A postoperative CT scan demonstrated that nearly all of the hematomas had been removed.

Nine days after admission, the CT scan was repeated and the following day an MRI scan was performed using the 0.12-Tesla system. The volume and general appearance of the damaged frontal lobes were the same with the two imaging modalities. The temporal lobes now appeared to be normal on the CT scan, although some artifacts were present, whereas the MRI scan demonstrated several temporal lobe contusions more numerous and more extensive than any that the three
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TABLE 1

Ratio of regional to whole-brain glucose metabolism values

<table>
<thead>
<tr>
<th>Region of Interest</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8/2/84</td>
<td>1/16/85</td>
<td>8/6/84</td>
</tr>
<tr>
<td></td>
<td>Lt</td>
<td>Rt</td>
<td>Lt</td>
</tr>
<tr>
<td>frontal pole</td>
<td>0.69</td>
<td>0.46</td>
<td>0.62</td>
</tr>
<tr>
<td>orbital cortex</td>
<td>0.81</td>
<td>0.42</td>
<td>1.00</td>
</tr>
<tr>
<td>midfrontal gyrus</td>
<td>0.87</td>
<td>0.69</td>
<td>1.14</td>
</tr>
<tr>
<td>front eye field</td>
<td>1.19</td>
<td>0.94</td>
<td>1.34</td>
</tr>
<tr>
<td>superior temporal region</td>
<td>0.87</td>
<td>0.77</td>
<td>0.73</td>
</tr>
<tr>
<td>primary auditory cortex</td>
<td>1.21</td>
<td>1.14</td>
<td>1.20</td>
</tr>
<tr>
<td>auditory association area</td>
<td>1.09</td>
<td>1.15</td>
<td>1.15</td>
</tr>
<tr>
<td>cerebellum</td>
<td>1.14</td>
<td>1.21</td>
<td>1.17</td>
</tr>
</tbody>
</table>

CT scans had suggested. A smear subdural hematoma was present over much of the left hemisphere, but was not visualized on any of the CT scans.

On the 15th day after injury, the patient underwent the first of two PET scans. Throughout the previous week his neurological status had steadily improved and he had reached a GCS score of 15 at the time of this study. Glucose metabolism was reduced in the anterior portion of the right frontal lobe to less than one-half the value for the whole brain; it was also reduced in the left frontal lobe, but less so (Table 1 and Figs. 2 and 3). Metabolism was also reduced in both anterior temporal lobes, the right more than the left (superior temporal region, Table 1 and Fig. 3). The posterior temporal regions (primary auditory cortex and auditory association area, Table 1) had normal metabolism.

Shortly after the patient's surgery, about 36 hours following admission, a 133Xe CBF study was performed. The patient had a GCS score of 7 at the time. In keeping with his depressed level of consciousness, the mean CBF for both hemispheres was 28.6 ml/100 gm/min compared to a normal value of 50 ml/100 gm/min, and the cerebral metabolic rate for oxygen was 2.15 ml/100 mg/min compared to a normal value of 3.5 ml/100 mg/min. There were focal decreases in CBF in the right frontal and temporal regions. Autoregulation was globally defective: the systemic arterial pressure was raised, and as CBF increased globally, the focal diminutions in flow disappeared. A repeat study at 21 days showed a mean CBF of 48 ml/100 gm/min with a right frontal but not a right temporal focus (Fig. 4).

The first neuropsychological evaluation was performed at 3 weeks after the injury. The patient's full-scale intelligence quotient (IQ) was 77. He demonstrated a substantial reduction in verbal skills and a constructional apraxia consistent with residual right frontal lobe damage.

A final battery of studies was performed in December, 1984, and January, 1985, approximately 6 months following the injury. He had returned to full-time work as a day laborer in a nursery, working for his father. Both his father and a sister considered him to be normal in all respects. A CT scan demonstrated decreased attenuation in both frontal lobes, greater on the right than the left, consistent with encephalomalacia, and at the tips of both temporal lobes (Fig. 1). The findings on the MRI scan were similar, but the boundaries

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between abnormal and ostensibly normal brain were much more distinct. Damaged brain gave a low-intensity signal on both T2 (axial) and T1 (sagittal) images. The volume of damaged brain could be estimated better from comparing the axial and sagittal MRI views than from the CT scans (Fig. 1). The sagittal views showed a confluence of the frontal and temporal encephalomalacia not evident on the axial CT scans and MRI.

A comparison of glucose metabolism values in the frontal lobes between this study and that performed in August, 1984, was particularly interesting (Table 1 and Figs. 2 and 3). Metabolism was reduced throughout the middle and inferior portions of the right frontal lobe even more than on the original PET study, and metabolism was essentially absent at the frontal pole. In contrast, metabolism in the left frontal lobe had improved except for the frontal pole. Metabolism in the right anterior temporal lobe that had been reduced earlier was even lower, and the left anterior temporal lobe had failed to recover.

A 133Xe-clearance CBF study on December 18, 1984, demonstrated a mean flow of approximately 60 ml/100 gm/min. The anterior frontal and anterior temporal probes showed a clear reduction in flow on the right at the sites of residual brain damage (Fig. 4), but there was no correlation with the metabolic abnormalities in the left frontal and temporal lobes. The patient’s full-scale IQ was now 87, and he demonstrated mild residual apraxia and problems with recall although, as noted, he and his family considered that he was normal.

**Case 2**

This 25-year-old male college graduate was interested in pursuing a career in medicine. On July 19, 1984, he fell 20 ft from a ladder; he landed upright, then fell backward striking his occiput on the pavement. He was declared to be unconscious for 20 minutes, and was admitted to our hospital 45 minutes after the injury with a GCS score of 13. The right pupil was slightly larger than the left, but there were no other localizing signs. An admission CT scan demonstrated a right frontal contusion that contained relatively little blood and moderate swelling of the right hemisphere (Fig. 5). There was a large and peculiarly shaped cisterna magna, which was thought to be a congenital anomaly and which contained subarachnoid blood. An MRI scan performed with the 0.12-Tesla unit 10 days after admission showed the right frontal contusion with about the same definition as a second CT scan performed a few days before the MRI study. The MRI study also demonstrated a contusion in the anterior portion of the right temporal lobe not visualized with certainty on either CT scan.

Considering the patient’s relatively brief period of unconsciousness and his neurological status on admission, we were surprised that he remained confused and intermittently combative for nearly a week following the injury. He then made a rapid recovery and, at the time of the first PET study 17 days following injury, he was alert and well oriented but clearly slow in both motor and intellectual responses. Glucose metabolism was reduced about equally in the frontal pole and orbital cortex of both hemispheres (Table 1 and Fig. 6) despite the absence of evidence of structural damage in the left frontal lobe on the CT or MRI scan. Metabolism was also reduced in both anterior temporal lobes (superior temporal region, Table 1). On Day 21 his full-scale IQ was 122 and the verbal and performance values were 124 and 116, respectively. His verbal fluency was decreased. The most significant finding was evidence of substantial inattention on a number of the tests. A 133Xe-clearance CBF study performed on the same day demonstrated a normal global value with no focal abnormalities despite the significant changes in metabolism in both the frontal and temporal lobes.

All of the studies were repeated in February and March, 1985, 6 to 7 months after the patient’s accident. The CT and MRI scans were similar to each other in appearance. The CT scan demonstrated decreased attenuation, and the T1-weighted MRI images showed decreased signal in the right frontal and temporal lobes (Fig. 5). As in Case 1, MRI defined the borders of the lesions better than did the CT scan. Also, the temporal abnormality was much larger on the MRI scan than on
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FIG. 6. Case 2. Positron emission tomography scans on August 6, 1984 (upper), and February 4, 1985 (lower). The regions of interest marked on each scan are delineated in Fig. 3.

the CT scan. The PET scan demonstrated increased glucose metabolism in both frontal lobes compared to the original study, but metabolism was still depressed on the right (Table I and Fig. 6). The left but not the right anterior temporal lobe had recovered, in keeping with the appearance of encephalomalacia on the right in both the CT and MRI scans. Metabolism was slightly reduced in both cerebellar hemispheres. It should be noted that careful inspection of the images is often required to distinguish encephalomalacia with an intact rim of cerebral tissue around it from an extracerebral cyst constituting an ex vacuo phenomenon. We believe that the distinction is not important because the loss of brain substance is essentially the same in both cases.

In February, 1985, the patient's full-scale IQ was 128, and he performed the entire battery of tests adequately. However, the inattention noted at the study 3 weeks after the injury was still evident at 7 months. For example, he placed an ear upside down in a profile puzzle without recognizing his mistake. His conversation was best described as slow and somewhat vague, out of keeping with his superior IQ. He could not read for more than an hour because of difficulty with concentration. The last $^{133}$Xe-clearance CBF study, performed nearly 8 months after the injury, again demonstrated normal global flow without focal reduction.

Case 3

This 46-year-old man had a history of alcohol abuse and two prior episodes of cerebral concussion. He was admitted to our hospital soon after a fall down a flight of steps. At the time of admission he was lethargic, disoriented, and inebriated. A CT scan on admission showed a hematoma and contusion in the left frontal lobe, with slight mass effect, and a contusion in the tip of the left temporal lobe.

By the 4th day after admission he was alert and well oriented but irritable, with clear memory impairment. A CT scan showed some evolution of the left frontal lesion, and the temporal contusion was again visualized (Fig. 7). An MRI scan performed the following day showed several lesions not demonstrated on the CT scan (Fig. 7). One was a left frontal intraparenchymal hematoma with two components. An apparent fluid level (arrows) separated a dependent, crescent-shaped posterior portion from a larger globular anterior portion of the hematoma. On the T1-weighted image, the anterior portion was isointense compared to white matter and the posterior portion was slightly hypointense compared to gray matter. In contrast, on the T2-weighted image, both portions of the hematoma were hypointense, the posterior more than the anterior portion. The hyperintensity surrounding the hematoma, seen on

FIG. 7. Case 3. Computerized tomography scans on December 20, 1984 (upper), and magnetic resonance imaging (MRI) scans made on December 21, 1984 with a 1.5-Tesla unit: T1-weighted images (center) and T2-weighted images (lower). Arrows on the superior cuts of the T1- and T2-weighted MRI images represent a fluid level between two portions of the intraparenchymal hematoma. See text for further description.
demonstrated mild reductions in metabolism in both T2- but not the T1-weighted image, represented tense on the T1- and hypointense on the T2-weighted images. There were also several additional hemorrhages, surrounded by edema, much better defined in the MRI compared to the CT scans.

The PET scan, performed on the 4th day postinjury, demonstrated mild reductions in metabolism in both frontal lobes with a large focal reduction in the left orbital cortex, the principal site of the hemorrhage (Fig. 8). As in the previous cases, metabolism was reduced in both anterior temporal lobes, the left more than the right, despite a normal appearance of the right temporal lobe on both CT and MRI scans. Shortly after these studies, the patient signed out of the hospital against medical advice and has not been seen again.

Discussion

At the acute stage after head injury, MRI was superior to CT in detecting the extent of intraparenchymal hemorrhage and edema surrounding hemorrhagic regions. However, on careful inspection, sometimes retrospectively, most of the brain hemorrhages identified by MRI were also present on the CT scans. Parts of the smear subdural hematoma in Case 3 were not visualized at all on the CT scans. We have recently analyzed the evolution of intracranial hematomas in a series of patients from the acute through the subacute to the chronic stages, using T1- and T2-weighted MRI techniques. The observations are complex and beyond the scope of this report, but our analysis has convinced us that MRI will prove to be a powerful means of localizing and following the evolution of intracranial hemorrhage. Regions of decreased density, presumably representing encephalomalacia, that were present at 6 months were seen equally well with MRI and CT, except that the margins were clearer with MRI and the temporal abnormality in Case 2 was significantly larger on MRI. Brain edema was especially well visualized on T2-weighted MRI images.

Positron emission tomography detected regions of brain dysfunction, manifested by decreased glucose metabolism, that were not visualized by CT or MRI. In addition, every structural lesion detected by the imaging modalities, except for the small brain hematomas seen on MRI, was accompanied by a reduction of metabolism in the appropriate region. Particularly striking was the reduced metabolism in both anterior temporal lobes with sparing of the posterior temporal lobes in all three patients during the acute phase. In cases of unilateral lesions, metabolism was most reduced in the temporal lobe that was shown by CT and MRI to be abnormal. In Cases 1 and 2, metabolism was significantly reduced in the ostensibly normal lobe at 6 months. We interpret these findings as evidence of either residual pathology not detected by the imaging modalities or diaschisis secondary to damage in the contralateral temporal lobe.

The 133Xe-clearance CBF studies showed both the frontal and temporal abnormalities in Case 1 but failed to detect frontal and temporal structural lesions in Case 2. They did not show the contralateral reductions in metabolism in the temporal lobe in either patient. In comatose patients during the acute phase of head injury, CBF is decreased in about one-half of patients and increased (hyperemia) in the other half. In both groups of patients, cerebral metabolism is reduced commensurate with the reduction in level of consciousness measured on the GCS. Therefore, metabolism and flow are dissociated in hyperemia. However, in the subacute and chronic phases of head injury, flow and metabolism are again linked, and CBF measurements can serve as a surrogate for metabolism. Probably the failure of the method to detect the anterior temporal lobe abnormalities seen with PET was due to inadequate detector coverage of the regions rather than failure of flow to decrease with the decrease in metabolism.

The neuropsychological studies in Case 1 clearly demonstrated right frontal lobe damage during both the subacute and chronic phases of the patient’s illness. The temporal abnormalities, especially the bilaterally reduced metabolism in Cases 1 and 2, were not identified by the neuropsychological battery.

We believe that these observations in this small number of patients demonstrate the potential usefulness of these studies in achieving a better understanding of the pathophysiology of head injury. A few problems need to be addressed in the course of further developing the methodologies and applying them to large numbers of head-injured patients. The first is the resolution of the instruments. Although the images obtained with the 0.12-Tesla MRI system were generally satisfactory and helpful in the diagnosis of traumatic intracranial lesions, both the quality of the images and the diagnostic accuracy of the resistive system are substantially inferior to the 1.5-Tesla instrument, especially for hemorrhage and its evolution. Therefore, there is risk in comparing studies with the two instruments in the same patient. The PET instrument used for these studies provides a...
full-width half-maximum resolution of approximately 17 mm compared to a resolution of about 0.5 mm obtained with the GE 9800 CT scanner and the 1.5-Tesla GE Signa MRI system. Smaller regions of depressed glucose metabolism might have been detected, and the boundaries of the demonstrably abnormal regions might have been better visualized with a higher-resolution system. Muehllehner and Karp\(^{10}\) have recently completed one ring of a new PET system, which will provide about 4 mm resolution in the brain. This system will soon be available and will be used to study our head-injured patients in the future.

A second problem is localization of abnormalities in the same plane and with the same anteroposterior angle with the three imaging modalities. The GE 9800 CT scanner, the GE Signa MRI system, and the PETT V scanner provide image planes 0°, -10°, and -20° to the canthomeatal line, respectively; this explains why there is not better correspondence between the anatomical structures identified on the CT and MRI scans illustrated in this report. We are in the process of developing methods for assuring that both the plane and the angle of the scans will be as identical as possible among the modalities.

A third issue is proper management of the enormous volume of data that will be obtained from populations of patients with head injuries and other intracranial disorders. Quantitative information for hundreds of brain regions in each patient is available from CT, MRI, and PET scans. Considering the number of patients required for an effective clinical investigation (each patient being studied several times), it is clear that the most rigorous method of data entry and data base management will be required to obtain the best use of the information. A specific research question to be answered is the value of quantitative T1 and T2 values, compared to visual inspection, in the identification of brain lesions.

We believe that these various modalities used in combination offer considerable promise for further elucidating the pathophysiology of head injury. However, assessment of their usefulness will depend ultimately on the accumulation of large volumes of data on many patients with a wide range of intracranial pathology produced by head injury.

References


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