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Neuroimaging of Head Injury

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Neuroimaging in head traumatology has received a decisive impulse with the advent of computed tomography (CT). CT scanning is noninvasive and therefore repeatable as the need arises; it affords direct and accurate visualization of brain damage at all stages, and permits intelligent planning of surgery for expanding lesions, sometimes before such lesions produce neurological deterioration. Also, as a byproduct of more realistic definition of traumatic brain damage, CT scanning has revealed the obsolescence of classic neurotraumatological terminology as well as some reliable correlations between different lesions and final outcome. Cerebral angiography, however, retains its full value in vascular traumatic pathology and the rare cases of traumatic aneurysm, which cannot be detected with certainty by CT scanning. Even newer methods are positron-emission tomography (PET) and nuclear magnetic resonance (NMR), the former yielding valuable information on brain tissue metabolism, the latter being effective in differentiating gray matter from white and estimating their water contents.

Severe head injury continues to represent a major problem in public health. While considerable progress has been made in the management of such cases, traffic accidents have not decreased in either frequency or severity, and both the morbidity and the mortality from such causes remain impressively high. To make things worse, the majority of victims are young people; also, initially mild injuries may be irreversibly aggravated by potentially preventable secondary lesions; and a sizable number of survivors end up with permanent neurologic sequels (Bricolo, Turazzi, and Ferriotti, 1980; Miller, Sweet, Narayan, and Becker 1978).

In recent years several work groups (Becker et al., 1977; Bowers and Marshall, 1980; Bruce, Shut, Bruno, Wood, and Sutton 1978; Gennarelli,

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Thibault et al., 1982; Langfitt, 1978; Marshall, Smith, and Shapiro 1979; Miller et al., 1981; Turazzi, Bricolo, Pasut, and Formenton, 1987) have achieved better overall outcomes by adopting a new therapeutic protocol based essentially on the early detection and timely surgical treatment of expanding intracranial lesions, intracranial pressure monitoring, immediate correction of hypertensive episodes, and systematic prevention of secondary brain damage. All this is the end result of a changing approach to the treatment of such cases, made possible by better understanding of the pathophysiology of brain injury—signally the fact that shock, hypoxia, hypercarbia, and internal brain displacement caused by an expanding lesion represent as many additional insults delivered to an already injured brain, themselves capable of aggravating cerebral ischemia, edema and hypertension to the effect of modifying the clinical course most adversely.

Thus, efforts were increasingly concentrated toward the prevention of functional deterioration rather than the implementation of extraordinary therapeutic measures once clinical worsening was extant. It is becoming more and more evident that many recuperable patients can survive in the best possible conditions only if they are treated in a specialized environment where emergency services are efficiently organized, neuroradiological facilities are available at all times for the timely detection of brain lesions, surgery is performed rationally, and intensive care measures are more refined (Becker et al., 1977; Bowers and Marshall, 1980; Bricolo and Pasut, 1984; Miller et al., 1981).

Diagnostic Evaluation of Head Injury

Every head injury entails involvement of skull contents commensurate with the severity of the traumatic event. From the anatomopathological point of view this involvement is the effect of a mechanical force delivered to the brain in the form of hyperemia, petechiae or overt bleeding, brain swelling, focal laceration and contusion, and all the way to brain tissue colliquation. All these manifestations may be confined to one or more areas of the brain or involve the brain as a whole (Adams, 1975; Adams, Mitchell, Graham, and Doyle, 1977).

By their characteristic pathogenesis, these events may be classified under at least two main headings, namely direct and secondary. Direct actions are those attributable solely to physical impact. These are produced by the traction and rotation forces that are exerted on the brain mass at the time of injury and the consequent traumatic action of the skull itself on brain substance; they are classified as immediate (focal laceration and contusion, diffuse white matter lesions, primary brainstem damage) and delayed (intracranial hematoma, cerebral edema). Secondary lesions represent complications of the initial event and comprise blood supply and vasomotor alterations conducive to cerebral

hypoxia or ischemia, increased intracranial pressure due to an expanding lesion, and the onset of cerebral and/or meningeal infection.

Thus, the diagnostic procedures used for assessing severe head injury should ideally provide information on the type, place, and magnitude of brain damage in the greatest possible detail and in the shortest possible time, mainly for the timely detection of expanding intracranial lesions requiring surgery—possibly before cerebral herniation becomes clinically apparent; and at the same time to afford the planning of medical and/or intensive care measures more likely to succeed in preventing secondary intracranial lesions.

The diagnostic approach to the patient with major head injury involves both clinical and instrumental procedures; the former to elicit a preliminary assessment of the injury sustained and to make a tentative layout of priorities in subsequent diagnostic tests; and the latter (skull roentgenography, CT scanning, cerebral angiography) to define the injury more accurately and extract guidance in regard to necessary surgery or targeted medical treatment.

Clinical Neurological Evaluation

The first and foremost aspect of the presenting picture of head injury is the assessment of consciousness, which may be altered within a broad gamut of conditions ranging from wakefulness to deep coma and may shift from one level to another on repeat examination of the same patient.

Because the classical procedures of neurological semiotics are often poorly applicable to head injury patients—particularly in emergency conditions—the need was felt for a sufficiently simple, contributory and easily interpreted method for assessing traumatic coma. Of all the classifications of coma proposed in succession, the Glasgow Coma Scale (Teasdale and Jennett, 1974) proved best in terms of these requirements and is therefore more widely used than any other. The Glasgow Coma Scale (GCS) is a practical score system designed to express the patient's neurological status numerically through the exploration of three main functions: namely eye opening, best motor response, and best verbal response, each being given a top score for normal responses and gradually diminishing scores for increasingly pathological responses. The sum of these three scores varies from three for deep coma to fifteen for fully retained consciousness; the value so obtained provides an immediate overall assessment of the patient's neurologic condition. The introduction of the GCS also made it possible to assemble neurotraumatological data banks to the effect of grouping separate series of patients into homogeneous categories and comparing the effectiveness of treatment protocols adopted in various Centers, at the same time providing valid elements for reliable prediction of final outcome already in the acute stage of trauma (Braakman, Gelpke, Habbema, Maas, and Minderhoud, 1980; Bricolo et al., 1982; Gelpke, Braakman, Habbema, and Hilden, 1983; Jennett et al., 1977; Narayan et al.,

1981; Teasdale, Parker, Murray, and Jennett, 1979; Young et al., 1981).

Despite these obvious advantages, the GCS suffers considerable limitation from being too concise and simplistic to afford exhaustive clinical assessment of the head injury patient. Accordingly, it should always be supplemented by a more traditional neurological examination taking into account pupil size and reactivity, spontaneous and reflex eye movements, motor alterations of all four limbs, and also some essential vital signs such as the pulse rate, arterial blood pressure and respiration (Bricolo, 1971; Bricolo et al., 1975; Plum and Posner, 1966).

The semiological exploration of these few nervous functions in addition to the three basic features of the GCS yields valuable information for the topographic localization of brain damage; taken together, these data constitute a useful yardstick for determining on successive examinations whether the patient is improving or deteriorating. As a general rule, deepening coma, progressive neurological deterioration and the emergence of vital function disorders suggest an expanding intracranial mass and definitely indicate further instrumental exploration.

Instrumental Evaluation

Skull Roentgenography

Before the advent of computed tomography, skull roentgenography was performed routinely in nearly all head injury cases regardless of clinical status. Now, with CT scanning widely available, skull roentgenography has lost part of its former value in severe head injury while retaining more validity in mild trauma, especially for identifying patients at high risk of later complications (Cooper and Ho, 1983; Jennett, 1980). The site and morphology of a skull fracture, at any rate, may yield indirect information on the magnitude of the traumatic event, the type of impact involved, and the localization of concomitant brain damage. In that respect it should be borne in mind that brain lesions do not necessarily correlate with the presence or location of a skull fracture. Very severe head injury may occur without any fracture; and conversely, large fractures may occur without concomitant brain damage (Galbraith, 1976; Jamieson and Yelland, 1968).

Skull radiographs are usually taken in the anteroposterior and lateral views. Towne's view is needed for occipital fractures. Skull fractures may be grouped schematically as linear, depressed, and open. Linear fractures are the most common, accounting for about 80% of all skull fractures. Depending on their location, they are divided into fractures of the vault, ditto irradiating to the base, and isolated fractures of the skull base. Fractures of the vault are very common and relatively unimportant unless their location suggests injury to the meningeal artery or dural sinus (see Figure 1), with the attendant risk of an

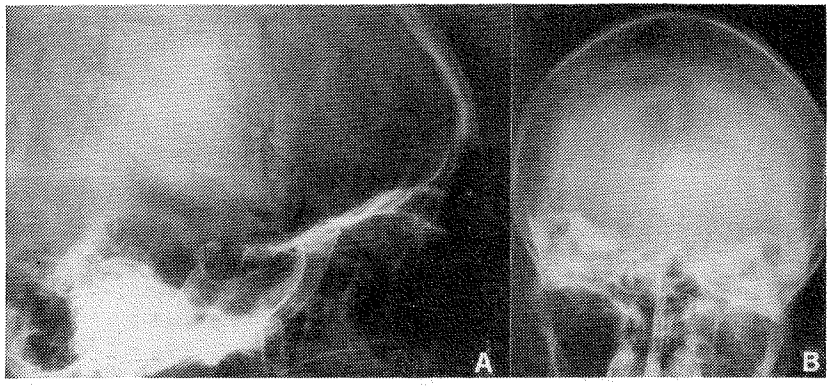

Figure 1: A: Double fracture line of temporal bone, crossing the course of the meningeal artery; B: Fracture of left occipital bone crossing the midline and involving the lateral sinus.

extradural hematoma. Fractures of the skull base are for the most part extensions of vault fractures and are often undetectable in routine skull radiographs. They can be suspected clinically by the presence of blood from the ear (otorrhagia) or cranial nerve palsies in an alert patient. Concomitant laceration of the overlying dura and brain may result in cerebrospinal fluid discharge from the ear (otorrhea). Fractures in the floor of the anterior fossa often extend into the frontal, ethmoid and sphenoid sinuses, possibly causing CSF leakage into the paranasal sinuses with resultant rhinoliquorrhea and/or pneumocephalus. All these ruptures may afford entry to bacterial invaders to produce secondary meningitis or brain abscess. In cases of this type, valuable information can be obtained with tomography, CT scanning, and water-soluble CT cisternography.

A depressed or impacted fracture is one in which one or more bone fragments are permanently displaced into the skull cavity; in the frontal and temporal areas, such fractures may be responsible for later epilepsy (see Figure 2A). Obviously of surgical interest, depressed fractures must be diagnosed as accurately as possible in terms of their precise site and topography, depth of fragment sinking, and relationship with neighboring vascular structures and paranasal sinuses. A tangential view is needed to delineate depressed fragments.

An open or compound fracture is one in which the integument over the fracture site is torn and the fracture itself is exposed; if the dura mater is injured, laceration of the underlying brain may be extant (see Figure 2B). Timely diagnosis is important in regard to immediate surgical debridement and adequate antibiotic coverage to avert the severe cerebromeningeal infection that is likely to develop in such cases.

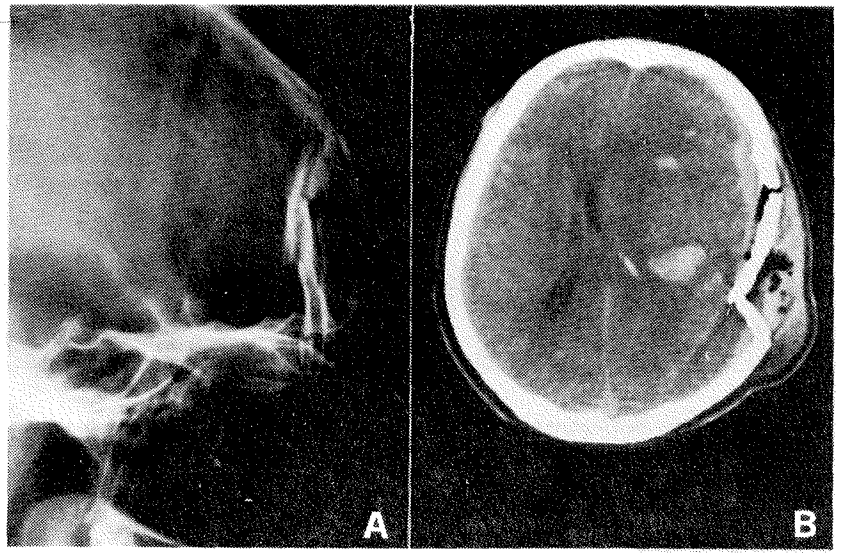

Figure 2: A: Severe depressed fracture of frontal bone with multiple fragments and frontal sinus involvement; B: Depressed fracture of right parietal bone with intraparenchymal hematoma, film of subdural hematoma and bilateral DWSI.

CT Scanning

The decisive meaningfulness of CT scanning in the management of head injury cases was apparent since its introduction in clinical practice (Davis, Taveras, Roberson, Ackerman, and Dreisback, 1977; French and Dublin, 1977; Koo and LaRocque, 1977; Merino de Villasante and Taveras, 1976; New, Scott, Schnur, Davis, and Taveras, 1974; Paxton and Ambrose, 1974; Zimmerman, Bilaniuk, Gennarelli et al., 1978). The new diagnostic procedure provided direct and accurate visualization of intracranial lesions, the foremost advantage in neurotraumatology being the prompt and reliable diagnosis of intracranial masses (Galbraith and Teasdale, 1981), in turn permitting accurate target surgery when necessary, sometimes even before the onset of important clinical deterioration. Also, the advent of CT scanning threw the classical terminology of neurotraumatology into obsolescence as a result of more accurate definition of traumatic brain damage; and it proved capable of detecting some formerly unsuspected lesions, such as the bilateral ones that angiography failed to show (Caillé, Cohadon, Becke, and Costant, 1975; Sweet, Miller, Lipper, Kishore, and Becker, 1978; Vigoroux, Legré, Guillermain, and Tapias, 1979), and also diffuse lesions that used to be diagnosed only at autopsy

(Snoek, Jennett, Adams, Graham, and Doyle, 1979; Zimmerman, Bilaniuk, and Gennarelli, 1978).

Furthermore, being a rapid and noninvasive procedure suitable for unlimited repetition, CT scanning has been used very extensively for monitoring the intracranial condition all through the acute phase of severe head injury, at the same time revealing the early morphological changes that occur in the brain after injury, describing their evolution in time, and affording target treatment as well as verification of its effects (Cooper, Maravilla, Moody and Clark, 1979; Pasut, Beltramello, Grosslercher, and Bricolo, 1981; Pasut, Beltramello, and Bricolo, 1983b; Roberson, Kishore, Miller, Lipper, and Becker 1979).

Because of these characteristics, CT scanning turned out to represent a major breakthrough in traumatic brain pathology; by now, an abundance of evidence has been assembled to show that its introduction in clinical practice has contributed materially to the improvement of treatment and hence of the outcome of severe head injury (Becker et al., 1977; Bowers and Marshall, 1980; Bricolo et al., 1982; Bruce et al., 1978; Clifton et al., 1980; Gennarelli, Spielman et al., 1982; Miller et al., 1981: Turazzi et al., 1987).

The opportunities offered by CT scanning are so great that practically all neurological departments are now equipped with this new diagnostic tool; in many centers, indeed, CT scanning ranks as the prime radiological technique for assessing acute head injury. In such cases, however, and more so if the patient is comatose, some precautions should be taken before scanning. First, one must always consider the possibility of associated cervical trauma; a radiograph of the cervical spine should be made, and suitable cervical immobilization provided in the presence of fracture or dislocation before the patient is removed to the CT room. Second, in positioning the patient on the radiological table, avoid excessive head bending, since this might cause venous engorgement of the brain followed by increased intracranial pressure and rapid clinical deterioration. Third, if the patient is agitated, mild anesthesia may help avoid motion artifacts that might confuse or hide an important brain lesion. Fourth, make each examination as complete as possible to provide visualization of all anatomical planes from skull basis to top, so as to detect and assess all possible brain lesions, particularly those in a subtemporal or subfrontal location. And fifth, bear in mind that contrast enhancement may be useful only in well-defined cases, such as chronic isodense subdural hematoma; other than that it is best avoided, since it may lead to misinterpretation of the CT picture or even cause harm to the patient if the blood-brain barrier has been disrupted (French, 1978).

CT Aspects of Traumatic Brain Lesions

CT scanning readily demonstrates the basic aspects of traumatic brain pathology such as hemorrhage, contusion, edema, and ischemia; it affords

direct visibility of brain ventricle displacements due to a space-taking intracranial lesion; and it permits accurate assessment of distant sequels of head injury such as cerebral atrophy and dilation of the ventricular system. The advent of CT further made it possible to detect and define pathological features formerly discovered only at autopsy (for instance, diffuse lesions of the white matter), or suspected from known correlations between the clinical picture and intracranial pressure (for instance, brain swelling).

CT scanning will readily tell an extracerebral from an intracerebral lesion. Current literature, at any rate, shows an increasing tendency to distinguish focal from diffuse lesions—on the strength, also, of the correlations that these groups of lesions have shown with clinical picture and outcome (Bricolo et al., 1982; Gennarelli, Thibault et al., 1982; Lobato et al., 1983; Turazzi et al., 1987).

Focal lesions include epidural hematoma, subdural hematoma, hemorrhagic contusion, and intracerebral hematoma. These are identified in CT scans as clearly outlined areas of altered density within or without the brain parenchyma; they can produce local brain damage (expressed by the corresponding clinical picture) or behave like expanding lesions to produce ventricular compression, cerebral herniation, and in extreme cases, brainstem compression.

Diffuse lesions comprise a number of as yet incompletely defined alterations, not always identifiable in CT scans, or at any rate, appearing as small hemorrhagic areas scattered in both hemispheres, invariably associated with severe neurological impairment and a poor outcome.

Epidural Hematoma

An acute epidural hematoma is a blood collection forming between the inner face of the cranium and the dura mater, usually caused by laceration of a meningeal vessel—more rarely of the diploetic veins or of a dural sinus. In most cases, the simultaneous presence of extradural hematoma and skull fracture is noted in roentgenograms or at operation (Bricolo and Pasut, 1984; Gallagher and Browder, 1968; Heiskanen, 1975; Jamieson and Yelland, 1968).

The best known location of extradural hematoma is the temporal area (Figure 3A), due to laceration of the middle meningeal artery caused by fracture of the squamous portion of the temporal bone. Less common locations are the parietal and frontal areas (Figure 3B); and even less common (indeed, rare) are occipital extradural hematomas in the posterior fossa or involving the superior longitudinal sinus (Bricolo and Pasut, 1984; Cordobés et al., 1981; Kvarnes and Trumpy, 1978; Stone, Schaffer, Ramsey, and Moody, 1979) [Figure 4]. One atypical location is the frontobasal (more often temporobasal) region, where extradural hematoma is sometimes missed because of movement artifacts in the CT scans.

The CT image of extradural hematoma is characteristically a clear-cut area of increased density, adherent to the inner table of the skull and appearing

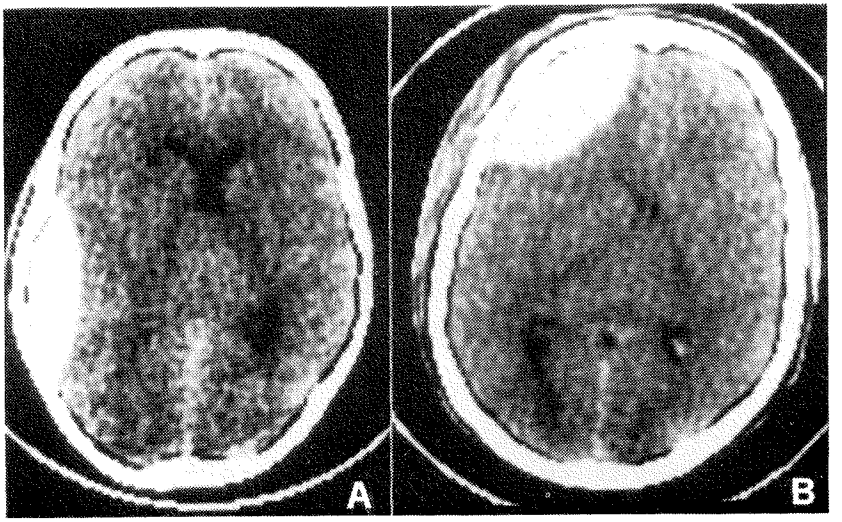

Figure 3: A: Extradural hematoma in typical left temporal location; B: Extradural hematoma in atypical left frontal location.

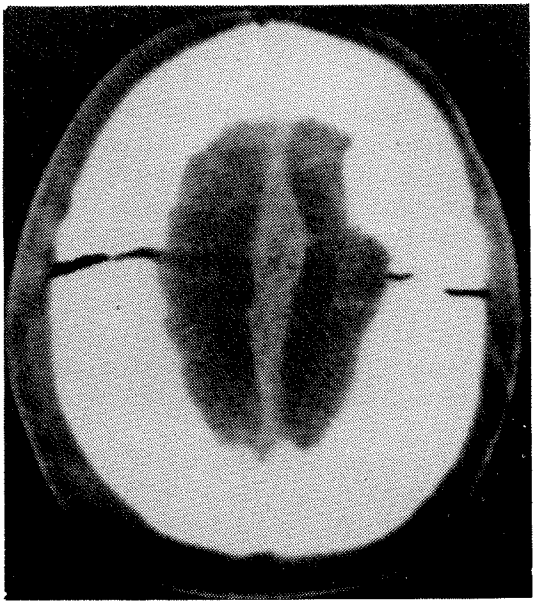

Figure 4: Extradural hematoma of superior longitudinal sinus caused by biparietal fracture extending to vertex.

much like a biconvex lens because of the detachment of the dura from bone by the agency of the blood collection. Because blood generally accumulates under considerable pressure, the hematoma grows in size very rapidly and the patient's neurological deterioration follows suit (Figure 5A and 5B). For this reason, extradural hematoma constitutes a neurosurgical emergency.

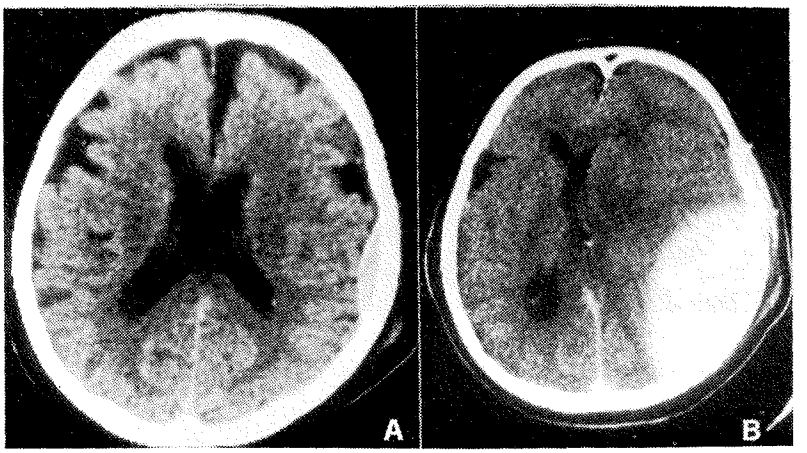

Figure 5: A: Small right temporal extradural hematoma with no significant mass effect; B: Three hours later: very great increase of lesion size with evidence of uncal impingement.

Following a low-velocity impact, extradural hematoma often occurs as a single lesion, causing effacement of the underlying cerebral sulci with ipsilateral ventricular compression and midline shift. In high-velocity impacts, extradural hematoma may be compounded by subdural hematoma—a situation in which the distinction of the two blood collections is not always apparent on CT scans; other possible combinations are with hemorrhagic contusion of the brain and secondary brainstem lesion due to transtentorial herniation. Serial CT scans have shown that extradural hematoma may be small at inception or even be masked by a contralateral subdural hematoma, only to enlarge after surgical evacuation of the latter and the resultant cerebral decompression (Roberson et al., 1979).

Sometimes an extradural hematoma, especially of venous origin, may run a subacute course (Bergstrom, Ericson, Levander and Svedsen, 1977); also described are some instances of chronic extradural hematoma, probably reflecting the tamponade of bleeding by the pressure of the collected blood (Bullock and Van Dellen, 1982; Hirsch, 1980). In such cases, neurological signs are more subdolous and the CT scan image may appear less dense than the adjacent parenchyma. Serial CT scans afford effective monitoring of chronic extradural hematoma and timely planning of evacuative surgery.

Subdural Hematoma

An acute subdural hematoma is an extravasated blood collection forming in the virtual space between the dura and the leptomeninx, variously admixed with cerebrospinal fluid. There is a pure form of subdural hematoma with no underlying brain lesion (Figure 6), in which the collected blood originates from

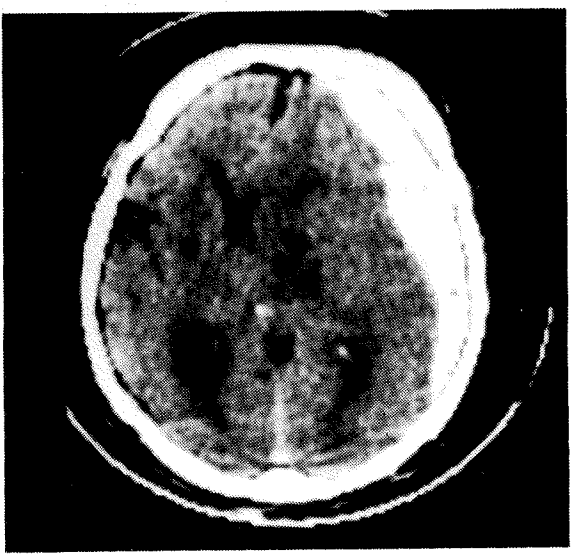

Figure 6: Pure acute subdural hematoma of right convexity with severe compression of the ventricular system.

venous vessels bridging the dura and arachnoid; but the more frequent occurrence is subdural bleeding associated with hemorrhagic contusion (Figure 7A and 7B) or intracerebral hematoma, in which blood comes primarily from lacerated brain cortex and meninges and then expands into the subdural space (Adams, 1975; Jamieson and Yelland, 1972). In some cases, subdural hematoma develops after surgical evacuation of an intracerebral hematoma, this being equivalent to removal of the former tamponade of lacerated blood vessels (Davis et al., 1977).

The typical aspect of subdural hematoma in CT scans is a hyperdense crescent image, usually localized in a parietal or frontal area but often involving the whole brain convexity sometimes on both sides (French and Dublin, 1977; Merino de Villasante and Taveras, 1976; Zimmerman, Bilaniuk, and Gennarelli, 1978); subdural hematoma is accompanied by effacement of the underlying sulci and by ventricular compression and midline shift commensurate with coexisting brain atrophy. When subdural hematoma is compounded

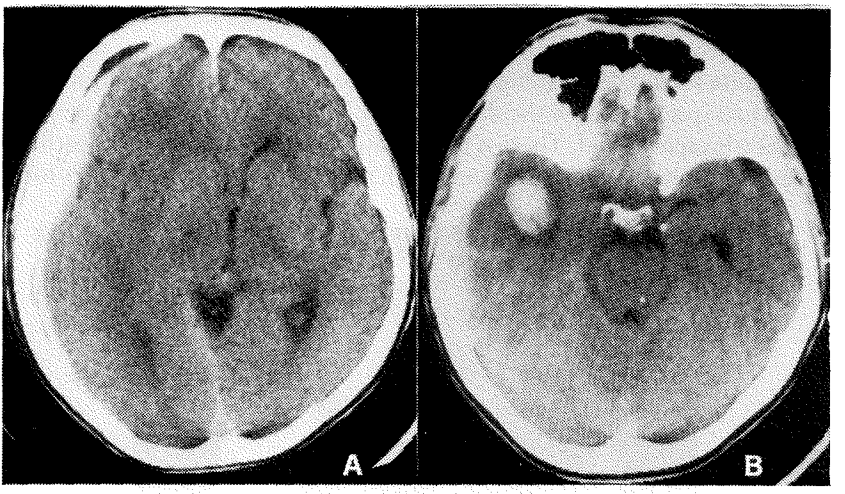

Figure 7: Subdural hematoma of left convexity (A) with left temporal hemorrhagic contusion (B).

by other intracerebral lesions such as hematoma or hemorrhagic contusion, these appear in the CT scan as areas of altered parenchymal density right below the subdural blood collection (Fell, Fitgerald, Moiel, and Caram 1975; Forbes, Sheedy, Pipgras, and Houser, 1978).

One uncommon location of subdural hematoma is the interhemispheric area, usually due to traumatic laceration of veins bridging the superior longitudinal sinus and mesial surface of the brain hemisphere. The corresponding CT image is a hyperdense area, usually in the parieto-occipital region, with a broad base adherent to the falx and a convex outline facing the mesial aspect of the involved hemisphere (see Figure 8) the resulting compression is responsible for more or less marked subfalcial herniation of the corpus callosum (Glista, Reichman, Brumlik, and Fine, 1978; Pasut, Beltramello and Bricolo, 1982; Zimmerman, Russell, Yurberg, and Leeds, 1982). Differential diagnosis is with extradural hematoma of the superior longitudinal sinus, also featuring a large blood effusion at the vertex; in that case, however, unlike hemispheric subdural hematoma, the hematoma extends contralaterally and is often associated with a transverse biparietal fracture across the vertex (see Figure 4).

Subdural hematoma also exists in a subacute and a chronic variety (the latter representing a sequel of head injury). These two forms can be distinguished by the CT aspect of blood collections, which differs according to the time of their formation. The density pattern of intracranial hematomas is determined primarily by the amount of hemoglobin contained in the blood

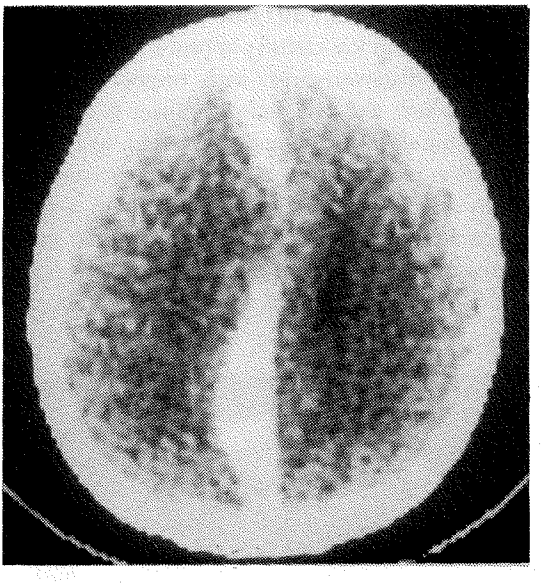

Figure 8: Left interhemispheric subdural hematoma: hyperdense blood collection involving the mesial surface of the left hemisphere, not reaching beyond the falx.

collection and by the duration of its degradation process (Bergstrom et al., 1977; French and Dublin, 1977; New and Aronow, 1976).

In the first three days following the traumatic event, subdural hematomas are markedly hyperdense—especially at the start of the coagulation process. Over the next ten days, hemoglobin undergoes progressive degradation and the CT density decreases gradually until it matches that of normal brain parenchyma, usually between one and three weeks after the injury. An isodense subdural hematoma is easily detected with last-generation CT machines but not so with older ones because of poor resolution. The presence of an isodense hematoma, at any rate, can be suspected from indirect evidence such as the midline shift, mass effect on brain ventricles in the absence of areas of abnormal density in the brain, and effacement of cerebral sulci on the ipsilateral brain convexity (Kim, Hammati, and Weinberg, 1978).

In some cases of this type one may find it advantageous to use contrast enhancement, which will visualize the membrane that forms around the hematoma in the chronic phase (Amendola and Ostrum, 1977; Forbes et al., 1978; Lusin, Nakagawa, and Bender, 1978).

Within a period of four to six weeks, the corpuscular part of a subdural hematoma is completely reabsorbed and only the liquid component is left, this being less dense than the surrounding brain parenchyma. Thus begins a

chronic subdural hematoma, usually identified in CT scans without difficulty due to its crescentic shape and low-density appearance (Figure 9). Sometimes, problems of differential diagnosis arise with respect to pictures of post-traumatic atrophy, in which the subarachnoid space is dilated as a result of cerebrospinal fluid accumulation (subdural hygroma). In that case, however, the hypodense collection is more often bilateral and the cerebral sulci appear enlarged rather than effaced. Rebleeding may occur in a chronic or near-chronic subdural hematoma, to produce hyperdense areas interspersed with isodense or hypodense areas in repeat CT scans (Bergstrom et al., 1977; Davis, Taveras, Roberson, and Ackerman, 1976; French and Dublin, 1977).

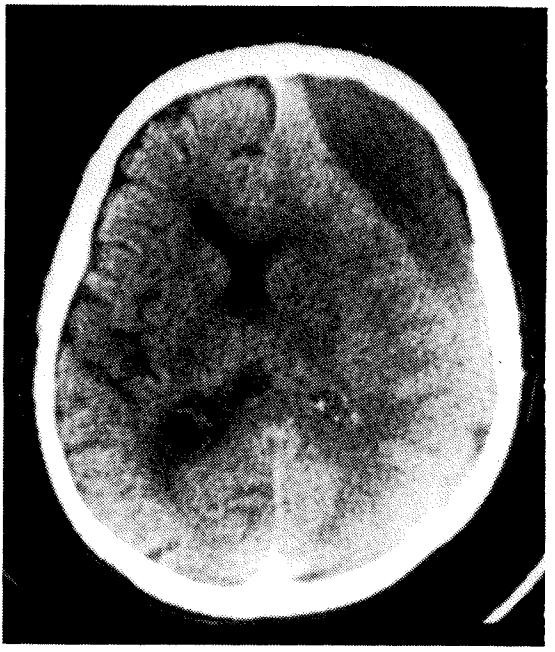

Figure 9: Chronic right frontoparietal subdural hematoma, completely hypodense.

Hemorrhagic Contusion

Hemorrhagic contusion usually appears as an admixture of small cortical hemorrhages and focal areas of brain tissue necrosis beneath the site of the impact. Such lesions are formed at the time of injury, more commonly in the frontal, temporal and occipital poles—that is, in areas of the brain that are

projected by the traumatic force against rigid, irregular structures of the inner skull: thus the rugosities of the anterior skull base for the frontal pole (Figure 10), the petromastoid and minor wings of sphenoid bone for the temporal pole, and the inner surface of the occipital bone for the occipital pole (Adams, 1975).

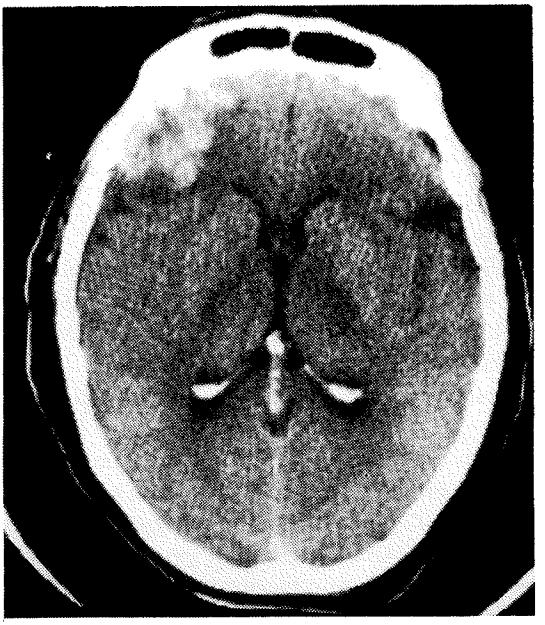

Figure 10: Left frontobasal hemorrhagic contusion.

The CT scan rendition of hemorrhagic contusion is an irregular combination of hyperdense and hypodense areas scattered in cortical and subcortical areas of the brain—sometimes described as a salt-and-pepper appearance (French and Dublin, 1977; Koo and LaRocque, 1977; Merino de Villasante and Taveras, 1976).

Hypodense areas stand not only for brain tissue necrosis but also for the edema that is invariably associated with this lesion—sometimes to such a degree that hemorrhagic contusion behaves just like an expanding intracranial process with brain compression and midline shifting. Serial CT scans are needed at this stage to watch the progress of lesions and plan decompressive polectomy when necessary.

Hemorrhagic contusion may occur as a single lesion or as multiple lesions. In the former case the contused area is right below the point of impact; rebound contusions are usually in the contralateral hemisphere at or near the end of the blow line and for the most part in the frontobasal and temporal regions.

With time, as blood is reabsorbed, hemorrhagic contusion evolves into areas of cerebral atrophy; these can be seen in CT scans as hypodense spots (Zimmerman and Bilaniuk, 1978).

Intracerebral Hematoma

An intracerebral hematoma is a blood collection within the brain parenchyma, more commonly located in frontal or temporal areas. The predominant cause of intracerebral hematoma is the traumatic laceration of small intraparenchymal vessels; in rare cases, bleeding originates from a branch of a cerebral artery. The CT scan expression of intracerebral hematoma is an irregularly shaped area of high density surrounded by a low-density halo representing perilesional edema (French and Dublin, 1977; Koo and LaRocque, 1977; Merino de Villasante and Taveras, 1976). Intracerebral hematoma often extends in depth into the white matter, thereby exerting an important mass effect on adjacent structures (Zimmerman and Bilaniuk, 1978) and sometimes opening into a lateral ventricle through ependymal lacerations. Unlike intracerebral hematomas of vascular origin, which are usually single and sharply outlined, those of traumatic origin are often multiple. Sometimes, however, angiography is needed to differentiate the traumatic or spontaneous (arteriovenous malfunction) nature of these blood collections.

The spontaneous evolution of intracerebral hematoma takes two to four weeks, during which the blood collection is gradually reabsorbed to leave a permanent poroencephalic cavity. During reabsorption, perilesional edema becomes more marked; the mass effect persists through the subsequent phase of isodensity (Zimmerman and Bilaniuk, 1978). In a small percentage of cases, intracerebral hematoma develops in the first week after injury, mostly after surgical evacuation of an extracerebral hematoma (Figure 11) [Baratham and Dennyson, 1972; Brown, Mullan, and Duda, 1978; Diaz, Yock, Larson, and Rockswold, 1979; Gudeman et al., 1979].

These are also called delayed hematomas, the involved mechanism being in all likelihood a vacuum effect that occurs when the plugging effect of the original lesion ceases to work. Favoring factors may include the loss of cerebral self-regulation, increased cerebral blood flow, and increased venous pressure, all contributing to promote diapedesis and hence the gradual development of the blood collection. The mass effect of intracerebral hematoma is important enough to require additional surgery in about half the cases. Serial CT scans made with last-generation machines may help clarify the origin of this rare but serious complication of head injury, hopefully to the effect of preventing it.

Diffuse Brain Lesions

The term "diffuse brain lesions" covers a group of brain lesions (not yet fully identified) characterized by diffuse brain damage, not always macroscopically

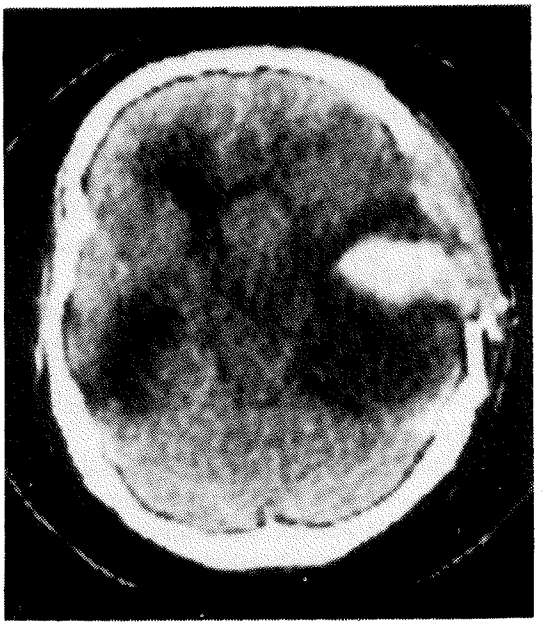

Figure 11: Right temporal intracerebral hematoma with marked perilesional edema, developed after surgical evacuation of a subdural hematoma in the right convexity.

evident but invariably associated with severe impairment of consciousness.

With few exceptions (Strich, 1961), the literature on diffuse brain lesions in neuropathology is relatively recent (Adams, 1975; Adams et al., 1977; Adams, Graham, Murray, and Scott, 1982). Experimental work (Gennarelli et al., 1982; Ommaya and Gennarelli, 1974) has shown that these lesions are formed at the time of impact, for the most part in blunt high-velocity traumatic events, and particularly when rotational acceleration is at play, as a result of strain and dislocation of brain tissue with direct tearing of axonal nerve fibers. The clinical and anatomical pictures vary both in description and in severity (Gennarelli, 1982).

Diffuse brain injury is characterized by nonfocal anatomical alterations of varying extension, sufficient at any rate to cause an impairment of consciousness lasting for several days after the injury. This distinguishes diffuse brain injury from cerebral concussion—a far less severe condition characterized by only microscopical neuronal abnormalities, probably physiological rather than structural, and at any rate producing a loss of consciousness that resolves within 24 hours and leaves post-traumatic amnesia as its sole sequel. The CT picture of diffuse brain injury is noncontributory for focal lesions; in some cases, especially in children, it is associated with CT evidence of brain swelling (discussed later) in terms of small ventricles and poorly evident basal cisterns

(Figure 16A and B later section), sometimes leaving a post-traumatic hydrocephalus as a permanent sequel (Zimmerman and Bilaniuk, 1979). Serial CT scans may be useful in diffuse brain injury to verify the effects of medical and intensive-care treatment, especially in the presence of brain swelling or raised intracranial pressure, and also for the early detection of hydrocephalus.

The clinical evolution of diffuse brain injury depends on the extent and location of anatomical damage; while most patients make fairly good long-term recoveries, some show severe residual disability and a few go into persistent coma and may die from intercurrent disease.

The most severe instance of diffuse brain lesion is represented by white matter shearing injury, characterized by true disruption of axonal nerve fibers, particularly in the corpus callosum, superior cerebellar peduncles, upper brainstem, and the white matter of cerebral and cerebellar hemispheres (Adams et al., 1977; Gennarelli, Thibault et al., 1982).

The typical clinical picture of white matter shearing injury is a deep and immediate coma with signs of decerebration and brainstem involvement. Such lesions are often incompatible with life; many survivors, too, show permanent and severe neuro-psychic sequels or a persistent vegetative status (Gennarelli, Spielman et al., 1982; Miller et al., 1981; Turazzi and Bricolo, 1977).

CT scanning reveals the presence of small high-density areas of hemorrhage with no mass effect, located predominantly in the corpus callosum, around the third ventricle, and in the cerebral white matter [Figure 12A] (Pasut, Beltramello, Faccioli, and Bricolo, 1983; Snoek et al., 1979; Zimmerman, Bilaniuk, and Gennarelli, 1978).

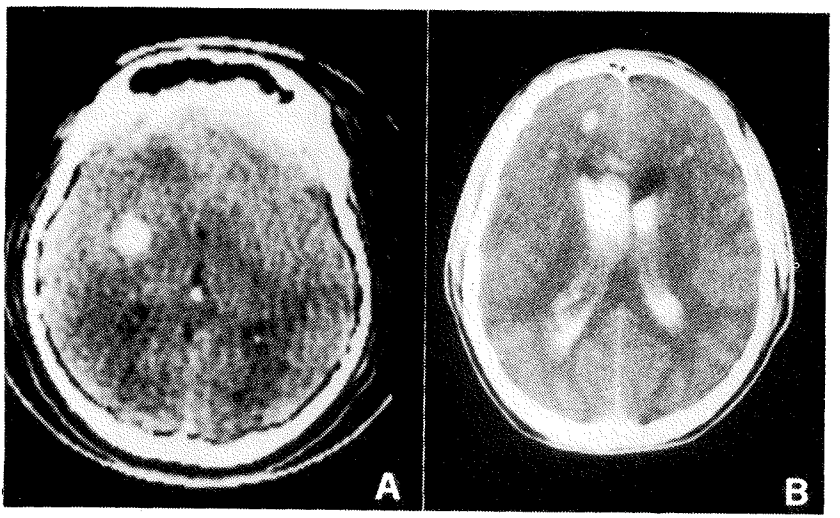

Figure 12: A: White matter laceration near the left internal capsule; B: Diffuse injury of white matter in the frontal lobes with massive hemorrhagic clots in the ventricles.

In some cases, massive ventricular flooding occurs as an expression of the devastating effects of these high-velocity impacts on brain parenchyma (Figure 12B) and in a few instances, evidence is detected of the so-called "primary brainstem lesions" the pathogensis of which is debated (Bricolo, Turazzi, Alexandre, and Rizzuto, 1977; Mitchell and Adams, 1973; Snoek et al., 1979). These are characterized by the presence of small hemorrhagic areas, usually single and irregularly shaped, localized mainly in the rostral brainstem (Figure 13), in the absence of significant supratentorial lesions (Pasut, Beltramello, and Bricolo, 1983; Tsai et al., 1980; Tsubokawa et al., 1980). Possible associated lesions are intraventricular hemorrhage, bilateral brain swelling with ventricular and cisternal compression, and subarachnoid hemorrhage.

Diffuse white matter lacerations become isodense in a short time (approximately 10 days); and because of their small size they seldom result in definite areas of encephalomalacia (Zimmerman and Bilaniuk, 1978). Still, the initial mass effect produced by bilateral swelling evolves into atrophic cerebral dilatation in a relatively short time, concomitant with axonal degeneration of the white matter. CT scanning with the last generation, high-resolution machines will probably afford more accurate localization of damaged white matter areas, at the same time yielding information on the time course of diffuse axonal damage; also, emergent correlations between these findings and the patient's neurological status may suggest new ways of treating this severe condition.

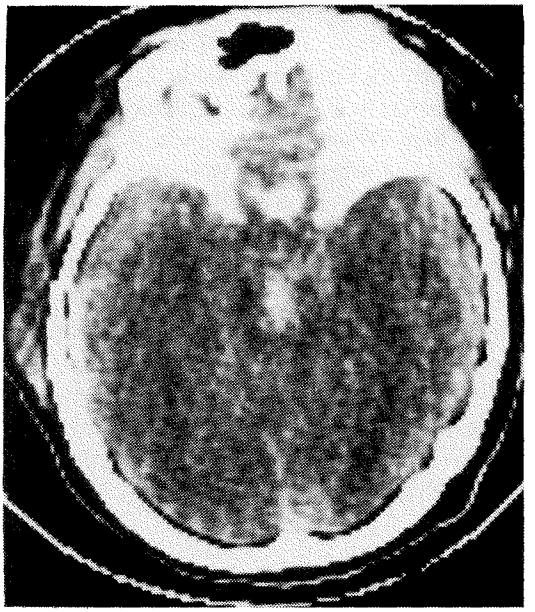

Figure 13: Primary hemorrhagic lesion in upper brainstem.

Cerebral Edema

Brain edema is an increase in brain bulk produced by an increase in brain tissue water content (Fishman, 1975), this being a very frequent accompaniment to acute traumatic brain lesions. The pathogenesis of cerebral edema has been long debated in the literature, and several classifications have been proposed. Of these, the more widely accepted distinction is between a vasogenic and a cytotoxic edema (Klatzo, Winiewski, and Steinwall 1967), with the understanding that the two forms may coexist.

Vasogenic edema is characterized by the passage of fluid into the extracellular space as a result of increased permeability of cerebral capillary endothelia; it is classically associated with major head injury and often shows a focal distribution, so that it may produce the effects of a true space-taking intracranial lesion.

Cytotoxic edema is characterized by swelling of glial and neronal cells due to intracellular accumulation of water and sodium caused by a disturbance of cellular osmoregulation. This is typically a diffuse edema, usually secondary to ischemic brain damage. Cerebral ischemia, in turn, may damage the blood-brain barrier and bring about a superimposed vasogenic edema.

The advent of CT scanning has added considerably to our knowledge of cerebral edema, in particular by making it possible to distinguish two important morphological variants as early as in the first week after the injury: namely perilesional edema and focal hypodensity (Miller, Gudeman, Kishore, and Becker, 1980; Sweet et al., 1978; Yoshino, Yamaki, Higuchi, Horikawa, and Hirakawa, 1985; Zimmerman, Leeds, and Naidich, 1977).

Perilesional edema occurs as a low-density, shaded halo that develops early after injury around hemorrhagic lesions such as FLC and intracerebral hematoma; its nature is characteristically vasogenic, and the local increase of brain tissue bulk may compound the mass effect of the primitive lesion to cause brain distortion and midline shift; but it may also produce deleterious effects through compression of the surrounding brain parenchyma and increased intracranial pressure (Figure 11).

Focal hypodensity is another CT expression of cerebral edema in the absence of other detectable intracranial abnormalities. Although it may represent areas of true brain edema, it may alternatively represent ischemic lesions. The absence of brain shift and elevated ICP in many cases suggest that the latter possibility is a very real one. Ischemic brain damage following head injury is not a rarity (Miller et al., 1980).

Brain Swelling

Brain swelling is a condition not yet fully understood, characterized by the presence of increased intravascular blood within the brain. It is caused by

cerebral vasodilation secondary to the traumatic event, brain engorgement, and raised intracranial pressure (Langfitt, Weinstein, and Kassell, 1965; Langfitt, Tannanbaum, and Kassell, 1966; Obrist, Dolinskas, and Gennarelli, 1979).

Two main types of brain swelling can be distinguished by CT scanning, namely acute general brain swelling and hemispheric swelling. The picture of acute general brain swelling is characterized by squeezing or obliteration of the lateral ventricles, third ventricle and perimesencephalic cisterns (Zimmerman, Bilaniuk, Bruce, et al., 1978). This may occur as an isolated finding in the absence of detectable brain lesions (Figure 16A and B, later section) or in association with diffuse brain lesion or subarachnoid hemorrhage, seen for the most part as an area of increased density in the posterior portion of the interhemispheric fissure. Unlike cerebral edema, in which the extravascular brain water is increased and parenchymal density at CT scanning accordingly reduced, brain swelling occurs with normal or even slightly increased CT density; this is especially evident in children and in the acute phase, and reflects the presence of a greater quantity of blood in the cerebral vessels—blood having a higher density than normal brain parenchyma (Bruce et al., 1981).

Hemispheric swelling is a unilateral condition associated with the presence of an expanding lesion in the same hemisphere (Figure 14A) and a clinical

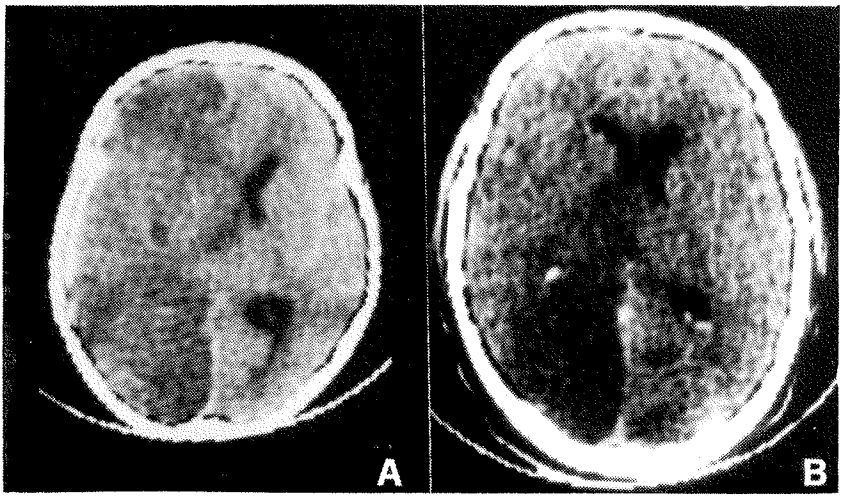

Figure 14: A: Left hemispheric swelling associated with acute subdural hematoma of the left convexity. Not evident contralateral temporal horn widening indicative of transtentorial uncal herniation; B: Left occipital and thalamic areas of ischemia secondary to occlusion of the posterior cerebral artery due to uncal herniation.

picture of deep coma. This type of brain swelling becomes particularly evident after surgical evacuation of a subdural hematoma—this being the most common associated lesion; its mass effect may be so marked as to make the brain protrude from the surgical opening in the bone. Pathogenesis can be traced back to initial ischemia due to a marked reduction of cerebral blood flow whenever the subdural hematoma is large enough to cause a sufficient increase of ICP (Yoshino et al., 1985). In such a situation, surgical emptying of the hematoma is followed by massive brain swelling. The CT picture characteristically reveals diffuse hypodensity of the involved hemisphere with various degrees of brain distortion and midline shift (Kobrine, Timmins, Rajoub, Rizzoli, and Davis, 1977; Lobato et al., 1983).

Cerebral Herniation

In severe head injury any expanding lesion, whether intracerebral or extracerebral, supratentorial or subtentorial, results in a progressive increase of intracranial pressure, whose effects are responsible for neurological deterioration and secondary brain damage. The mass effect exerted by an intracranial expanding lesion, indeed, produces a pressure cone that discharges itself on midline brain structures, thereby leading to a characteristic clinical picture with predominant evidence of brainstem compression. CT scanning reveals the increased ICP caused by the expanding lesion as a progressive compression of the ipsilateral ventricles with various degrees of brain structure distortion and herniation.

Most traumatic expanding lesions are supratentorial, and unless they are corrected surgically they eventually produce one of two characteristic CT scan pictures, namely subfalcine herniation and transtentorial uncal herniation.

Subfalcine herniation is more likely to occur as a consequence of a predominantly unilateral lesion that causes the gradual displacement (shift) of midline structures to the opposite side, ultimately to cause herniation of the gyruscinguli under the free edge of the falx cerebri. The corresponding CT picture consists of compression rotation of the frontal horn and displacement across the midline; effacement of the anterior portion of the suprasellar cistern; and stretching and bowing of the falx (Figure 5B, above). In the case illustrated, the same situation was expressed angiographically as a contralateral shift of the anterior cerebral artery with displacement of the pericallous artery under the falx. The important possible complication of subfalcine herniation are cingulate gyrus hypodensity and frontal hypodensity consistent with anterior cerebral artery infarction (Greenberg, 1984).

Transtentorial uncal herniation results from dislocation of the uncus and mesial part of the parahippocampal gyrus through the tentorial incisure on the side of the lesion. The CT scan picture shows progressive dilatation of the cisterna ambiens as a premonitory sign of impending transtentorial herniation;

contralateral brainstem displacement; distortion of the tentorial incisure; and contralateral temporal horn widening (Figure 14A) (Greenberg, 1984; Osborn, 1977; Stovring, 1977). Again in the case illustrated, angiography revealed medial stretching of the ipsilateral posterior cerebral artery, the vessel being compressed against the posterior clinoids. The complications of transtentorial hernia include occipital and thalamic low-density lesions representing infarction secondary to occlusion of the posterior cerebral artery (Figure 14B) and focal high-density lesions representing hemorrhages in the cerebral peduncle secondary to compression against the edge of the tentorium. These secondary brainstem lesions must be differentiated from shearing axonal injuries that may occur primarily in the brainstem in patients not harboring an expanding supratentorial lesion (Pasut et al., 1983a; Snoek et al., 1979; Tsai, Teal, Quinn et al., 1980).

Subtentorial traumatic lesions, such as extradural hematoma of the posterior fossa, intracerebellar contusion or hematoma, are comparatively rare (Tsai, Teal, Itabashi et al., 1980); yet their particular location and proximity to brainstem structures makes any increase of ICP that they may produce almost immediately life-threatening. The more severe cases of intracranial hypertension of the posterior fossa may even result in tonsillar herniation, or prolapse of the cerebellar tonsils through the foramen magnum, resulting in obliteration of the cisterna magna, compression of the medulla oblongata, and apnea. The CT picture is one of obstructive hydrocephalus (Greenberg 1984); at angiography, the typical sign was the sinking of the postero-inferior cerebellar artery beyond the foramen magnum.

Ischemic Lesions

Ischemic and anoxic lesions are extremely common in skull traumatology (Overgaard and Tweed, 1974), indeed representing the main cause of death in fatal cases (Adams, 1975) and also the factor most important in determining the severity of permanent neuropsychic sequels in survivors (Miller et al., 1978; Overgaard, Mosdal, and Tweed, 1981). Pathogenesis is manifold, encompassing as it does both extracranial causes like respiratory insufficiency and post-traumatic shock and intracranial mechanisms such as intracranial hypertension, cerebral edema, blood vessel compression due to brain distortion or herniation, and spasm or thrombosis of the carotid artery or its branches. Whenever ischemic pathology is suspected, one should not delay angiography to discover possible (albeit rare) post-traumatic thrombosis of the large arteries in the neck. The CT scan picture varies according to the stage of post-traumatic illness. Cerebral ischemia is seldom detected in CT scans during the acute phase; it begins to emerge a few or several hours after injury in the form of clear-cut hypodense areas in places involved in ischemic events. A very typical finding at this stage is the appearance of an area of occipital ischemia in

the territory supplied by the posterior cerebral artery as a result of distortion and compression of that artery due to descending transtentorial herniation. Less common is the onset of ischemia in the territory of the anterior cerebral artery from its compression due to subfalcine herniation of the cingulate gyrus. Focal areas of ischemia may be detected in the brainstem due to stretching of the perforating arteries, as well as multiple bilateral areas of diffuse ischemia in cases of severe intracranial hypertension.

In the most severe cases of prolonged and irreversible intracranial hypertension, the CT picture may amount to bilateral diffuse supratentorial hypodensity, paralleling the clinicl picture of "coma dépassé" and constituting the CT aspect of cerebral death from complete arrest of the blood supply to both hemispheres (Beltramello, Pasut, and Bricolo, 1982).

Intraventricular Hemorrhage

Intraventricular hemorrhage seldom occurs as an isolated manifestation; it is seen more often in a small proportion of patients who have suffered a severe head injury, along with intracerebral or extracerebral hematomas, diffuse white matter lesions (Figure 12B), or subarachnoid hemorrhage. CT scanning reveals intraventricular hemorrhage as a high-density area within the ventricular system, of which it often marks the whole profile. In time, the fluid component of hematoma settles in the dependent parts of the ventricles with the clotted part uppermost, sometimes to constitute a characteristic "liquid-level" image (French and Dublin, 1977; Merino de Villasante and Taveras, 1976; Zimmerman, Bilaniuk, Gennarelli, Bruce et al., 1978).

The blood collection turns hypodense in three or four days and is reabsorbed spontaneously in about one week—sometimes leaving behind a secondary hydrocephalus. Serial CT scans afford effective monitoring of the time course of intraventricular hemorrhage and early detection of a complicating hydrocephalus (New et al., 1976; Scott, New, Davis, and Schnur, 1974).

Subarachnoid Hemorrhage

Subarachnoid bleeding is a fairly common event in skull traumatology, usually due to traumatic laceration of leptomeningeal vessels—much more rarely of a major cerebral vessel or branch thereof. Subarachnoid bleeding is often associated with intracerebral or extracerebral hematoma, but may also occur in conjunction with pictures of brain swelling or diffuse white matter injury. The CT scan picture consists of a hyperdense image, more often located in the intercerebral fissure, sylvian fissure, cortical sulci, or basal cisterns (French and Dublin, 1977; Merino de Villasante and Taveras, 1976; Zimmerman, Bilaniuk, Gennarelli, Bruce et al., 1978; Zimmerman et al., 1982).

Complications of Head Injury

Cerebrospinal Fluid Leak

The leakage of cerebrospinal fluid (CSF) from the ear (otoliquorrhea) or nose (rhinoliquorrhea) occurs as a complication of fractures of the skull base with associated dirsuption of the dura mater creating a communication between the subarachnoid space and the exterior via the middle ear or the sphenoid, ethmoid, or frontal sinuses. Clinical diagnosis is usually easy; sometimes, however, the loss of CSF goes undetected and diagnosis suspicion arises later when recurrent episodes of meningitis supervene. Radiological diagnosis rests mainly on tomography, CT scanning and CT metrizamide cisternography. The CSF fistula is usually located in the cribriform plate; liquorrhea may stop of its own accord or require surgery (Drayer, Wilkins, Beohnke, Horton, and Rosenblum, 1977).

Pneumocephalus

Air may penetrate the skull cavity through a meningeal laceration and collect in the subdural or subarachnoid space, or even reach the ventricular system. If the brain parenchyma is lacerated along with the meninges, air may accumulate within the brain tissue itself. Both conventional roentgenography and CT scanning will produce the typical image of one or more gas bubbles located in the subarachnoid or subdural space, brain parenchyma, or cerebral ventricles (Osborn, Daines, Wing, and Anderson, 1978).

Intracranial Abscess

Pus collections may form in the extradural and subdural space (empyema) as well as within the brainparenchyma (abscess) as a complication of open craniocerebral wounds or fractures of the skull base communicating with the sinuses. If a brain abscess proper is to occur, it does so between one and two months after injury in most cases; at CT scanning it appears as a sharply outlined low-density area surrounded by perilesional edema and exerting a mass effect on adjacent brain structures (New, Davis, and Ballantine 1976). Empyema appears in CT scans as a hypodense or isodense crescentic area next to the inner table of the skull, causing ipsilateral ventricular compression and brainshift if large enough. Contrast infusion may reveal a rim of enhancement at the medial margin of the empyema. The brain beneath an epidural empyema is usually normal (Sharif and Ibrahim, 1982); beneath a subdural empyema, instead, brain tissue may appear hypodense due to edema secondary to ischemic and/or inflammatory vasospasm (Sadhu, Handel, Pinto and Glass, 1980).

Carotid-Cavernous Fistula

Traumatic laceration of the carotid artery may result in the formation of a fistula emptying into the cavernous sinus, usually after a fracture of the skull base or a penetrating orbitocranial wound. The condition is recognized clinically from orbital bruit, conjunctival chemosis and pulsatile exophthalmos, and confirmed angiographically by the presence of early opacification of the cavernous sinus and its draining veins. CT scanning will reveal dilatation of the superior ophthalmic vein associated with exopthalmos. The fistular passage may be obliterated through the vascular approach with detachable balloons by the technique of Serbinenko and Debrun [Figure 15] (Debrun et al., 1981; Serbinenko, 1979).

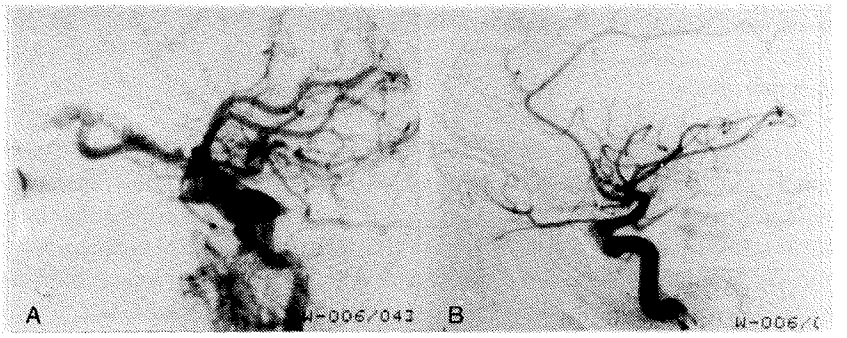

Figure 15: A: Post-traumatic carotidocavernous fistula. Note marked opacification of cavernous sinus and superior ophthalmic vein with poor injection of intracavernous carotid branches; B: after intravascular treatment with detachable Debrun ballon. Note effective closure of fistular passage with excellent opacification of intracranial carotid branches.

Sequels of Head Injury

Cerebral Atrophy

Generalized brain atrophy is a frequent sequel of severe head injury, reflecting diffuse neuronal degeneration for the most part secondary to brain tissue ischemia or anoxia or to a shearing injury of nerve fibers in the white matter (Adams, 1975). The CT picture reveals dilatation of the ventricular system, cerebral cisterns, cortical sulci, and intercerebral and sylvian fissures.

There is also a circumscribed form of brain atrophy representing the final result of hemorrhagic contusion or intracerebral hematoma; the extent of the permanent lesion is commensurate with the loss of neurons in the affected

area. CT scans will reveal focal areas of reduced density, with ipsilateral ventricular dilatation if the atrophic area is large enough (Gudeman et al., 1981; Merino de Villasante and Taveras, 1976; New et al., 1974.

Subdural Hygroma

This is a CSF collection in the subdural space, usually resulting from the normal reabsorption process of chronic subdural hematoma. The CT scan expression of subdural hygroma is a crescentic low-density image, usually in a frontal or frontoparietal position and sometimes bilateral. This type of lesion differs from chronic subdural hematoma by showing dilated rather than effaced cerebral sulci (Cornell, Chiu, and Christie, 1978).

Post-traumatic Hydrocephalus

Post-traumatic hydrocephalus is a fairly frequent sequel of severe head injury, occurring in two main types, namely ex-vacuo ventricular dilatation (associated with generalized cerebral atrophy secondary to neuronal loss) and obstructive hydrocephalus, due to a disturbance of CSF circulation secondary to subarachnoid bleeding in the basal cisterns. Periventricular low-density zones may be present in CT scans as evidence of CSF reabsorption through transependymal fissures (Gudeman et al., 1981).

A less common variant is normal-pressure hydrocephalus, or the presence of ventricular dilatation in the absence of increased CSF pressure, associated with a characteristic clinical picture of mental impairment, gait apraxia, and sphincteric incontinence. The CT appearance is one of ventricular dilatation with obliteration of cortical sulci (Beltramello, Pasut, Rosta et al., 1982).

Prognostic Value of CT Scan Findings

Making a prognostic judgment in the acute phase of severe head injury is far from an easy proposition; yet it is important in view of planning rational and adequate treatment in each case, and also to permit a-posteriori assessment of such treatment.

For many years the clinical elements of neurologic examination and the Glasgow Scale scores elicited on admission provided the basis for all prognostic studies seeking reliable correlations with the final outcome assessed six months after the injury (Becker et al., 1977; Bowers and Marshall, 1980; Braakman et al., 1980; Jennett and Bond, 1975; Jennett et al., 1979; Narayan et al., 1981; Young et al., 1981).

With the advent of CT scanning, and the attendant possibility of diagnosing intracranial lesions easily and accurately, it has become apparent that also the type of pathology that underlies the clinical situation has real prognostic value,

to the extent that the type of lesion revealed by CT scanning is important in determining outcome (Cooper et al., 1979: Gennarelli, Spielman et al., 1982; Lobato et al., 1983; Miller et al., 1981; Van Dongen, Braakman, and Gelpke, 1983). In that respect we should like to briefly discuss some data emerging from our own consecutive series of 385 cases of severe head injury (Table 1) assessed by systematic serial CT scanning (Bricolo et al., 1982; Pasut et al., 1981; Turazzi et al., 1987).

As expected, extradural hematoma turned out to be the type of lesion carrying the best prognosis. Still, while completely extracerebral and therefore theoretically benign, extradural hematoma in comatose patients continues to show relatively high percentages of unsatisfactory outcomes (10% severe disability or vegetative status, 17% dead), largely due to delayed surgery for time lost in transportation, and in some cases because of unwarranted observation periods in peripheral hospitals. In point of fact, extradural hematoma must be considered a surgical emergency since, unless treated in time, it can rapidly produce irreversible brain damage from intracranial hypertension and cerebral herniation. Thus, no efforts should be spared to insure timely diagnosis and round-the-clock access to emergency neurosurgery in such cases. It was indeed convincingly demonstrated that the earlier the surgery, the better the prognosis (Bricolo and Pasut, 1984). In our experience, at any rate, the overall balance sheet of extradural hematoma has shown sizable improvement in the CT era—also because the easy and repeatable diagnostic procedure makes it possible to perform the necessary surgery in a number of still conscious patients, sometimes in the absence of any neurological deterioration (Turazzi et al., 1987).

Conversely, subdural hematoma carries the highest percentages of unfavorable outcomes in all published series. Here too, however, evidence has been offered that final results are better, the earlier the surgical evacuation of subdural blood (Seelig et al., 1981). Unfortunately, other factors contribute to obscure the prognosis of subdural hematoma (Richards and Hoff, 1974): the patient's age, often advanced in such cases; the more severe neurological deterioration; and the nature of surgery itself. CT scanning has indeed demonstrated that pure acute subdural hematoma is a fairly rare occurrence; far more often, the condition is variously associated with hemorrhagic contusion or intracerebral hematoma, both tending to be more prominent than the subdural hematoma and causing altogether greater brain damage.

In our series the two last-named focal lesions (hemorrhagic contusion and intracerebral hematoma) have produced a total incidence of unfavorable outcomes (SD/VS and Dead) in the region of 60%, slightly higher (63%) in operated cases than in those managed nonsurgically (57%). In both subgroups, focal lesions have constituted the type of pathology in which the positive effects of CT scanning were more evident, both in regard to the choice of management and in terms of outcome. In surgical cases, CT scanning afforded

Table 1

Outcome in Surgical and Non-Surgical Lesions on CT Scan

CT Scan Picture	N° of patients	(%)	GR/MD (%)	SD/VS (%)	Dead (%)
Epidural hematoma	29	8	72	10	17
Subdural hematoma	36	9	19	8	72
Cerebral contusion/hem.	62	16	37	11	52
All surgical	127	33	40	10	50
Focal non operated	113	29	43	10	47
Shearing injury	22	6	14	27	59
Cerebral swelling	34	9	35	18	47
Normal CT scan	89	23	87	4	9
All non surgical	258	67	55	10	35
Total Cases	385	100	50	10	40

valid guidance to timely intervention—sometimes before the lesion had a chance to produce major neurological deterioration. In the nonsurgical group, serial CT scans afforded effective monitoring of the evolution of extant lesions, at the same time offering useful verification of the effects of medical and intensive-care treatment. On balance, the percentage of patients treated surgically was higher after the advent of CT than before, mainly because CT scanning brought more patients to surgery before their expanding lesions produced neurologic deficits in alert patients or caused final deterioration of brain functions. Repeated CT scans made after an initial examination that had shown no indication for immediate surgery produced definite surgical indications in fully 15% of these patients, all requiring delayed surgery for slowly-maturing intracranial lesions such as focal hemorrhagic contusion plus a lesser quota of new hematomas (Turazzi et al., 1987). Thus, the overall outcome improvement of surgical patients seen after the advent of CT encompasses not only patients undergoing surgery while comatose but also, or especially, those who were diagnosed and treated before the onset of major neurological deterioration thanks to the ease of execution of the new diagnostic procedure.

In most published reports the heading "diffuse lesions" covers all patients being comatose for more than six hours with a CT scan indicating no visible lesions—or at any rate no lesions constituting a space-taking mass (Gennarelli, Spielman et al., 1982). In our view, however, the term "diffuse lesions" is applied to pathologic events too diverse to justify grouping together, especially in terms of prognostic significance (Pasut et al., 1983). Accordingly, we found it

more expedient to set up three subgroups of more homogeneous lesions, which indeed showed better correlation with outcome: namely primitive diffuse lesions of the white matter, brain swelling, and the so-called "normal" CT scans.

The group of primitive diffuse lesions essentially includes the shearing injury of cerebral white matter—namely the widespread disruption of axonal nerve fibers that was formerly identified only as a neuropathological entity (Snoek et al., 1979). CT scanning afforded accurate description of such lesions, revealed their widely scattered location, and showed good correlation with outcome. In our series, indeed, the shearing injury of white matter carries the poorest prognosis of all, with fully 59% mortality and 27% severe residual disability. These patients are for the most part the victims of severe blunt head trauma, go into deep coma very shortly after injury, and often die in the first few days; those surviving the acute post-traumatic phase often go through a stage of prolonged coma; and if they emerge from that, they usually show severe permanent disability. The small percentge of positive results in this group represents patients emerging from coma within a few days after the injury—namely those in which the site and extent of white matter lesions does not preclude the recovery of neurologic function.

In the group of brain swelling we have assembled patients with no CT evidence of gross intra- or extraparenchymal lesions but showing constricted ventricles and no recognizable basal cisterns. Like the white matter shearing injury, brain swelling was detected before the advent of CT only as a neuropathological entity (Evans and Scheinker, 1945). CT scanning has clarified the nature of this phenomenon and made it possible to follow its time course—thanks also to the possibility of correlating CT pictures with measurements of physiologic variables such as cerebral blood flow and intracranial pressure (Langfitt et al., 1965). In this group also, the clinical picture is very severe and the incidence of unfavorable outcome is high (47% Dead and 18% SD/VS); CT scanning, however, revealed close correlation between the course of brain swelling and the patient's age. In children, indeed, brain swelling constitutes the most common finding in the early course of severe head injury (Zimmerman, Bilaniuk, Bruce et al., 1978) interpreted as reflecting the vascular response to the traumatic event or to secondary insults such as hypotension and hypercarbia, causing cerebral vasomotor paralysis and cerebrovascular congestion (Langfitt et al., 1966). Brain swelling in a child is an acute and diffuse phenomenon, entailing severe impariment of consciousness; yet if it is diagnosed in good time and treated aggressively it contributes little mortality and morbidity (Bruce et al., 1978). Brain swelling in the adult, however, is a different proposition, at once more rare and more severe, responding less readily to medical and intensive-care treatment, often affecting only one hemisphere and being associated with shearing white matter injury (sometimes with acute subdural hematoma), and carrying a higher risk of cerebral herniation.

Finally the subgroup of so-called "normal CT scans" comprises patients whose scans are noncontributory in regard to evident intra- or extraparenchymal lesions and show normal ventricles and basal cisterns. This subgroup accounts for about 23% of all cases of "diffuse lesions;" it is clinically the least severe at admission and rates best in terms of outcome (87% GR/MD). Still, 9% of these patients die within the first few days after injury despite their "normal" CT scans—probably because their lesions kill them before they become detectable. With serial CT scanning, indeed, we have found that a negative scan elicited at admission must not be taken at face value, since inapparent lesions may be present or developing, only to become detectable in the next few days (Pasut et al., 1981). In our series, fully 20% of patients whose scans were rated "normal" at admission developed new unilateral or bilateral lesions, sometimes requiring surgery. With serial CT scans made systematically in all cases, therefore, the number of cases with no evidence of traumatic pathology dropped to less than one-fifth of all comatose patients, probably representing the effect of the anoxic component of brain trauma and at any rate showing a tendentially good outcome. In a broader sense, serial CT scans made without waiting for evidence of neurological deterioration afford businesslike monitoring of the patient's course (Pasut et al., 1981) and show that intracranial traumatic pathology is a dynamic process more likely to change in time than one might imagine. In fact, fully 23% of patients in our series showed radical changes of their CT pictures in the first three days after the injury (Figure 16). It follows that a single CT scan made on admission is not enough to assess the incurred brain damage either reliably or completely, and that serial CT scans must be made systematically in all cases of severe head injury, lest one overlooks the subsequent development of an expanding intracranial contusion, hematoma, or secondary brain damage, and fails to implement the necessary therapeutic measures.

Cerebral Angiography

Lacking CT scan facilities, or having such equipment temporarily out of order, one can still rely on cerebral angiography for diagnosing an expanding intracranial lesion requiring surgery. True, cerebral angiography yields only indirect evidence of an expanding process by revealing the resulting displacement of blood vessels, and so a precise diagnosis is possible only in regard to extracerebral lesions; with an intracerebral lesion, angiography will not tell whether a local increase of brain volume is due to focal edema, hematoma, or hemorrhagic contusion.

The angiographic picture of extradural hematoma is a lens-shaped avascular area reflecting the detachment of the dura mater from the inner table of the skull and the inward displacement of vessels responsible for the bleeding, namely a meningeal artery, the sagittal sinus, or the torcular or transverse sinuses.

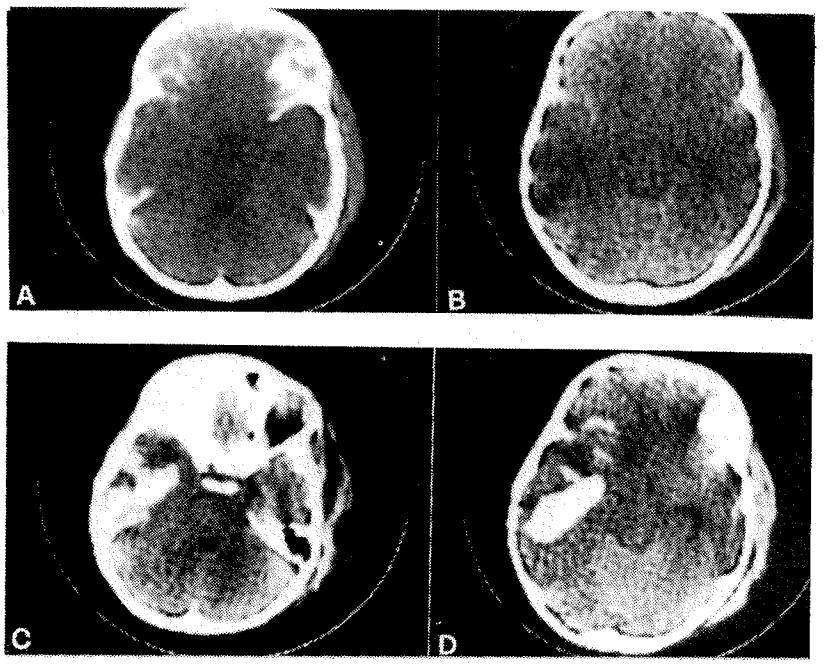

Figure 16: Severe closed head injury with brain swelling and nearly complete disappearance of ventricular images (A and B). Next day: evidence of left temporal intracerebral hematoma (C and D).

In acute subdural hematoma the blood collection finds room for diffusion over a larger surface of the brain; accordingly, the angiographic image is an avascular crescent exerting less of a mass effect on midline structures than does an extradural hematoma. In time, as the subdural hematoma undergoes chronic evolution, it becomes enclosed in a limiting membrane and may therefore show a lentiform outline at angiography, quite similar to that of an extradural hematoma. A clinical history of rapid evolution and the finding of a fracture line, for instance, along the course of a meningeal artery, suggest extradural hematoma.

Both hemorrhagic contusion and cerebral edema produce angiographic evidence of a space-taking mass with appropriate displacement of surrounding blood vessels. Intracerebral hematoma appears as an avascular area with a fairly prolonged mass effect, commensurate with the time needed for reabsorption of the hematoma and with the development of perilesional edema. The angiographic pictures of cerebral herniation, cerebral ischemia and carotidocavernous fistula were mentioned in the respective paragraphs. We must at this point stress that cerebral angiography retains its principal

indication in vascular traumatic pathology, signally obsructive lesions and the rare cases of traumatic aneurysm, which may escape detection by more modern methods including CT. The CT scan, indeed, can reveal only the sequels of a vascular trauma—namely parenchymal hyperdensity for hemorrhage and hypodensity for ischemia; beyond that, only angiography can tell the exact location and origin of the lesion.

In a recent study (Turazzi et al., 1987) we have compared the clinical evolution and outcome of two series of head injury cases, one managed with angiography and the other with CT scanning, trying to discover the possible influence of this shift in diagnostic procedures on the management of such patients. The two series compare very closely in many respects, signally in the incidence of surgical cases; but the overall outcome was better in the CT scan series both in surgical and in nonsurgical cases. This is understandable in view of the following considerations:

(a) At admission, cerebral angiography and CT scanning were equally effective in detecting lesions of surgical domain; later in the course of illness, however, CT scanning proved far more effective in detecting changes, with fully 15% of the patients being referred for surgery in the light of repeated CT scans as opposed to only 4% undergoing surgery on the indications of repeated angiography.

(b) Systematic CT scans made it possible to operate on many patients with expanding brain lesions before they went into coma, and saved a good deal of postoperative deterioration. Because of its invasive nature and attendant risk to the patient, on the other hand, cerebral angiography was performed only after careful assessment of coma and associated focal neurological signs; and it was repeated only if the neurological situation deteriorated or suggested the presence of an expanding intracranial lesion (Turazzi, Bricolo, and Pasut 1984).

(c) The greater ease of execution of CT scanning compared to angiography resulted in shorter intervals from trauma to surgery in the second series.

(d) The accurate description of the nature, location and evolution of brain lesions, afforded by CT scanning, made for more rational and timely application of therapeutic measures in the second series than in the first.

Other Diagnostic Procedure

The diagnostic exploration of comatose patients following severe head injury usually includes the measurement of intracranial pressure, EEG and averaged evoked potentials recording and, if feasible, the measurement of cerebral blood flow.

The more widely used technique for measuring intracranial pressure consists of placing a catheter in the lateral ventricle, to afford accurate and direct

pressure readings as well as immediate correction of hypertensive crises by the direct subtraction of CSF. It was indeed demonstrated that a high intracranial pressure influences the course of severe head injury adversely, whereas its control is conducive to a better outcome (Marshall et al., 1979; Miller et al., 1977).

The EEG tracing (Bricolo et al., 1978) and visual, somatosensory and auditory evoked responses yield information about the function of the central nervous system of the comatose patients that neither clinical examination nor CT scanning can reveal. Also, if such evoked responses are recorded serially in multimodal fashion since the first day of trauma, they can be shown to predict final outcome with far greater accuracy than all neurological and neuroradiological indicants taken together (Greenberg, Newlon, Hyatt, Narayan, and Becker,1981; Narayan et al., 1981).

Measurements of the cerebral blood flow (CBF) have come into decisive importance since accurate neuropathological studies have demonstrated that ischemic brain damage is prominent in 90% of patients dying after severe head injury (Graham and Adams, 1971). More recent studies revealed that post-traumatic coma is associated with marked alterations of the CBF ranging from severe reduction to persistent hyper-perfusion, and only the accurate recognition and target treatment of these diverse (sometimes opposite) hemodynamic situations can curb the emergence of secondary brain insults responsible for death or severe permanent disability (Obrist et al., 1979; Overgaard et al., 1974; Overgaard et al., 1981).

Among the mechanisms of brain damage incurred by patients after severe head injury, diffuse axonal shearing lesions, ischemia, and brain swelling result from basic alterations that escape detection by any of the traditional diagnostic methods. The CT scan is often noncontributory in such cases, and the condition is diagnosed only tentatively when diffuse brain damage is suspected merely on the strength of clinically severe coma. A more accurate diagnosis of diffuse brain damage may be afforded by last-generation CT machinery, by CBF studies, and above all by new technologies such as Positron Emission Tomography (PET) and Nuclear Magnetic Resonance (NMR).

PET provides valuable information on cerebral metabolism by measuring the rate of glucose consumption in the brain; and NMR clearly discriminates gray from white matter and their respective water contents, as illustrated in detail by other contributors.

From comparative studies published to date it appears that both CT scanning and NMR exploration are effective in revealing acute hemorrhagic lesions in patients with severe head injury. NMR, however, is far superior to CT in detecting shearing injury, subdural hematoma, and hemorrhagic and nonhemorrhagic contusion, as well as in separating them from cerebral edema, both in the acute and in the subacute stages of head injury; and only NMR proved capable of demonstrating chronic hematomas or small hypothalamic or

brainstem infarcts not detectable by CT scanning. And last, NMR has produced a far more accurate definition of cerebral atrophy than could be obtained with CT scans.

Thus, in summation, CT scanning offers the great advantages of rapid execution and unlimited repeatability, and therefore remains the method of first choice for deciding between surgery and medical management and for monitoring head injury patients in the acute stage. NMR exploration with currently available machinery requires materially longer scan times; accordingly, its greatest value is not so much in acute situations as in the detailed study of subacute or chronic condition, particularly in terms of making a correct prognosis (Snow, Zimmerman, Gandy, and Deck, 1986; Zimmerman, Bilaniuk, Hackney, Goldberg, and Grossman, 1986).

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