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# CT Changes in Dementing Diseases: A Review

Marjorie LeMay<sup>1,2</sup>

**The present review describes the primary CT features of a variety of dementing disorders. An understanding of the differential neuropathologic and CT changes occurring in dementing illnesses can greatly increase the diagnostic utility of CT. CT is valuable not only in diagnosing space-occupying lesions but also in recognizing numerous degenerative brain diseases.**

There are a number of diseases that can cause dementia. Some of these include presenile and senile illnesses of the Alzheimer's type, Pick's disease, Creutzfeldt-Jakob disease, multiple infarcts, dementia pugilistica, normal pressure hydrocephalus, and a variety of metabolic encephalopathies. While numerous studies have attempted to differentiate the CT scans of dementia patients from those of age-matched controls, few have compared and contrasted the CT scans of patients with a variety of dementing syndromes [1-4]. The present review summarizes CT features that typify a number of commonly encountered dementing disorders.

## Dementing Disorders

### *Alzheimer's Type Dementia*

Alzheimer's type dementia (ATD) is a primary degenerative brain disease usually occurring in middle and late life that is diagnosed by clinicopathologic findings. Microscopic examination of the aging brain shows a decreased number of neurons, amyloid accumulation, senile plaques, neurofibrillary tangles, and granulovacuolar degeneration. These changes are greatly exaggerated in number and extent of distribution in patients with ATD [5-8]. Neuropathologic changes in patients with dementia are marked in the region of the angular gyrus and the inferior and middle temporal gyri; they are particularly striking in the amygdala, hippocampus, and the parahippocampal gyri, which lie in the medial temporal region [9-11] (Fig. 1).

Previously, patients who developed Alzheimer's type dementia at age 65 and older were designated as having "senile dementia" and, then, "senile dementia of the Alzheimer's type." Those who developed symptoms earlier were said to have "Alzheimer's disease." At present, the illness in both age groups is often referred to as Alzheimer's disease, although some clinical differences have been cited. One report noted that patients with dementia beginning before age 65 have a stronger family history of dementia, a relatively shorter life span, more language difficulties, and more left handedness [12]. Neuropathologic changes have been reported to be more marked in patients with an early onset of dementia [9, 11, 13-16].

A close examination of the CT scans of patients with ATD show that a number of characteristic changes can be seen in the temporal regions that appear to correlate with the above neuropathologic findings. As shown in Figure 2, atrophy is striking in the anterior and medial portions of the temporal lobes. This is reflected by widening of the suprasellar cistern, the cisterns between the midbrain and temporal lobes, and, particularly, the temporal horns of the lateral ventricles. The widening of the temporal horns occurs not only at their tips, which lie between the amygdala and hippocampus, but also extends posteriorly. This latter finding may be due to atrophic changes in the white matter of the temporal lobes as well as to cellular changes in the hippocampus. It seems likely that the widening of the cisterns at

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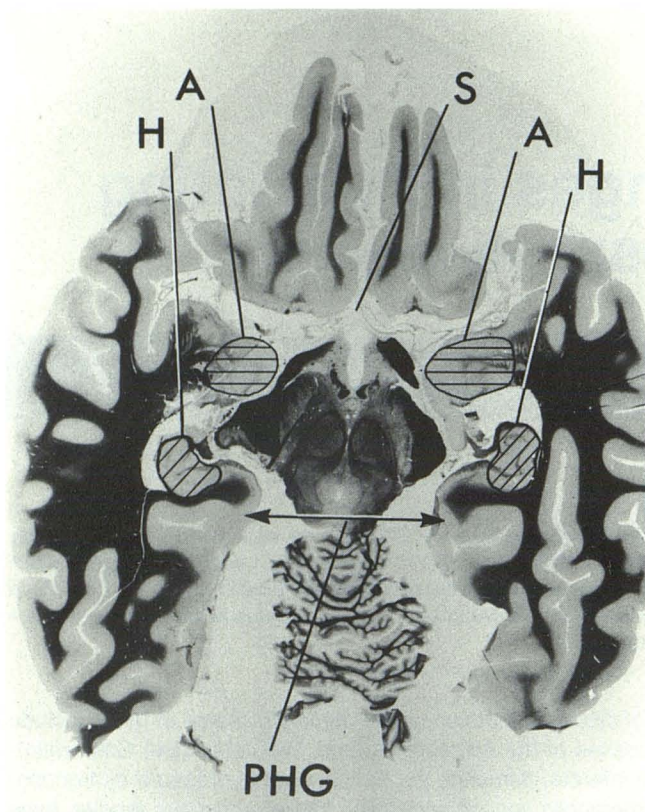


Fig. 1.—Section of brain through temporal lobes showing suprasellar cisterns (S), amygdala (A), and hippocampus (H). Tips of temporal horns of lateral ventricles lie between A and H. Arrows overlying midbrain point to parahippocampal gyri (PHG). (From the Yakovlev brain collection, AFIP, Washington, D.C. Courtesy T. Kemper and M. Haleem.)

the lateral margins of the midbrain is largely the result of tissue loss in the adjacent temporal lobes rather than in the midbrain. Most CT scans of patients with ATD, besides showing atrophy in the medial temporal regions, also show increased widening in the anterior portions of the sylvian fissures. Similar changes may be seen in the aging brain, but they are less striking, and the temporal horns are not often appreciably widened in nondemented elderly individuals [17, 18]. Fewer patients with ATD show lesser alterations in the medial temporal lobes but marked atrophy of the gyri in the temporal lobes and along the insula, widening both the sulci and sylvian fissures in these regions (Fig. 3).

The majority of radiographic studies, using both pneumoencephalography (PEG) [19–22] and CT [1, 2, 4, 23–32] have found the third ventricle and bodies of the lateral ventricles, and often the superficial sulci, to be statistically larger in patients with ATD, but there is a great overlap between the studies of patients with dementia and those of mentally normal controls. A progressive widening of the third ventricle is usual with aging [17], but PEG [20] and some CT studies [26, 27, 29] have found widening of the third ventricle to be significantly greater than widening of the bodies of the lateral ventricles in patients with ATD compared to CT studies of age-matched controls. Along with the described changes in the temporal lobes, prominent widening of the third ventricle and global atrophic changes help to strengthen the diagnosis of ATD by CT.

#### Pick's Disease

Pick's disease is another form of progressive dementia. It is not a common disease but is easily mistaken clinically for ATD. Neuropath-

ologic changes are less generalized, however, and Pick's disease is sometimes called "lobar atrophy" or "progressive circumscribed cerebral atrophy." Microscopically, the brain shows atrophy and sclerosis, especially in the frontal and temporal lobes; however, there is often sparing of the posterior two-thirds of the superior temporal gyrus [33, 34], a gyrus that usually shows atrophy in ATD. Atrophy is often asymmetrically distributed in Pick's disease, and more frequently involves the left hemisphere than the right [34, 35]. There may also be severe atrophy of the basal ganglia [34, 35]. Microscopically, some neurons show local areas of cytoplasmic swelling, which are called Pick's bodies. Clinically, abnormal behavior and difficulty with language are more common in these patients than memory disturbances, which are more likely to be an early manifestation of ATD.

The diagnosis of Pick's disease can be suggested by CT when atrophy is localized and/or asymmetrical in the hemispheres [34, 36]. Figure 4A shows a CT scan through the temporal regions of a 52-year-old man who had presented 1 year earlier with hypomanic and depressive symptoms. At the time of the CT study, he showed a progressive decline in memory and an inability to learn new things. He died 5 years later, and neuropathologic examination showed moderate widening of the third and lateral ventricles. The temporal lobes appeared "shriveled up" bilaterally. The amygdalae could not be identified, but Pick's cells were seen in the hippocampi. No senile plaques or neurofibrillary tangles were seen. Figures 4B and 4C are scans of a 70-year-old woman who presented with paranoid ideation and a 4-year history of progressive dementia. Her EEG showed minor asymmetry in activity organization, with that on the left less well organized than that on the right. Clinically, she was thought to have Pick's disease and she was sent to a nursing home. No autopsy was performed when she died.

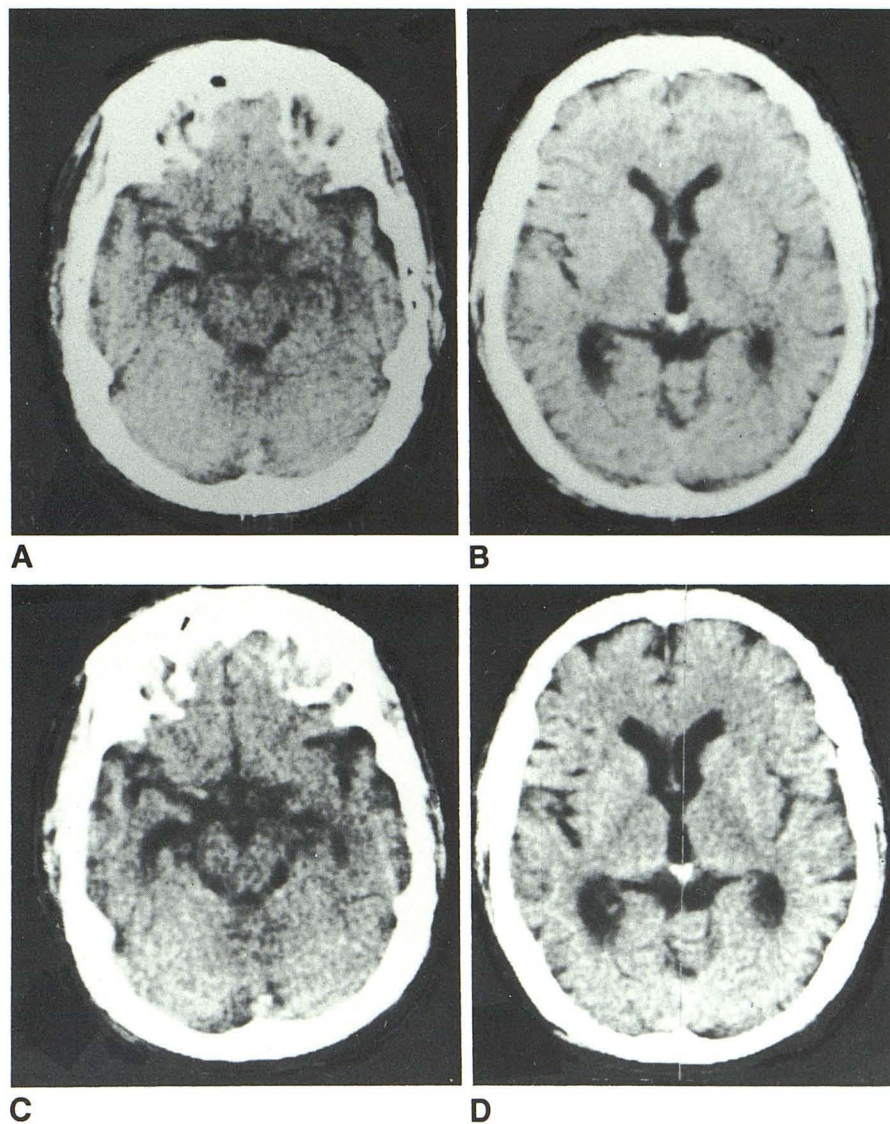
#### Creutzfeldt-Jakob Disease

A more common dementing illness than that described by Pick is Creutzfeldt-Jakob disease, a form of subacute spongiform encephalopathy. It is usually associated with an incubation period of several years and has been attributed to a "slow virus" or what is sometimes referred to as "a slow infectious pathogen, or prion." Patients typically develop the disease between the ages of 40 and 60, but some individuals have developed symptoms in their late teens and early twenties [37, 38]. The average course of the disease after symptoms develop is less than 1 year [38], although some patients have lived longer—one as long as 16 years [39]. Behavioral changes and visual disturbances, such as cortical blindness and hemianopsia, are often early symptoms of the disease. These are usually followed, in weeks or months, by progressive dementia, loss of motor control, and gait disturbances [40]. EEG changes with periodic sharp wave complexes appear during the course of the disease and are said to be characteristic of a viral infection [37, 38, 41, 42]. A familial history of the disease has been reported in at least 15% of the cases of Creutzfeldt-Jakob disease, possibly due to a genetically inherited susceptibility [38]. The disease is not rare. Person-to-person transmission has been reported from neurosurgical operations in which silver electrodes were used that had previously been implanted in a patient with Creutzfeldt-Jakob disease [43] and in patients who were recipients of human pituitary growth hormone [44, 45]. Transmission of the disease via a corneal transplant has also been reported [46], and some scientists speculate that it can be transmitted via absorbable animal-tissue sutures used in head trauma and/or surgery [47].

Histologic changes show widespread loss of neurons and astrogliosis in the cerebral cortex and in the central gray matter and cerebellum. The brainstem also is often affected [38]. The presence of spongiform changes in these areas is thought to suggest active disease, and when present in this distribution is strong evidence of



Fig. 2.—Alzheimer's disease. Scans of 58-year-old man who presented with memory deficit, spatial difficulties, and problems with concentration. His mother was said to have had senile dementia. Scans from Feb. 1983 (A and B) show slight widening of CSF spaces, involving particularly the cisterns above the sella and about the midbrain and slight widening of third ventricle. A shows significant widening of temporal horns. Bodies of lateral ventricles showed slight widening on slice above B. C and D, Scans taken 16 months later (June 1984), when symptoms had progressed, also show progression of atrophic changes. The extent of visualization of temporal horns depends particularly on angulation of slice. If scan had been more nearly horizontal, it is probable that one would see widening of temporal horns extending more posteriorly.



Creutzfeldt-Jakob disease [48]. White-matter degeneration is seen in some cases and may be more frequent in those individuals who have a longer course of disease [38, 49]. Extensive white-matter degeneration has been reported in some patients, and this may be caused by a different strain of Creutzfeldt-Jakob virus [50, 51]. Some cases of rapidly progressive Alzheimer's disease develop myoclonus and may be mistaken for Creutzfeldt-Jakob disease. Occasionally, ATD and Creutzfeldt-Jakob disease occur in the same families [38].

The CT findings in Creutzfeldt-Jakob disease vary according to the brain site most affected and to the time at which the scan is performed. Progressively atrophic changes are seen on CT [42, 52–54], with gradual widening of the ventricles and superficial sulci (Fig. 5). The temporal lobes may also be involved but not to the extent that is seen in patients with ATD. If the white matter is involved, this may also show on the CT study [42].

#### *Angiopathic Dementia*

Dementia may occur in patients who have suffered multiple cerebral infarcts. These infarcts often follow arteriosclerotic changes in

the neck vessels, with ensuing thromboemboli or occlusion [55]. There is usually a neurologic history of focal deficits, and the progressive decline of mental function is usually more punctuated than in individuals with ATD. The presence of dementia depends on the location and the amount of brain loss. The patients are commonly hypertensive and often diabetic. CT studies may show local areas of low density and asymmetry of the lateral ventricles and sylvian fissure enlargement. It is difficult, however, to diagnose multiple infarct dementia radiologically because many patients thought to have multiple infarct dementia by clinical evaluation do not show infarcts by CT or avascular areas by angiography [56]; and many patients with primary degenerative brain disease often show local low-density areas consistent with infarction [56–58].

Subcortical arteriosclerotic encephalopathy is a form of multiple infarct dementia. This condition was described by Binswanger in the late 19th century and is frequently referred to as Binswanger's disease. It is a type of arteriosclerosis that particularly affects the vessels to the white matter about the ventricles and to the basal ganglia. Histologic changes show diffuse and/or localized demyelination in the white matter about the lateral ventricles. Lacunar infarcts



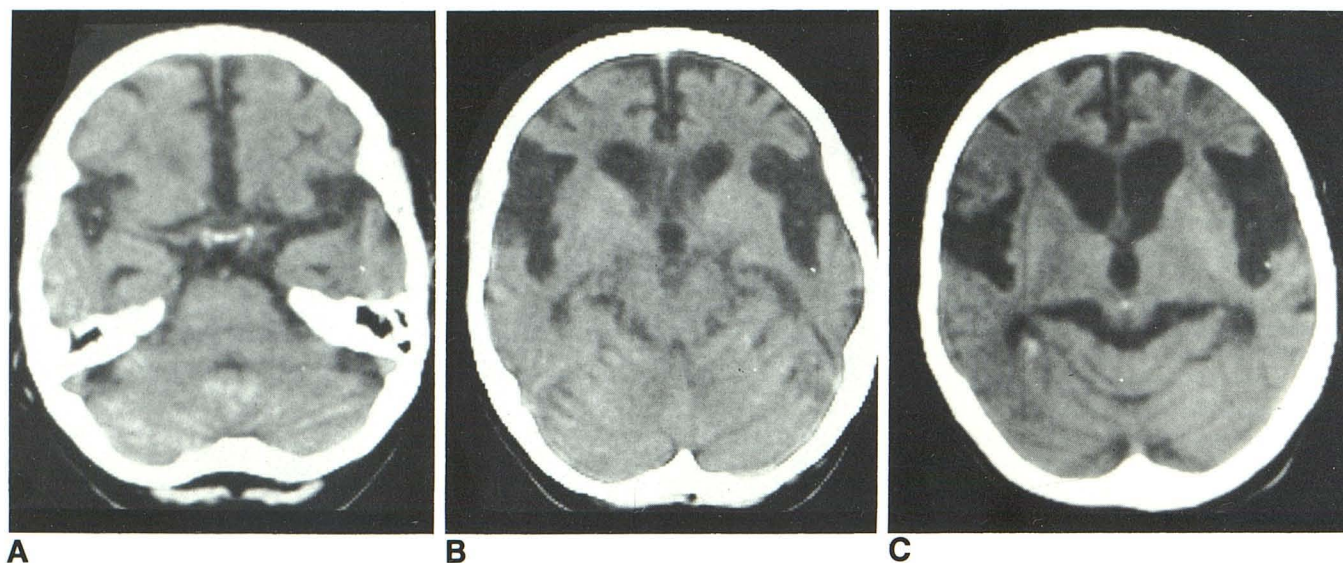


Fig. 3.—Alzheimer's disease. A, B, and C, Scans of 74-year-old woman with 4–5 year history of progressive memory loss that had progressed markedly during past 6–12 months. Four of five siblings also had severe memory loss in their late 60s, which prevented them from functioning normally. A scan was

not taken through suprasellar cistern, but scans A and B do show widening of temporal horns. The most striking atrophic changes are widening of sulci of the temporal opercula and of anterior ends of sylvian fissures.

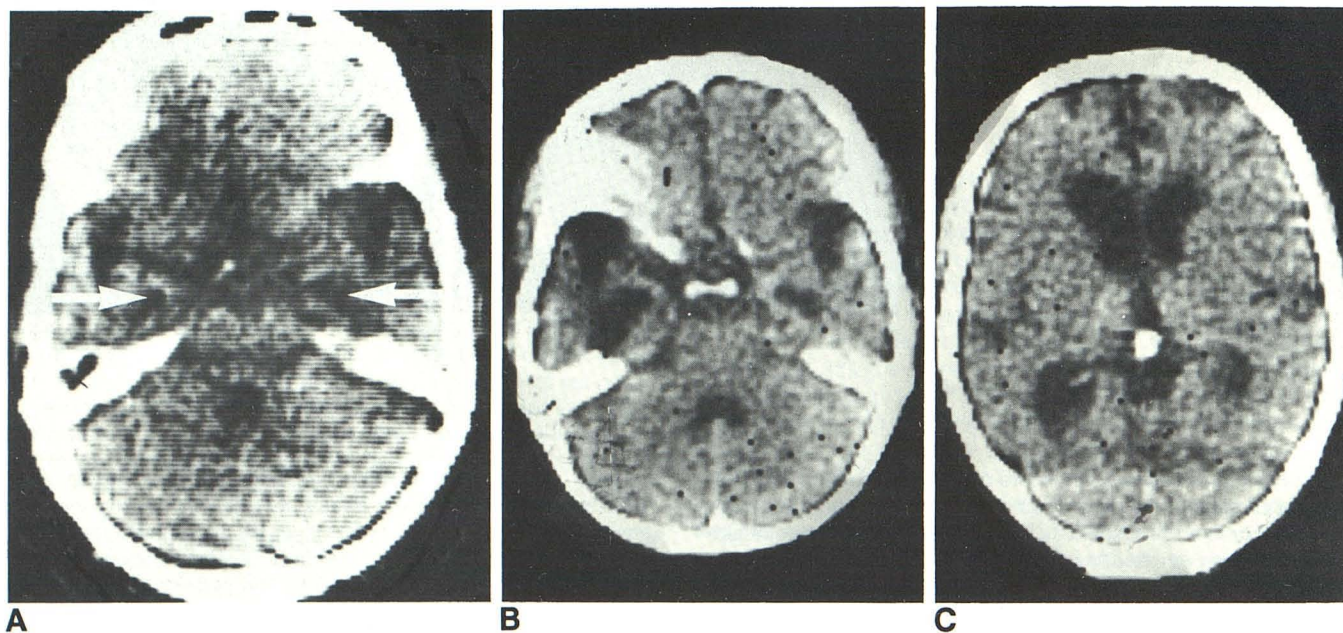


Fig. 4.—Pick's disease. A, CT scan of 52-year-old man with progressive memory impairment for 3–4 years that prevented him from functioning normally. He died 5 years later, and neuropathologic changes showed findings of Pick's disease. At time of CT, the bodies of the lateral ventricles were not large but the anterior ends of temporal horns showed asymmetrical widening. (Courtesy,

of R. K. Rosales, Boston University Medical School and Boston VA Hospital, and E. P. Richardson, Jr., Harvard Medical School and Massachusetts General Hospital.) B and C, Scans of 70-year-old woman who was thought to have Pick's disease. Scans show asymmetrical ventricular atrophy, particularly striking in temporal region, suggestive of Pick's disease.

may also be seen in the white matter and basal ganglia [59–61]. These patients are frequently hypertensive and commonly develop slowly progressive dementia, pseudobulbar palsy, and signs resembling parkinsonism. CT changes usually show a patchy decrease in density of the white matter about the lateral ventricles and widening

of the third and lateral ventricles and superficial sulci [60, 62, 63]. Local small areas of low density suggesting lacunar infarcts may also be seen. Figure 6 shows CT scans consistent with Binswanger's disease, from a study of a hypertensive 71-year-old man with dementia. The patient was in cardiac failure and had diffuse arteriosclerotic



Fig. 5.—Creutzfeldt-Jakob disease. **A**, Scan of 43-year-old man who had a herpes zoster infection on his thorax 1 year previously, which cleared. He developed vertigo 7 months before hospital admission. On admission to hospital, he was dysarthric, showed diplopia and horizontal nystagmus, and had a tremor. He soon became confused and developed jerking movements. **B**, Scan 3 months later. Patient was confused and incontinent. There was progressive widening of third and lateral ventricles compared with the study 3 months earlier. He also had a cyst of the septum pellucidum.

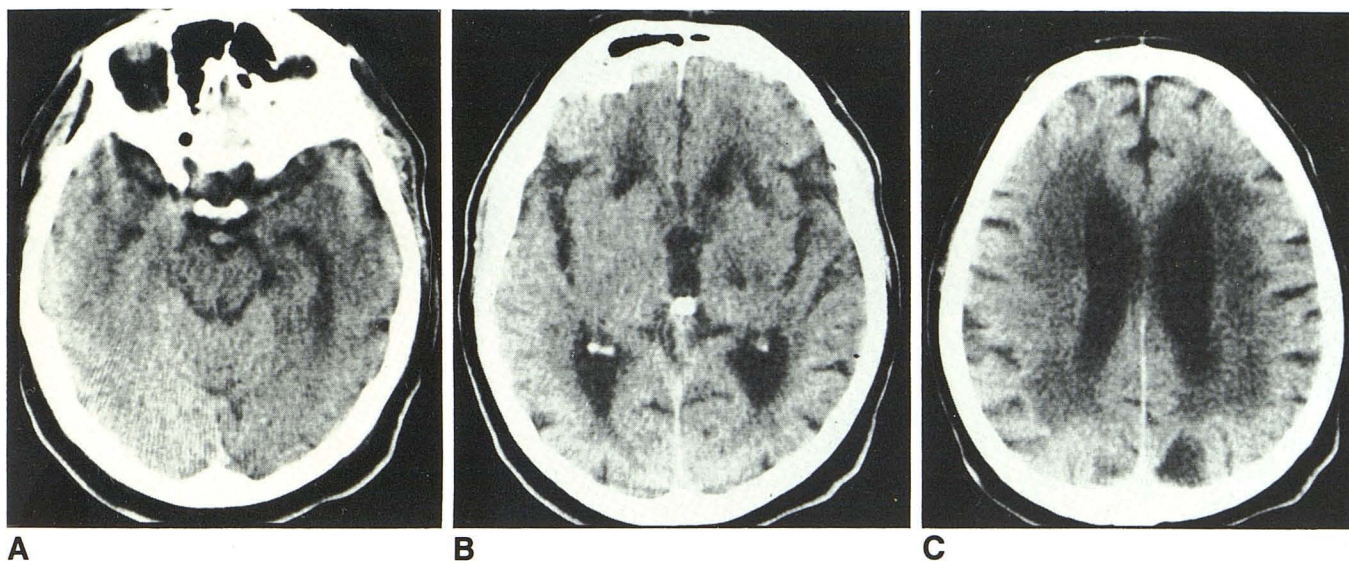
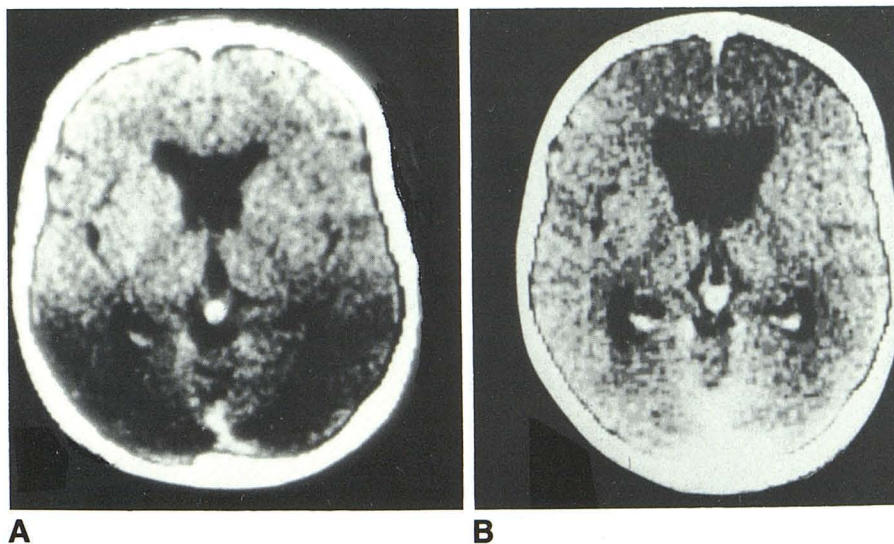


Fig. 6.—Binswanger's disease. **A**, **B**, and **C**, Scans of 71-year-old man with hypertension and 5-month history of dementia. There is widening of third and lateral ventricles, including some asymmetrical enlargement of temporal horns.

There is suggestive evidence of small infarct in left thalamus. Suprasellar cistern was not significantly widened. The most striking changes are in white matter, about the bodies of the lateral ventricles. (Courtesy of M. Savoiardo, Milan.)

disease. Figure 7 shows head scans of a man who died at the age of 67 and whose CT and pathologic findings were consistent with global and Binswanger's type of multiple infarct dementia. There was a history of mild hypertension, multiple episodes of difficulty of speech and gait, and progressive dementia over a period of nearly 20 years. Autopsy showed evidence of large infarcts in both cerebellar hemispheres and the right occipital lobe. Multiple lacunar and microscopic infarcts were present in the basal ganglia bilaterally, both hippocampi, throughout the cortex and both cerebellar hemispheres, and in the brainstem. There was also extensive disease in the white matter, particularly in the periventricular regions, consistent with Binswanger's disease. CT scans were repeated at intervals over a 10-year period; these clearly showed the larger infarcts that occurred in the cerebellar hemispheres and the right occipital lobe and ill-defined densities about the margins of the lateral ventricles. Suggestions of

small infarcts were reported in the pons and corona radiata.

Another cause of vascular dementia is congophilic or primary amyloid angiopathy. This is a disease in which amyloid deposits develop in the walls of small vessels and may cause occlusion or necrosis of the vessels, in turn leading to hemorrhage [64–69]. Plaques occur similar to those found in multiple sclerosis (MS) and it has even been suggested that the disease may be a variant of MS [64]. Plaques are common in the periventricular white matter and have also been reported in the optic nerves, brainstem, and spinal cord. Some patients have shown evidence of systemic amyloidosis, and hemorrhage can occur in other organs, such as the gastrointestinal tract [64–65], as well as in the brain. Familial cases of amyloid angiopathy have been reported. Hemorrhages can occur in all the lobes of the brain, most being superficial or subcortical and sometimes extending into the subarachnoid space. These hemorrhages



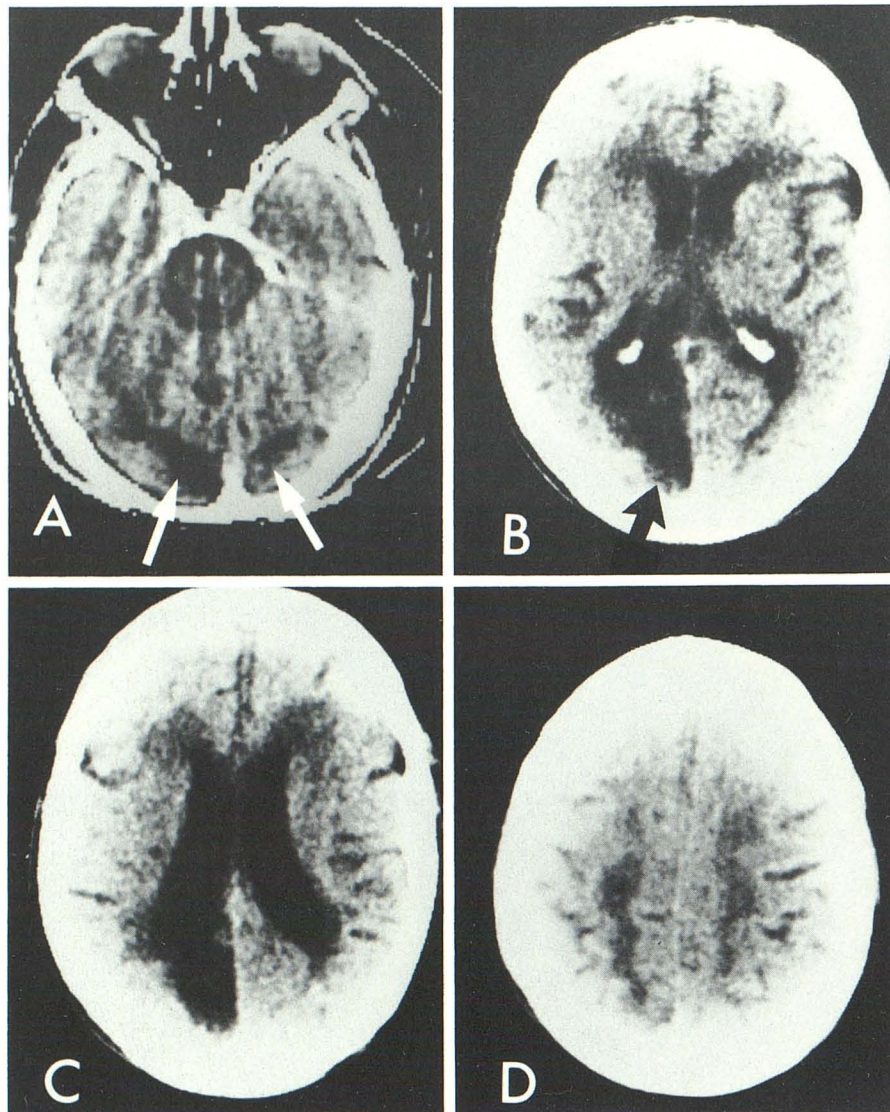


Fig. 7.—Multiple infarct dementia and Binswanger's disease. Scans of 67-year-old man with repeated syncopal episodes and progressive dementia over a 20-year period. Tissue loss due to infarcts is clearly seen in cerebellar hemispheres (A) and in right posterior occipital region adjacent to enlarged occipital horn of lateral ventricle (B). C and D, Low densities are present in white matter about and above bodies of lateral ventricles secondary to microangiopathic encephalopathy. Temporal horns were not significantly widened.

differ from those associated with arteriosclerotic hypertensive disease, which more classically involves the basal ganglia, pons, and cerebellum [68]. The patients occasionally present with slowly progressive dementia or, more frequently, with an intracerebral hemorrhage. The changes on CT will of course depend on the form of the disease [70]. Scans taken after a large hemorrhage that invades the ventricles show the hemorrhage to be more often above the basal ganglia and at times to extend over the surface of the brain [68] (Fig. 8). Patients presenting with dementia usually have demonstrated global atrophic changes and show the neuropathologic findings of ATD. If the temporal lobes are involved, the temporal horns are likely to be enlarged. Figure 9 shows CT scans of a 76-year-old man with progressive failure of intellect over a 4-year period. He was thought to have Alzheimer's disease and was placed in a nursing home. When he died, the neuropathologic findings were those of extensive amyloid angiopathy, and there were insufficient neuritic plaques or neurofibrillary tangles to classify the patient as having ATD.

#### *Hydrocephalic Dementia*

Hydrostatic hydrocephalus may also be responsible for dementia, and patients' symptoms often clear after ventricular shunting. Spinal

cord tumors occasionally prove to be the cause of hydrostatic hydrocephalus and dementia, both of which clear when the tumor is removed [71].

Some patients with hydrocephalus and dementia do not have elevated spinal fluid pressures and are classified as normal pressure hydrocephalus. Many of these treatable patients have obstruction of the CSF pathway outside the ventricles. Normal pressure hydrocephalus may result from many causes, but the most common cause is probably previous hemorrhage or meningeal infection resulting in interference with absorption of the CSF. Normal pressure hydrocephalus may also be caused by long-standing obstructive hydrocephalus, such as that resulting from aqueduct stenosis.

Apart from dementia, the most common symptoms in patients with normal pressure hydrocephalus are gait disturbances and incontinence. Difficulty in walking appears to be particularly frequent, and decreased cognitive functioning may at times be mild [72]. The diagnosis is often difficult to make. Some patients found to have normal pressure hydrocephalus were originally diagnosed as having had lacunar strokes [73] or progressive arthritis and aging because of their increase in walking difficulty and decrease in cognitive function [74]. Normal pressure hydrocephalus has at times been found to be responsible for parkinsonian dementia [75–78].



Fig. 8.—Amyloid angiopathy with hemorrhage. Scans of 62-year-old, nonhypertensive man who had been in good health but suddenly developed a hemorrhage in right temporal lobe (A). Angiogram was normal except for showing a mass effect. The clot was evacuated, and patient recovered but developed mild hypertension. One year later a hemorrhage occurred in right frontal lobe, extending into ventricle (B). Angiograms were again normal except for a mass effect. The clot was evacuated and a biopsy of adjacent cerebral tissue and leptomeninges showed intramural amyloid deposits consistent with amyloid angiopathy. Patient developed a superficial hemorrhage in left frontal lobe 2 weeks later.

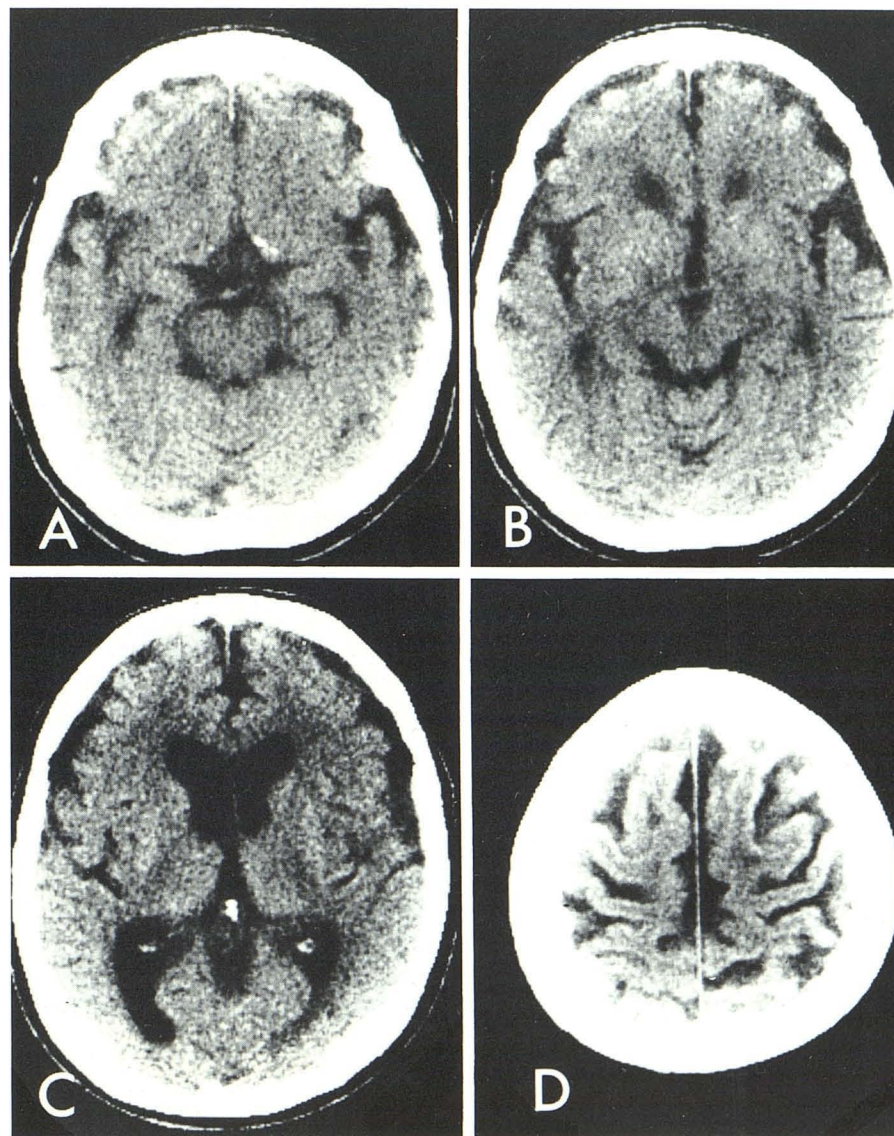
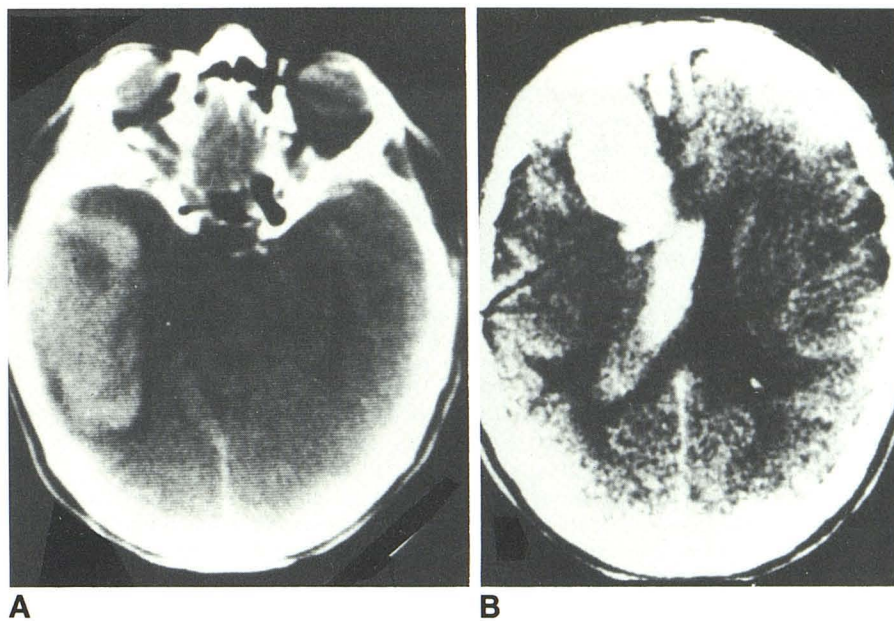


Fig. 9.—Amyloid angiopathy without hemorrhage. A–D, Scans of 76-year-old man with progressive dementia over a 4-year period. He was thought to have Alzheimer's disease and placed in a nursing home. He died 3 months after the CT study. Neuropathologic studies showed evidence of extensive amyloid angiopathy, but there were insufficient neuritic plaques or neurofibrillary tangles to make a diagnosis of Alzheimer's disease. (Courtesy of Eric D. Caine, University of Rochester Medical School, Rochester, NY.)



Cisternography shows abnormal CSF flow, but this may also be found in patients with degenerative brain disease [79, 80]. Removal of a small amount of CSF by spinal puncture may improve the symptoms of patients with normal pressure hydrocephalus; when this occurs, it is a good sign that the patient may be helped by shunting fluid from the ventricles [72].

Visualization of the anterior ends of the temporal horn is usually the earliest sign of hydrostatic hydrocephalus, unless it is caused by a centrally located lesion, such as an expanding third ventricle tumor, which may compress the tips of the temporal horns and prevent their enlargement. The temporal horn tips are usually rounder and wider in patients with hydrostatic hydrocephalus than in patients with primary degenerative disease of the brain, but it is sometimes difficult to separate the two disease states by CT. At times, a low density is seen about the margins of the lateral ventricles, particularly the frontal horns, which is thought to be caused mainly by leakage of CSF through the ventricular walls. The third and lateral ventricles are usually larger in patients with normal pressure hydrocephalus than in patients with primary degenerative dementia. On CT studies, the frontal horn ratio (i.e., the width of the frontal horns divided by the internal diameter of the vault at the same level) is often over 0.5 in patients with normal pressure hydrocephalus, which is much less common in patients with ATD [81]. The superficial sulci over the hemispheres and the cisterns about the midbrain and in the suprasellar region tend to be less prominent in patients with normal pressure hydrocephalus than in patients with ATD. Figure 10 is a scan of a patient who presented with difficulty walking and diminished cognitive function who improved with shunting of CSF from the ventricles.

#### Posttraumatic Dementia

Ventricular enlargement and widening of superficial sulci are often seen by CT after closed head injuries [82–84], but severe reduction of intellectual function is not a common sequel. ATD has been reported after head injuries, usually, but not always, in elderly individuals [84]. Neuropsychiatric symptoms and evidence of cerebral atrophy by CT have been reported in young torture victims [85].

Dementia pugilistica, a dementia occurring in boxers, appears to be related to the length of the boxer's career and to the evidence of brain damage occurring during the fights [86]. Neuropathologic studies usually show some enlargement of the lateral ventricles and fenestration, and sometimes large cysts of the septum pellucidum. There may also be thinning of the corpus callosum and scarring of the lower surface of the cerebellum. Neurofibrillary tangles are often seen in the cerebral cortex and brainstem, but especially in the gray matter of the medial temporal lobes [82, 87]. CT studies [86–88] usually show rather striking ventricular enlargement; large cysts of the septum pellucidum may be apparent, and sometimes expansion of a cavum vergae as well. The temporal horns are often enlarged. A clinical history of repeated head trauma should help to differentiate the changes of dementia pugilistica from those seen in patients with ATD.

Figure 11 shows CT scans of a 77-year-old man who presented with ataxia, memory loss, and diminished cognitive function. When the question of dementia pugilistica was raised by CT, the attending physician at first denied that his patient had any history of boxing. On further questioning, however, the patient admitted that he had boxed in his youth but had quit during his twenties because of head injuries.

#### Huntington's Chorea

This is a well-known hereditary disease characterized by the development of progressive choreiform movements and dementia. It usually becomes manifest in adult life although it is occasionally seen in young children. The basal ganglia, cerebral cortex, and white matter are involved, but the caudate nuclei, particularly, become shrunken as the disease progresses. CT studies usually show progressive widening of the intercaudate region of the lateral ventricles [89–91]. The extent of the caudate atrophy does not necessarily parallel the apparent clinical stage of the disease, and patients may have gross dementia although their CT scans appear relatively normal. A positive family history is an important aid in the diagnosis of Huntington's chorea, and it has been reported that Huntington's patients have decreased levels of gamma aminobutyric acid (GABA) in their CSF,

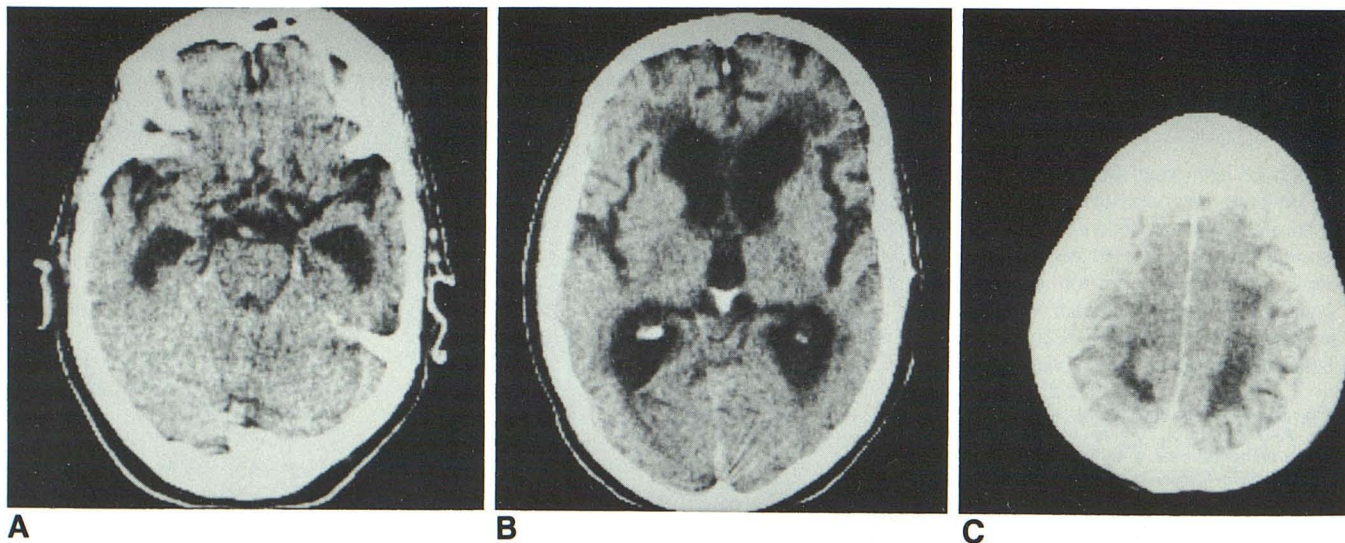
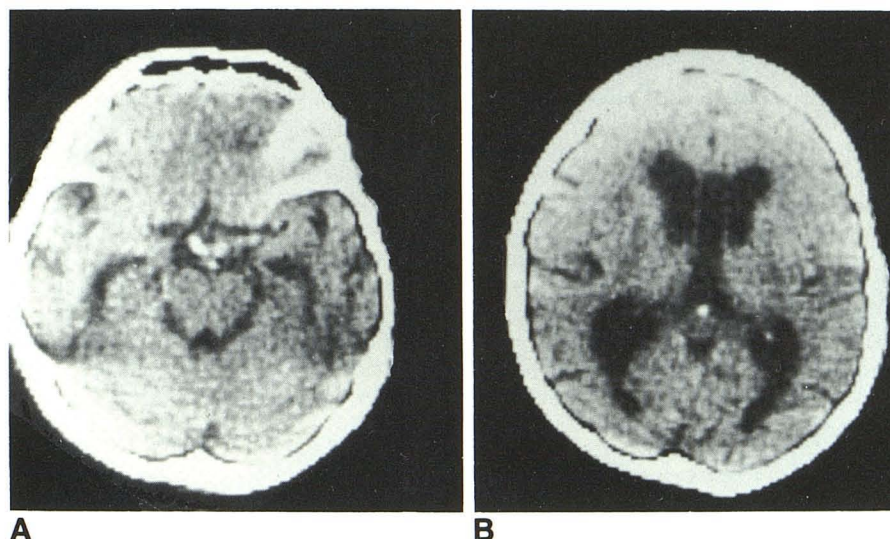


Fig. 10.—Normal pressure hydrocephalus. A, B, and C, Scans of 87-year-old man with normal pressure hydrocephalus who presented with walking difficulty. Frontal horn ratio was over 0.5. Temporal horn tips are very prominent,

and there are areas of low densities about lateral ventricles. Sulci were not prominent over the hemispheres.



Fig. 11.—Dementia pugilistica. **A** and **B**, Study of a 77-year-old man with ataxia, memory loss, and some decrease in brain function. Patient had been a boxer in his youth. Temporal horns are very prominent and there is a large cyst of the septum pellucidum.



which can help confirm the diagnosis [92]. Huntington's disease has now been found to be linked to a DNA fragment of chromosome 4 [93, 94]. It is probable that soon it will be possible to identify persons who are at high risk for the disease. Chromosomal studies may also lead to effective treatment [95].

Figure 12 is a CT scan of a 37-year-old woman with Huntington's chorea. There was a family history of the disease, she had a severe movement disorder, was demented, and had a decrease of GABA in her CSF.

#### *Dementia Associated with Metabolic Disorders*

Dementia, which is often reversible, may be caused by metabolic disorders of the nervous system [96]. One not uncommon condition is the Wernicke-Korsakoff syndrome, which is associated with alcoholism. These patients often show unsteadiness of gait and a marked memory deficit that is thought to be related primarily to a thiamine deficiency [97]. Histologically, lesions are seen in the thalamus and hypothalamus at the margins of the third ventricle and also in the cerebellum, particularly in the vermis and in the floor of the fourth ventricle. CT studies usually show widening of the CSF spaces, including the temporal horns of the lateral ventricles and vermal sulci of the cerebellum. Although they resemble the changes of ATD in some respects, the temporal lobe alterations are usually not as striking in patients with metabolic disorders. PEG [98, 99] and CT studies of chronic alcoholics have often shown the third ventricle to be strikingly widened. Figure 13 shows CT scans of a 32-year-old chronic alcoholic who entered the hospital mentally confused and with severe difficulty walking. Alcoholics are particularly susceptible to trauma resulting in head injuries, and evidence of asymmetrical tissue loss may be seen on CT scans. The ventricles and sulci of chronic alcoholics can return to normal, or near normal, size if alcohol intake is greatly reduced [100, 101]. Marchiafava-Bignami disease is a rare disorder sometimes seen in chronic alcoholics. It was first reported in Italy and thought to be caused by certain wines. It is associated with loss of tissue in the corpus callosum and usually also some lesions are present in the white matter. Evidence of lesions in the corpus callosum have been reported by CT in two patients who died of the disease [102].

Decreased brain function is sometimes seen in patients with Cushing's disease and other illnesses associated with increased steroid

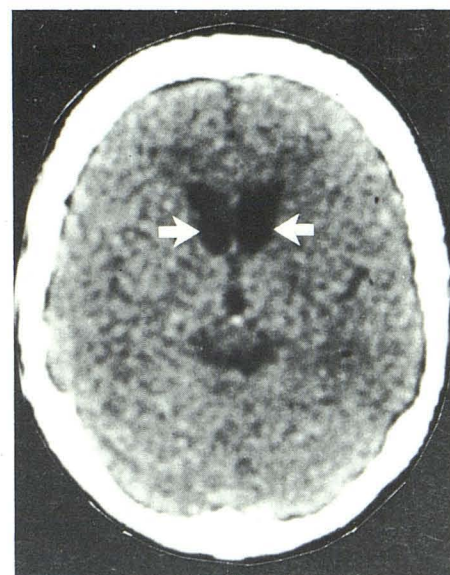


Fig. 12.—Huntington's chorea. Scan of 37-year-old woman with severe movement disorder and decreased mental functioning. There is marked widening of intercaudate distance (arrows), which is nearly as wide as frontal horns anterior to caudate nuclei. An earlier scan, taken when her symptoms were developing, showed no obvious widening of intercaudate distance.

levels in the body [103]. CT changes in these individuals can resemble those seen in patients with ATD; as in alcoholics, however, the brain changes are less striking, and are often reversible when the cause of the metabolic changes is eliminated. Similar CT changes also may occur in individuals who are malnourished or are in renal failure or are receiving chemotherapy for cancer. Figure 14 shows the CT scan of a young man with carcinoid tumor of the ileum and metastasis to the liver. He had increased 5-hydroxyindol acetic acid in the urine resulting from increased serotonin in the body.

Hypoxic encephalopathy is often a more serious form of metabolic disorder of the nervous system and usually causes some permanent



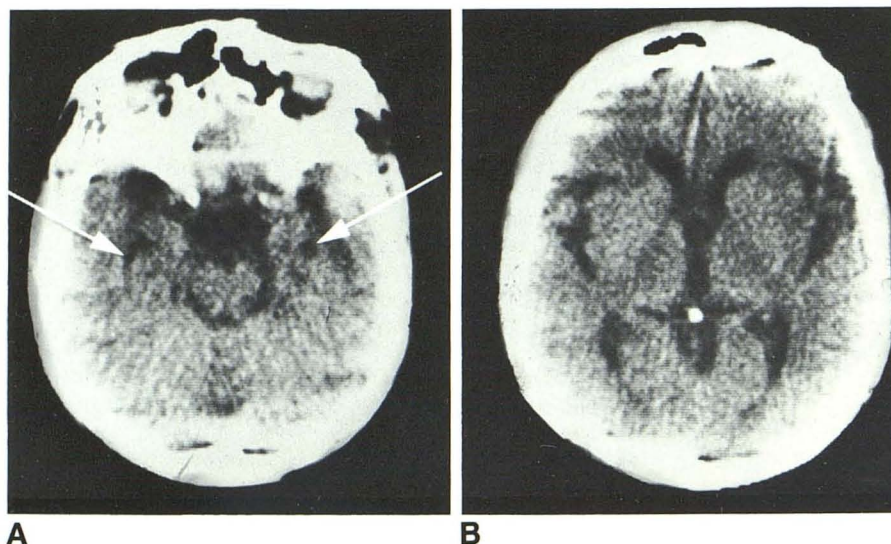


Fig. 13.—Alcoholism. **A** and **B**, Scans of 32-year-old male chronic alcoholic, who entered hospital confused and had marked gait difficulty. Third ventricle, sulci, and fissures are strikingly widened, and there is also some widening of temporal horns of lateral ventricles.

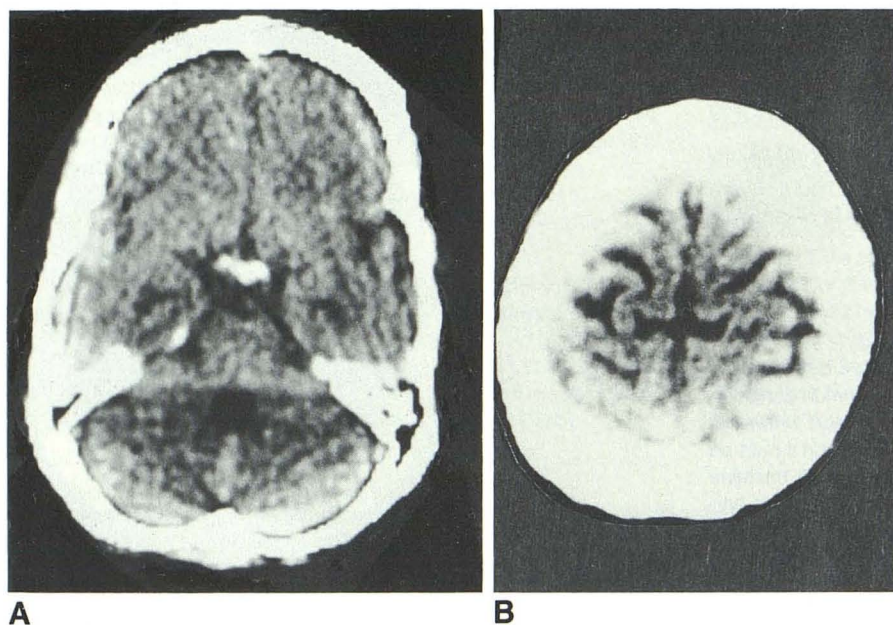


Fig. 14.—Carcinoid tumor. **A** and **B**, Scans show some widening of CSF spaces in 27-year-old man with carcinoid tumor of the ileum and liver metastasis. Patient had periods of flushing of the face and intermittent diarrhea for many months and showed some changes in mental status. Widening of suprasellar cistern and temporal horns are not as striking as is commonly seen in patients with Alzheimer's type dementia.

damage to the brain. It may occur after severe respiratory and heart disease or exposure to chemical poisoning, such as carbon monoxide inhalation, or after the ingestion of methyl alcohol. Neuropathologic studies usually show changes particularly in the globus pallidus, cerebellum, and hippocampus. Clinical symptoms depend on the degree of brain damage; memory loss is frequent. CT studies, besides showing well-defined, low-density lesions in the basal ganglia may also show widening of the temporal horns of the lateral ventricles [104].

### Discussion

Less than 15 years ago the diagnosis of mental illnesses depended mainly on clinical criteria, aided by plain x-rays of the skull and PEG. CT has revolutionized the diagnostic

capabilities in this area. Positron emission tomography (PET) is another relevant addition to the imaging of mental disease. PET studies [105–107] have shown hypometabolism, especially in the parietal regions, in patients with ATD compared with age-matched controls. The findings cannot be considered entirely diagnostic, since similar changes have been reported with Creutzfeldt-Jakob disease [108]. PET is also able to show, in patients with Huntington's disease, decreased activity in the caudate nuclei. This becomes evident before any changes are seen by CT, and it may help predict the likelihood of young family members developing the disease [105, 107]. In conjunction with CT studies, it is also helpful in the study of patients with strokes [109, 110]. PET is a developing field [111]. MRI is rapidly proving valuable in



the diagnosis of mental illnesses, and many small lesions can be seen with it that are not seen by CT. It will probably be some time, however, before enough data are collected from patients with global degenerative brain disease to utilize MRI to its greatest advantage.

A decrease in mental functioning in adult life may result from innumerable causes such as depression, endocrine disorders, systemic illnesses, drugs, toxins, and space-occupying lesions that may be successfully treated [96]. Alzheimer's disease is presently the most common dementing illness seen. Although global atrophic changes may be observed in brains of Alzheimer patients, the most striking alterations are seen in the temporal lobes. If on CT of a patient suspected of having ATD one does not see enlargement of the temporal horns, a tenacious search should be made for another cause of the dementia. Some scientists have suggested that ATD may be caused by a slow virus, but the difference in CT findings between patients with ATD and those with Creutzfeldt-Jakob disease would suggest they are not caused by similar pathogens. CT, at present, is valuable in diagnosing not only space-occupying treatable lesions, but also in diagnosing primary degenerative brain diseases.

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