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Two Examples of "Presenile Dementia"

(Pick's disease and Stern-Garcin syndrome) With a History of Trauma*

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With 6 Figures in the Text

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Trauma has been postulated as a causal or contributing factor in a variety of degenerative conditions of the brain. The number of instances however in which the symptoms have appeared insidiously within a few weeks of an accident for which the patient has not in any way been responsible and who was indeed adjudged to be in perfect health before it must be few. In a case of Creutzfeldt-Jakob disease of little more than twelve weeks evolution that fulfilled these conditions, a healed contusion of the frontal lobe provided at necropsy unsuspected confirmation of the trauma (BEHRMAN et al. 1962). Even in such instances however the possibility of the disease being established but subclinical at the time of the accident can never be excluded: for example, the person who develops general paralysis soon after an accident must be syphilitic at that time although his general infection may be quiescent. The man who after a faulty parachute landing developed symptoms of Huntington's chorea (CLAUDE et al. 1930) previously represented by only mild choreiform movements is probably an instance of accentuation of a genetically installed disease following trauma. The 81 year old patient described by VON BRAUMÜHL (1928) who died some months after an accident with a subdural haematoma and Pick's disease had clearly been dementing for two years prior to the accident. We report here two examples of "presenile dementia" which appear to have developed shortly after an accident: the trauma in the second case was apparently trivial.

Case 1. A spinster aged 47 was a back seat passenger in an automobile which ran into a concrete post. She subsequently stated that she remenbered talking to her sister when the impact occurred but that her next memory was of making an effort to open the door: she had however no recollection of how she came to be released from the wreckage although she recalled being greatly moved on seeing her sister in the roadway with blood dripping from her face. The injuries discovered in the casualty hospital consisted of orbital contusion with effusion closing the right eye, aching and pain in the right flank, bruising over the right lower jaw, abrasions on the front of both shins, and a cut on the bridge of the nose necessitating two

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stitches. She returned to her secretarial work 3 weeks later, but shortly afterwards complained of daily headaches and a feeling of depression. She also noticed that with the right hand she was unable to type with her customary ease, while her handwriting had also deteriorated.

When seen 9 months after the accident she complained in addition to the above symptoms, all of which had become worse, of an undue liability to fall. Examination revealed early spastic paresis of her right limbs and bilateral extensor plantar responses.

Sixteen months after the accident movements and walking were found to be extremely slow, with loss of arm-swing and tendency to drag the right leg. All tendon jerks were increased. Her speech was slow but there was neither dysarthria nor dysphasia. She was scarcely able to write her name. There was no disturbance of sensory functions. No Kayser-Fleischer ring was found. The E.E.G. was within normal limits.

Eighteen months after the accident she was quite unable to give a satisfactory history and throughout the interview was unduly lachrymose. When tested on the Wechsler adult intelligence scale her performance was inconsistent, but it was felt that there was some decline from the average, with impairment of visual memory and of the ability to learn verbal material. All limbs showed increased resistance to passive movements and increased tendon jerks. The jaw jerk was brisk and the plantar responses were extensor. When walking, the right limbs maintained a hemiplegic posture. The total urinary amino acids were within normal limits.

Six months later she was still able to mount stairs although with considerable difficulty but when seen by one of us (S.B.) thirty months after the accident she had a spastic bulbar paresis with anarthria although some voluntary movements could be elicited on the left side of the face. She was able to deviate her eyes to the right and close her eyes on command. The jaw jerk was still exaggerated and it was reported that she found it difficult to masticate and swallow food. All limbs were stiff and immobile being maintained in a fixed attitude. The shoulders were adducted and internally rotated, the arms flexed at the elbows with wrists pronated and flexed, and hands clenched. Her lower limbs were extended and adducted at the hips, extended at the knees and plantar-flexed at the ankles with slight inversion. Fasciculation was seen in the left deltoid and triceps muscles. She died 2 years and 11 months following the accident, a clinical diagnosis having been made of Creutzfeldt-Jakob disease.

A postmortem examination revealed nothing of note other than an abnormal brain. It weighed only 869 g and showed a moderate degree of symmetrical atrophy maximal in the posterior parietal region but sparing the occipital lobes. The leptomeninges were not thickened and the vessels at the base of the brain were devoid of atheroma. After detaching the brain stem and cerebellum, both of which were noted to share in the general atrophy, the cerebrum was divided sagittally, the left half being processed in paraffin-wax and the right in celloidin. A mild degree of ventricular dilatation was associated with some thinning of the corpus callosum and considerable shrinkage of the caudate and lentiform nuclei, the putamen having a greyish discoloration (Fig. 1). The cortical ribbon in some parts of the parietal lobe was reduced to 2 mm. The spinal cord was normal in appearance.

Histology: only the brain and spinal cord were available for study.

Brain: thickening of *leptomeninges* over right frontal pole, especially in sulci with adherence to underlying brain and occasional ingrowing of capillaries from meninges into cortex accompanied by glial fibre formation; some haemosiderin contained in histiocytes around blood vessels. *Cerebral cortex:* lamination obscured in many parts

of frontal and temporal lobes and to a lesser extent in parietal lobes by reason of marked microglial and astrocytic proliferation, the latter especially in laminae 4, 5 and 6; "twinning" of astrocytic nuclei apparent in laminae 2, 3 and even 1. In Holzer preparations accentuation of fibre formation maximal in lamina 4 becoming less so as white matter is reached; no gliosis of subcortical arcuate fibres. Cortical gliosis most marked in T3 anteriorly; less so in cingulate gyrus, F1 and precentral gyrus; in remaining temporal and frontal gyri, slight gliosis in the deeper

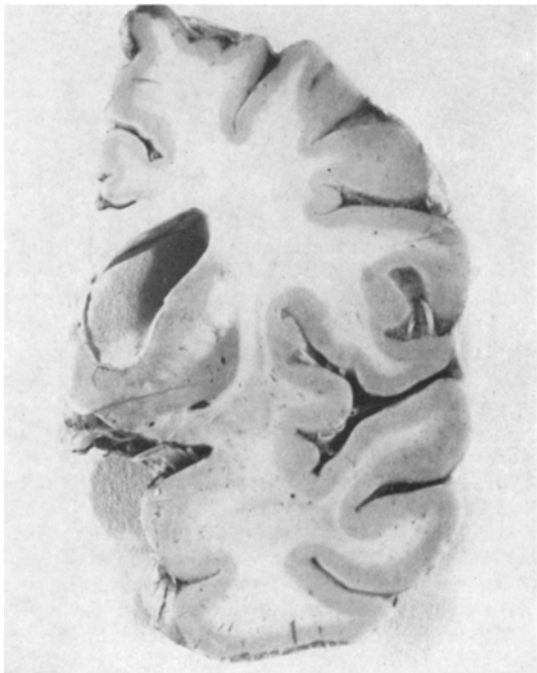

Fig. 1. Case 1. Left hemisphere cut in coronal plane at level of the chiasma. The putamen has a noticeably grey colour. (Formalin fixation)

laminae; none in parietal and occipital cortex. In T3 the normal occurrence in lamina 4 of pyramidal cells spread over from laminae 3 and 5 is accentuated because many of these cells are conspicuous by virtue of their size and their degree of vacuolation, this change being evident in other diseased parts of the cortex; several resemble Pick cells and silver-impregnated sections (Davenport's technique) reveal a fair number of the characteristic intracytoplasmic bodies; also some contracted and pyknotic pyramidal cells in which it is difficult to discern a nucleus. The distribution of Pick cells as revealed by Davenport's technique is confined to areas commonly involved in Pick's disease (throughout T2, T3 and gyrus fusiformis and in rostal part of T1 but not the caudal part of T1 except in the depth of the superior temporal sulcus). In the hippocampal gyrus and formation, Pick cells present (Fig. 4a); also in latter situation many swollen axons. Astrocytic proliferation and loss of neurons are not conspicuous in pyramidal cell layer, but several cells including some in end plate are diffusely argyrophilic, their apical dendrites standing out prominently; others show focal thickening and apparent clumping of the neuro-

fibrils, often only one side of a cell being involved. This "Alzheimer neuron" effect is seen in other diseased parts of the cortex (Fig. 4b), sometimes coexisting with an inclusion of Pick type. These altered neurofibrils do not completely resemble those seen in Alzheimer's disease and the argyrophilic features are neither positive with Congo red nor are the cells birefringent. In pyramidal cell layer of the hippocampus no typical granulovacuolar degeneration (SIMCHOWICZ). Axonal swellings seldom seen except in areas where there are argyrophilic inclusions: an occasional

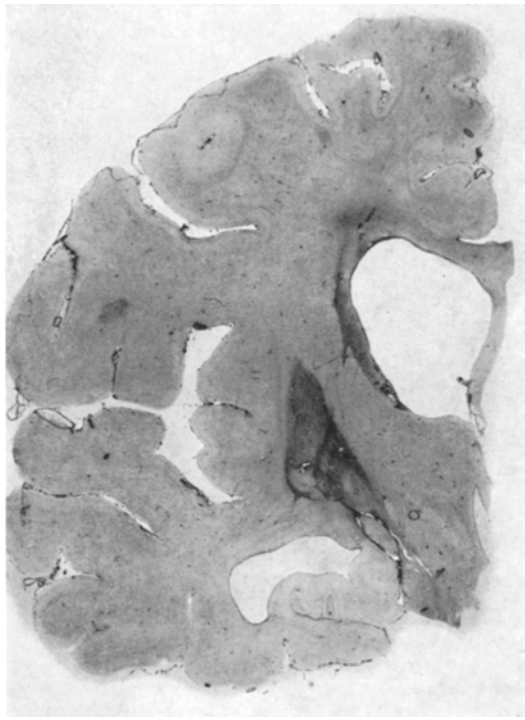

Fig. 2. Case 1. Right hemisphere cut in coronal plane at level of corpora mamillaria. Gliosis is marked throughout atrophied caudate and putamen, also in globus pallidus and radiation of corpus callosum. (Celloidin section. HOLZER)

one is present however in occipital lobe but not in striate area. No argyrophilic plaques. Blood vessels, seemingly normal. Slight generalised increase in microglia. *Caudate nucleus:* spongy appearance, especially adjacent to the ventricle; atrophied throughout but more marked rostrally than caudally; paucity of recognisable nerve cells but some large oval cells full of a yellow-pigmented substance are possibly altered neurons; no argyrophilic inclusion bodies and no axonal degeneration; astrocytic nuclei in increased numbers with dense subependymal gliosis tending to be isomorphic around the subependymal veins. *Putamen:* changes similar to caudate but atrophy less marked, glial overgrowth is more pronounced (Fig. 2) and there are probably more neurons (difficult to be certain since many cells are so altered that they may be interpreted either as enlarged and distorted astrocytes or degenerating neurons (Fig. 4f); cytoplasm of these swollen cells is encrusted with granules of

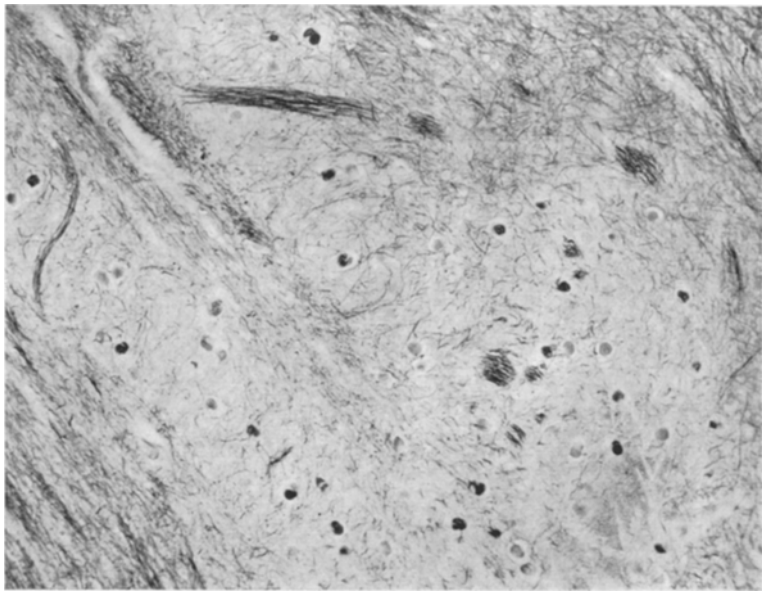

Fig. 3 (Legend see p. 133)

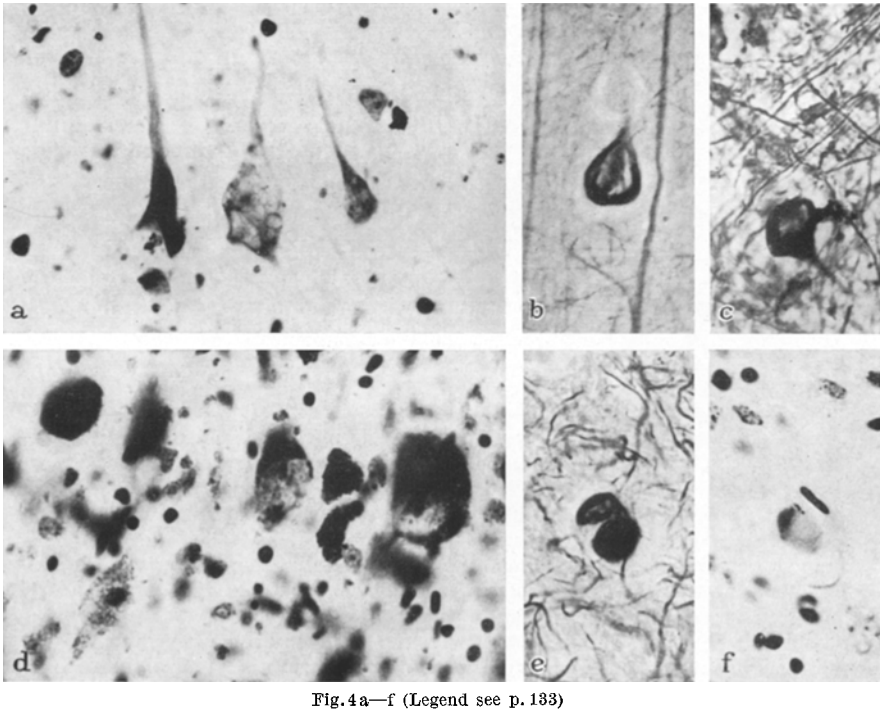

Fig. 4 a—f (Legend see p. 133)

dark brown pigment, identified by its reactions and its anisotropism as acid haematin. A small quantity of haemosiderin is also seen in the Virchow-Robin spaces. Capillaries seem plentiful, possibly due to the atrophy. *Pallidum* shares in putaminal gliosis but many nerve cells have survived although some are pyknotic and others are fatty; Pick cells not present. Both caudate and lentiform nuclei contain active microglial cells but compound granular corpuscles, few in number, are confined to the Virchow-Robin spaces. *Thalamus:* little altered apart from the lateral nucleus where argyrophilic inclusions of Pick type are noticeable and, in Nissl-stained sections, swollen nerve cells are correspondingly present. Microglia are in excess but there is no gliosis. *Subthalamic nucleus*: swollen and vacuolated nerve cells are more frequent here than elsewhere and Davenport preparations show a correspondingly large number of "Pick bodies". *Red nucleus* is similar (Fig. 4c): both here and in the subthalamic nucleus capillaries seem increased in number out of proportion to degree of atrophy. Astrocytes are undergoing proliferation with fibre formation; microglia in excess. *Corpora mamillaria:* normal. *Substantia nigra:* noticeably depigmented to the naked eye, shows moderate loss of pigmented cells with some free-lying melanin and occasional Pick cells with cytoplasmic inclusions (Fig. 4d): no Lewy bodies and no Alzheimer cell change. Marked proliferation of astrocytes and dense fibre gliosis. *Substantia ferruginea:* normal. *Nuclei pontis*: neurons show considerable chromatolysis and many are swollen: a few contain granules inside vacuoles. There are a few Pick cells. *Olives:* gliosis in excess of normal, with increase in microglia; neurons, swollen and fatty; in Nissl preparations occasional Pick bodies are discernible (Fig. 4e) but in Davenport preparations they are seen to be numerous (Fig. 3); occasional neuronophagia. *White matter:* Heidenhain-stained preparations show no demyelination; considerable gliosis of the radiation of the corpus callosum and the forceps minor and, to a lesser extent, of the corpus callosum seen with Holzer's stain; in Cajal's preparations hyperplasia and some proliferation of astrocytes in the temporal lobes, rather less so in the frontal and perietal and none in the occipital lobes. *Internal capsule:* no pallor of myelin staining; axons in posterior limb: occasional thickening and tortuosity. In the brain stem the *corticospinal tracts* are atrophied with noticeable pallor of myelin staining but no fibre gliosis: the temporo-pontile fibres are not involved but the fronto-pontile fibres are to a moderate degree: in the practically demyelinated areas of the basis pedunculi the axons are tortuous and irregularly swollen. *Cerebellum:* patchy gliosis but dense in one or two areas of the cortex; Purkinje cell population patchily reduced with misalignment and Bergmann astrocytes increased. Focal atrophy of cortex in most of one folium involving all layers with dense isomorphic gliosis of molecular layer. Dentate nucleus infiltrated with microglia sometimes arranged in knots: no Pick cells but lipochrome content of neurons excessive. *Spinal cord:* marked demyelination of pyramidal tracts throughout the cord. In the cervical enlargement a few of the anterior horn cells present abnormal forms, vacuolation of the cytoplasm and exceptionally dissolution of the nucleus; probably a reduction in the number of cells on one side.

Case 2. A fifty-one year old physician was said to have been in good health until one day when, while driving and waiting to turn right, his automobile was

Fig. 3. Case 1. Inferior olive: the abnormal neurons are conspicuously black. (Celloidin section. DAVENPORT's stain ×80)

Fig. 4a—f. Illustrations. Case 1. Celloidin sections. a 3 abnormal neurons in pyramidal cell layer of the hippocampus, one of them with vacuoles in the cytoplasm (NISSL). b Neurofibrillary degeneration in lamina 5 of F2 (DAVENPORT). c Neurofibrillary degeneration in red nucleus (DAVENPORT). d Abnormal neurons in the substantia nigra; "granulovacuolar degeneration" of cytoplasm (centre) and loss of pigment (bottom left) with free-lying pigment and increase of microglia (NISSL). e Pick cell in inferior olive (DAVENPORT). f Pick cell in putamen (NISSL)

bumped into from the back by another vehicle. He stated subsequently that he was shaken by the accident and had a bruised feeling round his shoulders. There is no record of head injury. He went to a public telephone-box and arranged for the police and a garage to be informed. He then set off to walk home but was recognised by a friend who gave him a lift: when he reached home he continued with his work. The following day he felt tired and although he continued to work he could not remember details and complained that his mental processes seemed not to be so clear and quick as they had been.

Three weeks after the accident he felt constrained to give up work because of his inefficiency. He had lost his appetite, could not sleep and had recourse to sodium amytal though without effect; he felt depressed. After a week in a nursing home he felt better and was anxious to return to work but when he did so he was disappointed to find that his memory—especially for names—was less certain than formerly and he was frequently having to consult his notes. He found it difficult to make decisions and was anxious to be left alone. His wife confirmed that he was depressed and said that he was often tearful. There was a family history suggestive of depressive illness. He was given chlorpromazine and later electric shock treatment without avail.

Eight weeks after the accident he was seen by a neurologist who found him to be suffering from a depressive illness brought on by the accident. However he found no evidence of organic disease. A further course of electric shock treatment brought about no improvement.

When, twelve weeks after the accident, he was admitted to a general hospital he was confused and unable to give any account of himself. He was sweating a little and had a generalised coarse tremor accompanied by myoclonic twitches of all limbs. His speech was slow and hesitant and it was noted that he had some dysarthria. The tone in all four limbs was increased but reflexes were normal and plantar responses were flexor. It was not possible to test co-ordination but there was no loss of sensation to pinprick. The optic discs appeared normal but the eye movements were somewhat restricted. A general examination revealed no abnormality. The blood pressure was 150/120. The urine was normal. X-ray of chest and skull were normal. Spinal fluid, under normal pressure, contained 1 cell/cmm and 57 mg per cent protein. Barbiturates not detected in the blood.

On the morning after admission he had improved slightly but was still confused. The rigidity of this limbs persisted and he was found to have a grasp reflex in both feet. Three days later the temperature went up to 101.2°, his respirations were periodic in character, he passed little urine and he developed a foetor oris; the blood urea was 165 mg per 100 ml and examination of the electrolytes revealed acidosis. He was seen by one of us (S.B.) and was believed to be a case of "subacute spongiform encephalopathy" aggravated by renal failure. He did not respond to hydrocortisone and "durabolin" and on the following day he was unconscious with a blood urea of 260 mg per cent, dying a few hours later.

A postmortem examination revealed a fairly well-nourished man showing some dehydration and loss of flesh. Brain congested but not swollen. Apart from slight coronary atheroma nothing abnormal was seen. The heart was not enlarged.

After fixation the brain weighed 1578 g and when cut in the coronal plane a symmetrical swelling of the white matter was noted with reduction in size of the ventricles. There was slight opacity of the leptomeninges especially over frontal cortex.

Histology: patchy occasional aggregates of fibroblasts particularly in the depths of the sulci where the pia is adherent to the underlying cortex; here proliferating capillaries are seen growing from meninges into brain through a felt-work

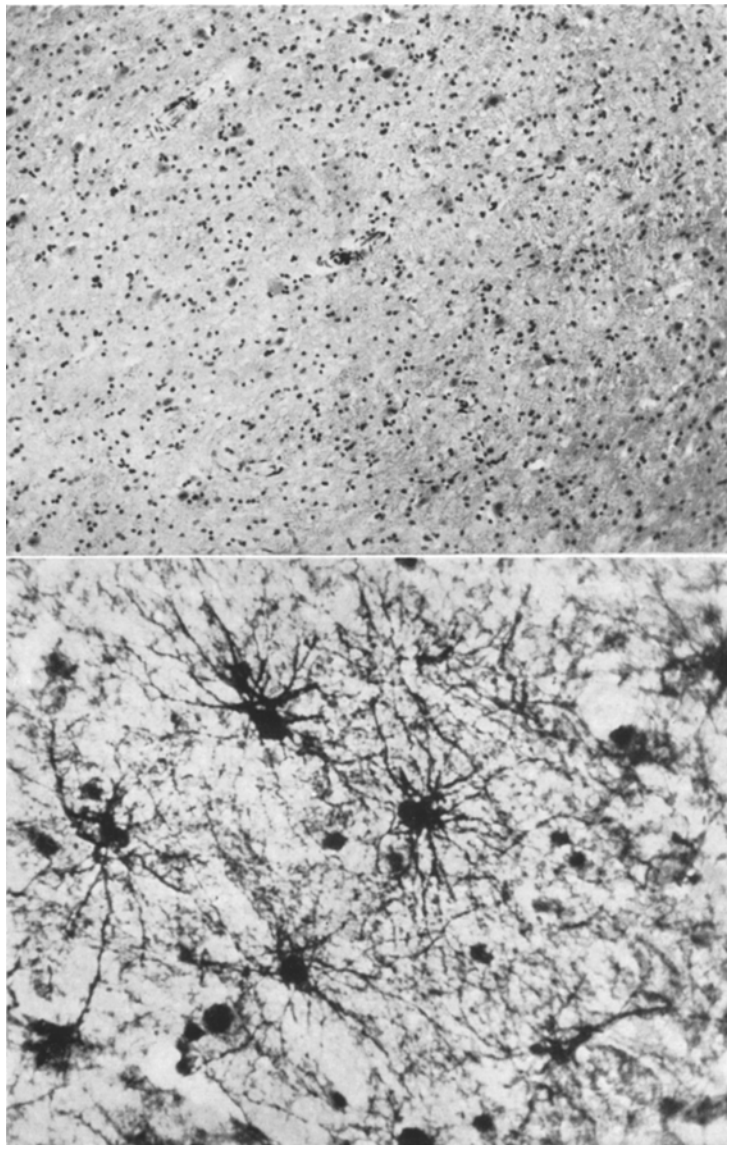

Fig. 5

Fig. 6

Fig. 5. Case 2. Lateral nucleus of thalamus: Loss of neurons and increase in glial nuclei. (Celloidin section. Haematoxylin and eosin ×80)

Fig. 6. Case 2. Lateral nucleus of thalamus: Proliferating astrocytes. (Frozen section. Cajal. ×400)

of glial fibres: most noticeable in the orbital sulci, these changes nevertheless seen in many areas apart from frontal lobes. Ependymal lining of third ventricle similarly thickened and adherent to subjacent thalamus. *Cerebral cortex:* some reduction in neuronal population; disruption of cytoarchitecture by invading microglia and

proliferation of astrocytes: these changes most marked in frontal, parietal and temporal areas. Large neurons of amygdaloid, not appreciably diminished in number, carry considerable quantity of acid heamatin granules in or on their cytoplasm. Markedly proliferated astrocytes and microglial cells do not contain any pigment and none is seen lying free. Endplate of hippocampal formation shows severe at its apex gliosis. Gliosis of cortex accentuated in areas beneath the adherent meninges; in these areas also proliferation of capillaries growing in from leptomeninges. Occasional deposits of haemosiderin identified in macrophages near blood vessels in all parts of cortex. *Caudate* and *putamen:* some diminution in number of large neurons, probably of small ones too though hard to judge since proliferated and swollen astrocytes apt to be confused with smaller neurons. Cytoplasm of neurons but not glial cells in caudate stippled with granules of haematin. Noticeable increase in microglia; fibrillary astrocytes pathologically increased beneath the ependyma overlying caudate. Microglial cells active in *globus pallidus:* apart from this and presence of haemosiderin in vicinity of several blood vessels it shows no abnormality. *Thalamic nuclei* show similar changes though loss of neurons is more patchy: most noticeable in lateral nucleus (Fig.5). Astrocytic overgrowth not only more pronounced in thalamus (Fig.6) than in the corpus striatum, also qualitativelydifferent: many astrocytes have large, slightly pale, nuclei with prominent nuclear membranes and very scanty cytoplasm; they often appear in pairs and are slightly suggestive of "noyaux nus". Occasional deposits of haemosiderin both lying free and in cytoplasm of macrophages near blood vessels; considerable irregular accentuation of gliosis beneath ependyma lining third ventricle. *Subthalamic nucleus,* normal; loss of neurons in *red nucleus* and many proliferating astrocytes here have large deformed nuclei. *Substantia nigra,* healthy. Apart from presence of haematin pigment in many neurons of *pons,* and haemosiderin in macrophages near blood vessels, no abnormality. *Medulla:* only abnormality is a severe degree of central chromatolysis of olivary neurons, in many cases distended and loaded with lipochrome. *White matter* of cerebral hemispheres shows some signs of oedema and moderate increase in astrocytes, particularly in junctional zone; no demyelination, and fat stains negative. *Cerebellum,* normal except for increase in microglia of dentate nucleus and some increase in fibrillary astrocytes in white matter of folia. Neuron population of dentate not depleted but noteworthy that cytoplasm of several neurons is heavily encrusted with acid haematin. Similar granules of pigment found in or on Purkinje cells and Bergmann cells. Haemosiderin deposited in macrophages seen in neighbourhood of larger blood vessels in white matter.

Commentary

The first case is regarded as an instance of Pick's disease because of the selective character of the atrophy—sparing for example the caudal two thirds of the first temporal gyrus—, the heavier involvement of the rostral as against the caudal end of the caudate and putamen and the presence in atrophied areas of Pick cells with argyrophilic inclusions. The severe involvement of the corticospinal tracts in the spinal cord and the presence of altered neurofibrils in some of the involved neurons are well-recognised in Pick's disease. Less usual features are the tendency for the atrophic process to affect the deeper rather than the superficial layers of the cortex, the severity of the process in the putamen and the major role played by the microglia, features however noted by Lüers and Spatz (1957) to be more commonly met with in the younger patients

afflicted with this disease. The scantiness of the gliosis in the white matter may be related to the short duration of the disease.

A greyish pigmentation of the putamen was noted when the fixed brain was cut, and histologically this centre contained acid haematin in all sections: elswhere there was very little. Such pigment ("formalin pigment") is usually discounted for it is found only if processed material has been fixed in unbuffered formalin in places where blood is to be expected. For instance, when the brain has been heavily contaminated with post-mortem blood, the pigment is to be expected in or near the brain surface. In the present case, however, it is to be seen almost exclusively in the putamen: furthermore the granules of pigment are on the surface of or even within the cytoplasm of its cells. One possibility accounting for a heavy deposition of acid haematin in any deeply situated centre might be a relative excess of blood vessels allowing the post-mortem liberation of a greater quantity of blood and consequent diffusion into the surrounding tissue: in this instance however the relative proportion of capillaries to parenchyma in the putamen does not appear to be as great as in the red nucleus, where there is no formalin deposit. Another possibility is that, as a result of the trauma, bleeding had taken place into the putamen: but in this eventuality, provided the bleeding had taken place at least a few days before death, haemosiderin would be expected unless for some reason the normal process of degradation of haemoglobin had been prevented. Here, had bleeding taken place or had necrosis developed in the putamen (as has been reported in 3 cases of trauma by MALAMUD and HAYMAKER 1947), more haemosiderin would be expected than the trace which was in fact found. A third explanation is that damaged nerve cells may attract haematin in the same way by which they can become ferruginised. The distribution of acid haematin in this brain is not easily explained. Putaminal pigmentation has been described in Huntington's chorea (MANSVELDT 1954) and has been indentified as acid haematin: we ourselves have seen another instance of this. It would be interesting to know if in these cases the putaminal pigmentation would have been seen had the brain been fixed in buffered formalin. In our experience the haematin granules which are most brightly anisotropic are actually the ones which are least pigmented: that is, they are practically invisible among their pigmented fellows until crossed prisms are used. Putaminal pigmentation in Case 1 of a series of 4 cases of striatonigral degeneration reported by ADAMS et al. (1964) was assumed to be due to the lipofuscin in cells.

Pick's disease has been described on several occasions in patients who have previously experienced trauma but the ensuing interval up to the onset of symptoms has seldom been under two years (JANSEN 1938, 8—12 weeks; GRUENTHAL 1930, Case I, a few weeks; REICH 1907,

8 weeks). In the present case the interval was eight weeks. It must remain uncertain as to why this patient developed Pick's disease eight weeks after her trauma, but the heavy involvement of the corpus striatum suggests that it was in this centre that the trauma may first have induced an atrophic process in a person genetically prone to this disease.

In the second case the pathology is consistent with the type of rapid dementia associated with thalamic degeneration (Stern 1939; Garcin et al. 1962). To our knowledge this particular clinicopathological syndrome has not been previously associated with trauma although an example of Creutzfeldt-Jakob disease (of which this syndrome is believed by several authors to be but a part) following trauma has already been reported (Behrman et al. 1962). Moreover, there are several examples of the closely related condition of "subacute spongiform cerebral atrophy" which have followed brain damage of one sort or another, although the interval of time after the incident has usually been longer.

The time of onset of the disease in this case is uncertain, for it would seem that the illness which immediately followed the accident was clinically an endogenous depression: because of this possible masking effect one cannot say when the Stern-Garcin syndrome began. Nor is it possible to exclude the various forms of treatment which he received from being concerned in the pathogenesis of his final disease. None of these however can be held responsible for the admittedly slight and patchy thickening of the pia-arachnoid and the areas of meningocortical adhesion: haemosiderin within macrophages points toward leakage of erythrocytes without however indicating the cause. The renal failure appears to have been terminal but renal disease in association with subacute spongiform encephalopathy has been reported (Case 8, Nevin et al. 1960) and a peculiar form of "spongiform encephalopathy" has been reported as a terminal state in long-standing but treated malignant hypertension (McMenemey and Pallis 1962).

Speculations as to the significance of the concentration of acid haematin in certain centres—amygdaloid, dentate, parietal and cerebellar cortex in particular—cannot be resolved since the extent to which acid haematin can diffuse after death is not constant.

Summary

Two cases of "presenile dementia" are reported in which symptoms developed soon after a traffic accident.

The first was a woman aged 47 whose first symptoms were apparent 8 weeks after an automobile accident and who died $2\,^1/_2$ years later. The histological findings are compatible with a diagnosis of Pick's disease affecting principally the putamen and caudate.

The second was a man aged 51 who developed depressive symptoms immediately following a motor car collision which was not deemed at the time to be important. Loss of memory and myoclonic twitching followed and he died 12 weeks later with a terminal uraemia. The histological findings are those of the Stern-Garcin syndrome.

Zusammenfassung

Es wird über zwei Fälle praeseniler Demenz berichtet, in welchen sich Symptome kurze Zeit nach einem Verkehrsunfall entwickelten.

Im ersten Fall handelte es sich um eine 47jährige Frau, bei der erste Erscheinungen 8 Wochen nach einem Autounfall auftraten; sie verstarb $2^1/_2$ Jahre später. — Der histologische Befund war vergleichbar demjenigen einer Pickschen Krankheit mit hauptsächlichem Befall von Putamen und Caudatum.

In der zweiten Beobachtung entwickelten sich bei einem 51jährigen Mann unmittelbar nach einem zunächst für nicht erheblich gehaltenen Autozusammenstoß depressive Symptome; anschließend Gedächtnisverluste und myoklonische Zuckungen. 12 Wochen später Tod unter den Zeichen einer Urämie. Histologisch fand sich ein Stern-Garcin-Syndrom.

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