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FIVE BOXERS

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[WITH SPECIAL PLATE]

The occasional but tragic cases of fatal injury to the brain in professional boxing have been recently well publicized. The facts are undeniable. It is not so with the so-called punch-drunk syndrome. Although the term has been known in medical circles for over thirty years there are only three published necropsies in the world literature, none of them from this country. There are obvious difficulties in following the subsequent careers of former boxers, but it is going to be necessary if the existence or otherwise of this syndrome is to be finally established and placed on a firm foundation of pathology.

In this paper I describe the cases of five former professional boxers, four of whom developed chronic cerebral disorder in later life. The fifth is not neurologically disabled but is subject to bouts of aggressive behaviour which bring him into conflict with the law. Taken together, these cases illustrate the difficulties of aetiological diagnosis and emphasize the extent of our ignorance of this subject.

Case 1

A painter and decorator aged 45 was referred to the Department of Neurology by his practitioner in July, 1960. A former professional boxer since the age of 17, he had retired from the ring at 35 "because my friends said I was walking unsteady and throwing a leg out, and they said that was a sign of punch-drunk." He had started boxing at the age of 13 and said he had had about 350 bouts and had only once been knocked out. His "peculiar" gait had developed in an insidious manner about 13 years ago, and for several years he was unaware of it despite the comments of his wife and friends. He was said to throw out the left leg at times, and some thought he was drunk. He only gradually became aware of this unsteadiness about eight years ago. The symptom fluctuated and was aggravated by alcohol, but he was able to do his work and had never fallen. If he hurries he catches his left foot in obstacles and frequently stumbles. Slight clumsiness of the fingers, as when tying shoelaces or fastening buttons, has developed in recent years.

The second symptom, again one which was noticed by others for some years before he became aware of it, was of slurring of speech. He has found that he has to speak slowly and carefully if he wishes to be understood. The difficulty seems to consist solely in defective articulation; there have been no aphasic symptoms.

Both the unsteadiness and slurring of speech have gradually increased over the years and seem to fluctuate. In the past two years two further symptoms made their appearance. There has been urgency and frequency of micturition with occasional incontinence and bouts of blurring of vision. The latter are infrequent and sometimes consist of transient altitudinal hemianopia; "I might be unable to see the top half of your head or the lower part of your body for a few minutes or sometimes on and off for a day." He had seen several ophthalmologists, who had found no abnormality. There were no complaints of headache, vertigo, diplopia, numbness, or paraesthesiae, and his general health was satisfactory. He complained of dyspepsia and anorexia in 1955 and was investigated in a hospital out-patient department, where it was noted that "there is marked slurring of speech and unsteadiness of gait." There was no family history of neurological or mental disorder.

Examination.—General physical examination showed no significant abnormality, except the hall-marks of his profession (broken nose and cauliflower ear). B.P. 110/80. The positive neurological signs consisted of slurring dysarthria, a spastic-ataxic type of gait, and an extensor plantar reflex (left). There was no ocular or optic abnormality; no nystagmus; optic disks, fundi, and visual fields were normal. Power in his limbs was good, although there was some distal weakness in the left lower limb. Finger and hand movements revealed slight clumsiness. All reflexes were brisk. There was no sensory loss. He possessed a pleasant, frank personality, and there was nothing to suggest any deterioration. He was rather slow, but there was no evidence of intellectual impairment.

Investigation in hospital revealed normal radiographs of skull, chest, and cervical spine. Blood W.R. was negative. Lumbar puncture produced a fluid under normal pressure; protein 53 mg./100 ml.; cells fewer than 5/c.mm.; and a normal Lange curve and negative W.R.

Electroencephalogram.—This was normal. There was a regular, normally responsive alpha rhythm arising from the occipital lobes, and hyperventilation produced no abnormality.

Lumbar Air Encephalogram.—The lateral ventricles were not enlarged or displaced; the third and fourth ventricles were well outlined and were of normal size and in the midline. The aqueduct was normal. There was no excess of air over the cerebral cortex. On standard antero-posterior and postero-anterior radiographs the two lateral ventricles were confluent medially. The thin linear shadow produced by the septum pellucidum was absent. In the postero-anterior view the normal separation of the lateral ventricles was seen, but they were confluent anteriorly. An interaxial view confirmed the absence of the septum.

Progress.—In the past two years he has been kept under observation, and the air encephalogram was recently repeated. Dysarthria and an unsteady gait remain the two chief disabilities. He is euphoric, and psychometric testing indicated that he possesses average intelligence (I.Q. 106) with little evidence of intellectual deterioration. Further views undertaken on the second air encephalogram confirmed the apparently total absence of the septum pellucidum (Special Plate, Figs. 1, 2, and 3). Antero-posterior tomograms clearly

demonstrate this, while the lateral horizontal view recommended by Dyke and Davidoff (1935) when they reported their first example of absent septum pellucidum diagnosed by air encephalography revealed the comma-shaped shadow which they described. It obviously represents the confluent portions of both lateral ventricles at the midline (Special Plate, Fig. 3). Finally an antero-posterior view with the head in the horizontal position and the patient lying on his side showed that all the air collected in the uppermost ventricle. If a septum separated the ventricles some of the air should remain trapped in the lower ventricle.

Case 2

This patient, aged 52, was referred to the Department of Neurology by his practitioner in December, 1960. He retired from the ring when he was 35 after about 300 professional fights, and at that time his only disability, he recalled, was defective vision of the right eye, the result of injury in one of his fights. As in Case 1, he said he had been knocked out on only one occasion.

His doctor had referred him because of increasing weakness and lack of control of his right leg. The patient stated that this trouble had begun 12 years previously. He recalled a sudden weakness of his right leg while carrying a sack of coal. At first the symptom was intermittent, but over the years it has increased and he has found that he catches the toes of the right foot in the ground as he walks. He stumbles, rarely falls, and there is no story of fluctuation of the complaint. During the past 12 years there have been periods when he has experienced paraesthesiae and numbness in both his feet. His right arm is weak and clumsy at times. He finds difficulty in writing and holding a teacup. Ten years ago he began to have difficulty in urination. There has been frequency, hesitancy, nocturia, occasional dribbling incontinence, and, on one occasion eight years ago, a bout of retention requiring catheterization. There has been no prostatic enlargement. During the past three years his gait has been increasingly slow and unsteady, and he has often been thought to be drunk. He is abstemious but has found that alcohol aggravates his ataxia. He works regularly and still feels strong. He has no headache, vertigo, or diplopia. He was not aware of any difficulty with speech, and had no complaints to make suggesting any alteration of personality or intellectual impairment. There was no family history of neurological or mental disorder.

Examination.—As in Case 1 general physical examination showed no significant abnormality apart from a characteristically flattened nose and one cauliflower ear. B.P. 140/90. The positive neurological signs consisted of slow slurred speech, unsteady gait, moderate right-sided hemiparesis, and partial right optic atrophy. There was no dysphasia, he dragged his right foot when walking, and all movements were performed slowly and carefully. There was moderate ataxia on formal testing in all limbs, more marked on the right. There was no diplopia, ocular movements were normal, but visual acuity on the right was 6/24 and the optic disk was very pale. Both visual fields showed moderate peripheral contraction. Visual acuity on the left was 6/12. Pupillary reflexes were sluggish on the left and practically absent on the right. The right pupil was larger than the left. The remaining cranial nerves were normal. There was diffuse moderate weakness of the right arm and leg; the arm reflexes were normal and symmetrical. The right knee- and ankle-jerks were brisker than the left. Both plantar reflexes were flexor. Sensory testing was not very reliable, but there was probably some impairment of superficial sensation in the distal parts of the right limbs; vibration sensation was also impaired on the right side, but joint position sense was apparently completely absent in the right hand and foot.

He was admitted to hospital, where radiographs of his skull and spine were normal. There was slight pneumoconiosis. W.R. in blood and cerebrospinal fluid was negative. Lumbar puncture revealed a fluid under normal pressure; protein 40 mg./100 ml.; cells fewer than 5; normal Lange curve. His memory was unreliable. Psychometric testing revealed moderate intellectual impairment.

Electroencephalogram.—This was normal. All activity was of low voltage but alpha rhythm appeared at 8–9 c./sec. and was augmented slightly by overbreathing.

Lumbar Air Encephalogram.—There was symmetrical enlargement of both lateral ventricles; the third and fourth ventricles were normal. Little air was seen over the cortex. The septum pellucidum could not be identified. On the antero-posterior projection a faint linear shadow could be seen which may have been the septum, but on the interaxial projection, which usually gives a good view of the septum, it could not be identified. In the postero-anterior view also the septum could not be seen, but, unlike Case 1, there was no shadow to indicate the confluence of the two lateral ventricles anteriorly.

Progress.—He has been kept under observation during the past two years and air encephalography was recently repeated. He is still working, although under some difficulty, but he is slower mentally. At times he is anxious or depressed, but on the whole his personality has not significantly altered. Psychometry showed that his level of intelligence was dull normal (I.Q. 81) and that there was significant intellectual deterioration. On the recent air encephalogram the septum could not be seen on the antero-posterior or interaxial views. Tomographic cuts, however, have shown that there is a cavum septi pellucidi with very thin and possibly defective walls (Special Plate, Fig. 4); only the lower third of each lamina of the septum could be clearly visualized. In the postero-anterior view it appears as if the lateral ventricles are in communication, but the confluence cannot be clearly identified as in Case 1. With the patient lying on his side an antero-posterior horizontal ray projection (Special Plate, Fig. 5) shows that no air remains in the lowermost ventricle. Lateral horizontal views were not satisfactory, as he fainted during the manœuvre and filling was inadequate; there was no suggestion of the comma-shaped shadow seen in Case 1, and which Dyke and Davidoff (1935) explained is also present when there is a cavum septi pellucidi. It seems, therefore, that there is a cavum septi pellucidi whose walls are very thin so that they cannot be clearly visualized, and also defective, allowing communication between the two ventricles.

Case 3

This man, aged 69, retired from the ring at 32 and was found to be suffering from diabetes six years ago. He had had several hundred fights from the age of 13. For about ten years it had been evident to his friends that his mental faculties were failing. His memory was seriously impaired, there were episodes of gross amnesia and confusion and disorientation. He was childish at times, had little insight, and was content and euphoric. He was nearly always smiling and optimistic. Short journeys often resulted in disorientation. On one occasion, while driving a car, he was involved in an accident and sustained a minor head injury, but was confused and disorientated for several days; he had complete amnesia for the accident itself.

In 1959 he was admitted to hospital with acute appendicitis, and it was then noted that he could not give a satisfactory history and was confused and disorientated.

Examination.—He was found to be confused, disorientated, and unsteady, and he complained of headache. Subsequent investigations showed that his diabetes was satisfactorily controlled on insulin (I.Z.S., 60 units daily). General physical examination showed that he was well preserved. B.P. 120/60. His speech was clear, but he was unsteady on his feet and stumbled frequently. His pupils were small but active; optic disks were normal. There was slight generalized tremor of his upper limbs at times, but power in all limbs was good. He was ataxic on formal testing; fell on the Romberg test; could not stand on one leg or walk heel-toe fashion. There was

no significant reflex or sensory abnormality. Plantar responses were flexor. His peripheral and retinal arteries were not sclerotic.

On admission he did not know what hospital he was in, did not know the day of the week, or the year, and was generally childish and petulant in his behaviour. He would sulk and complain if left unattended, and smile and giggle when anyone approached his bedside. His memories of his boxing career were reasonably retained, but places and boxers he could rarely name. He was talkative and happy.

Radiographs of his chest showed no cardiac enlargement, although there was some unfolding of his aorta. Radiographs of his skull showed some calcification of the falx and of both internal carotid arteries. Blood urea was 31 mg./100 ml. No glycosuria. Blood sugar (fasting) was 156 mg./100 ml. Blood and cerebrospinal fluid W.R. were negative. Lumbar puncture revealed a fluid under normal pressure; protein 32 mg./100 ml.; cells fewer than 5/c.mm.; Lange curve normal. Serum vitamin B_{12} was 248 $\mu\mu$g./ml.

Electroencephalogram.—This was abnormal. There was normally responsive alpha rhythm at 10 c./sec. arising in the post-central areas with short intermittent runs of moderate-voltage 2 c./sec. delta waves bilaterally, predominating in the pre-central leads. This abnormal activity was slightly increased by overbreathing.

Lumbar Air Encephalogram.—There was marked symmetrical enlargement of both lateral ventricles with a few pools of air in dilated sulci over the frontal, parietal, and temporal lobes. In addition there was a well-outlined cavum septum pellucidum (Special Plate, Figs. 6 and 7).

Case 4

This man, aged 33, had started boxing at the age of 13. He said he obtained a professional licence at 15 (giving his age as 16). He had had about 200 professional bouts, and retired when he was 28. He said he had won 150, lost 40, and had been knocked out on five occasions. He spent several years in the boxing booths and had 30 to 40 fights a day. He was a wild type but not unpleasant. He served in the Forces, enjoyed the life, had a considerable following, but resented discipline and deserted. He said he acquired and lost a fortune. He had many friends and possessed considerable charm, but his fighting instincts were a nuisance. There was no family history of nervous or mental disorder. He was a powerfully built man, in excellent physical condition, without any outward abnormality. He admitted that his memory was failing and that he had "thumping headaches" when he worked in a bending condition, but he had never sought medical advice. He felt his sense of balance was uncertain and that he was now intolerant of alcohol. His speech was quite normal, and I could find no abnormality on physical examination.

Blood W.R. was negative. Lumbar puncture revealed a fluid with raised protein content (60 mg./100 ml.); cells 3; W.R. negative; Lange curve normal. Electroencephalogram was normal. Radiographs of skull were normal.

Lumbar Air Encephalogram.—This showed (Special Plate, Figs. 8, 9, and 10) that the ventricular system was normal and a septum pellucidum was present; but it seemed to be absent posteriorly, and a few weeks later encephalography was repeated. On the second examination the septum was clearly seen on the antero-posterior view but not on the postero-anterior view, where the ventricles appeared to be confluent. On the interaxial view the septum could be seen anteriorly but not in its posterior part. An antero-posterior view with the patient lying on his side showed that all the air collected in the uppermost lateral ventricle; no air remained in the lower ventricle. In several antero-posterior erect tomograms the septum could always be seen. Lateral horizontal views were normal, except for the presence of a dense shadow within that cast by the lateral ventricles which may conceivably result from a defect in the posterior part of the septum pellucidum (Special Plate, Fig. 10). It was present in all horizontal projections in both encephalograms.

Case 5

A labourer aged 45 entered the Department of Neurology in March, 1962. He started to box when he was 10, and turned professional at 18. Until he retired at the age of 32 he had had approximately 300 fights in various countries. His wife thought that his ill-health began about that time. The first symptom noted was progressive slurring of speech. He had been drinking heavily for a few years, and there were bouts of depression. However, despite the alcoholism there were long periods of abstention, but his family and friends were puzzled because his speech remained slurred. Two pints of beer would often render it incomprehensible. His wife also noticed that nocturnal urinary incontinence, which she had formerly attributed to inebriation, persisted during his periods of sobriety. In the past ten years he has been a patient in a mental hospital on several occasions. During one such admission, about three years ago, it was observed that he was dragging his left leg when he walked and tended to hold his left arm flexed across his body. He had a perforated peptic ulcer in 1951 and a pleural effusion in 1955. In 1957 a partial gastrectomy was performed. At this time an enlarged liver was noted, but liver-function tests were within normal limits. There was no family history of mental or neurological disorder.

Examination.—On admission to hospital he was co-operative, and his general physical condition was good. He had been working regularly at a steel mill since August, 1961, and his wife informed me that he had taken no alcohol since then. He was morose and depressed. He had a broken nose and one cauliflower ear. His speech was thick and slurred and his gait unsteady. His pupils were normal. There was nystagmus on right and left lateral gaze. Power in all limbs was good, reflexes were brisk and symmetrical, and the plantar responses were flexor. There was some diminution of sensation (tactile, pin-prick, and vibration) in the left leg. B.P. 130/80. He was slow, his power of concentration and his memory for recent events were poor, but he was not demented. He was correctly orientated and his behaviour was quite normal. A speech therapist formed the view that in addition to his gross dysarthria there was slight executive dysphasia. Psychometry revealed a dull normal level of intelligence (I.Q. 85), with no indication of intellectual deterioration. He attributed his slurred speech, unsteady gait, and alcoholism to boxing, and he thought "professional boxing should be abolished altogether." He recalled one episode during his boxing career of 24 hours' amnesia. This included a journey of 50 miles (80 km.) to a neighbouring town, an eight-round contest which he won on points, and the journey home. Blood W.R. was negative. Radiographs of skull were normal. The cerebrospinal fluid protein content was raised (60 mg./100 ml.); cells 4; normal Lange curve; negative W.R. E.E.G. was normal. Lumbar air encephalogram was normal. There was no evidence of myxoedema. Liver-function tests were normal, and his liver was not enlarged.

He died suddenly at work of acute myocardial infarction two months after leaving hospital. At necropsy there were foci of softening on the left side in the cortex of the parietal, temporal, and cerebellar lobes. There was a cystic scar in the anterior limb of the left internal capsule. The meninges were thickened at several sites and the cerebral arteries showed slight to moderate atheroma. The pathological picture did not resemble the restricted form of cerebellar cortical degeneration of alcoholics described by Victor, Adams, and Mancall (1959) in which the main atrophy is largely confined to the vermis. The lesions were probably the result of vascular disturbance, but what part trauma played could not be determined.

Discussion

Accounts have been given of five former professional boxers with symptoms and signs of an organic cerebral

disorder. What clinical diagnosis can be made in each case. Is it related to boxing?

The *first* patient was 45 years of age. His professional career lasted 18 years, from the age of 17 to 35. During this time he had 350 fights, but says he was knocked out on only one occasion. His initial symptom consisted of the insidious development of weakness and ataxia of the left leg. It seems to have been noticed by his friends in the later years of his boxing career and probably had something to do with his retirement. Slurred speech, urinary symptoms, and periodic blurring of vision have since appeared. Although he is slow and euphoric there was no significant evidence of intellectual deterioration and he has continued to work regularly and is not very disabled. However, his speech is very dysarthric, his gait is unsteady, and there is some weakness in the left lower limb with an extensor plantar reflex. There is no nystagmus, no optic or ocular abnormality; the abdominal reflexes are brisk and there is no sensory loss. The disorder has slowly and progressively increased, without remissions or exacerbations, for thirteen years. E.E.G. and C.S.F. are normal. The possibility of disseminated sclerosis naturally arose, but when air encephalograms showed that the septum pellucidum was absent the question of aetiology became more obscure.

The septum pellucidum is a thin, triangular-shaped membranous partition separating the lateral ventricles anteriorly. On air encephalography it is seen as a narrow white shadow, 1 to 1.5 cm. in height, 2 to 3 mm. in width, stretching from the corpus callosum above to the anterior commissure and fornix below.

The first case of absence of the septum pellucidum was reported by Tenchini in 1880, and in the majority of necropsy reports the specimen was obtained from an infant or child with epilepsy or mental retardation, and anomalies of the fornix and corpus callosum were also present (Hochstetter and Sitzber, 1925; Hahn and Kuhlenbeck, 1930; Basu, 1935; and others). It was first recognized on air encephalography in 1933 by Forster. His patient died after a head injury, and at necropsy rupture of the septum pellucidum was observed.

Dyke and Davidoff (1935) were the first to provide a full encephalographic study of a case, and they concluded that the lesion was congenital in nature. Their patient was a woman of 23 suffering from the effects of epidemic encephalitis in childhood, but there was also a story of head injury in infancy. It was the only example they encountered in 2,500 air encephalograms. Sfintesco and Mihailesco (1938) reported the cases of two epileptics with an absent septum on air encephalography. Savain (1946), reporting a case, said that there were then only seven published cases of diagnosis by air encephalography. Later reports include those of Davidoff and Epstein (1955, one case), Vinken and Strackee-Kuijer (1957, two cases), and Bell and Summers (1958, one case). Davidoff and Epstein's patient, an epileptic aged 26, had sustained two head injuries, at 6 and 16 years respectively. The adult case of Vinken and Strackee-Kuijer, a man of 40, had sustained a fractured skull, followed by unconsciousness for 24 hours, and subsequently developed epilepsy. Gibson's (1924) necropsy report was of a man who had died in an asylum with post-traumatic dementia; there were several holes in the septum pellucidum. Perforations of the septum are well recognized and Fig. 11 illustrates one such example, kindly lent to me by Dr. D. B. Moffat, of the Department of Anatomy, Cardiff. Dandy (1931) thought that many of these lesions were not congenital but acquired. He commented on the ragged borders of the orifices and the shreds of tissue often attached to them.

The septum is supplied by blood via the antero-median ganglionic branches of the anterior cerebral arteries. In a necropsy report by Dolgopol (1938) of a case of an absent septum pellucidum, he noted that blood vessels ran vertically in the midline across the common ventricle, from corpus callosum to fornix. The brain was from a woman of 60 who had died with cerebral arteriosclerosis and occlusion of the left axillary and femoral arteries. The vessels in the ventricle were obliterated and Dolgopol considered that he was dealing with an example of resorption of the septum rather than congenital aplasia.

The *second* patient, aged 52, after a boxing career of 20 years during which he had 300 fights and was knocked out on only one occasion, retired at the age of 35. At that time his only apparent disability was defective vision in the right eye due to injury to his optic nerve. Five years later, at the age of 40, he first experienced weakness of his right leg. This has gradually increased and has been accompanied by unsteadiness of gait, slurring of speech, and urinary difficulties. There is moderate intellectual impairment and no significant alteration of personality, and the electroencephalogram and cerebrospinal fluid are normal. Air encephalography in 1960 revealed symmetrical enlargement of the lateral ventricles, and the septum pellucidum could not be seen in any of the standard views. It was repeated in 1962, and with the aid of tomography and additional projections it was shown that there was a cavum septi pellucidi whose walls were thin and deficient. Air passed freely from one lateral ventricle to the other.

The *third* patient, aged 69, was probably in good health until about ten years ago, but it is difficult to know when mental deterioration actually began. He had been retired from the ring for 30 years and his physical condition was good. He now has a progressive dementia, his electroencephalogram is diffusely abnormal, and air encephalography demonstrated considerable cerebral atrophy and a cavum septi pellucidi.

The *fourth* patient, aged 33, is in excellent physical health, there is no abnormality on neurological examination, but he is aggressive and violent, especially when he has been drinking. His E.E.G. is normal, but his cerebrospinal fluid contained 60 mg. of protein per 100 ml. The septum pellucidum is present but apparently deficient posteriorly.

A cavum septi pellucidi, as in Cases 2 and 3, is not so rare. Schwidde (1952) examined 1,032 brains at necropsy and found no case of an absent septum, but in 210 (20.34%) there was a cavity in the partition. Age did not seem to be a significant factor. There are three types of dilatation of the potential cleft-like space of the septum: (1) the walls of the cyst are intact—non-communicating; (2) the cyst opens into the ventricular system, owing to rupture of the cyst wall as a result of increasing intracystic pressure—communicating; and (3) the dilatation of the space results from acquired progressive internal hydrocephalus.

The question arises, in respect of Cases 2, 3, and 4, whether repeated blows to the head have in any way

damaged the septum. In addition, in Cases 2 and 3 we have evidence of cerebral atrophy.

The *fifth* patient, an alcoholic, had permanent dysarthria and ataxia. At necropsy the only intracranial lesions consisted of small foci of softening in the cortex of the parietal, temporal, and left cerebellar lobes and the left internal capsule.

Diagnosis of Perforations of Septum Pellucidum

Whatever the origin of apertures in this membrane, whether they are congenital or acquired, so far as I know their diagnosis during life has never been considered.

Complete absence of the septum, as in Case 1, is readily recognized on air encephalography, especially if, in addition to the standard antero-posterior and postero-anterior projections, lateral horizontal views are also taken. It is possible that large perforations in the septum might also be detected on this projection (Special Plate, Fig. 10).

An antero-posterior view with the patient lying on his side normally shows some of the air trapped in the lowermost ventricle below the septum pellucidum (Special Plate, Fig. 12). We do not of course know whether an aperture is necessarily present if all the air collects in the uppermost ventricle as in Cases 2 and 4 (Special Plate, Figs. 5 and 9). It is possible that the air could all escape into the uppermost ventricle through the normal aperture—the foramen of Monro—but normally it does not. On the other hand, I have known it to do so when there is marked dilatation of the lateral ventricles as in cerebral atrophy. It is conceivable that in such cases the foramen of Monro is dilated or the atrophy has involved the septum pellucidum and an aperture has appeared. Further studies are being undertaken; echoencephalography may prove helpful.

The Punch-drunk Syndrome

It is over thirty years since Martland (1928) introduced this term to medical literature. For many years it was accepted by clinicians all over the world, perhaps uncritically, as signifying a post-traumatic dementia of boxers. Critchley (1957) wrote that he had seen 69 cases of chronic neurological disease in boxers, the majority being examples of the punch-drunk syndrome. He encountered "almost any combination of pyramidal, extrapyramidal, and cerebellar signs."

In the boxing world, however, there are many who think there is no such syndrome. Mr. Jack Solomons is not alone in this. In medical circles, also, doubts about the existence of the syndrome have been raised. McCown (1959a), Medical Director of the New York State Athletic Commission, wrote that between 1952 and 1957 among 9,871 boxers there were 259 knock-outs and 9 serious head injuries requiring admission to hospital; 138 boxers retired or were denied licence—we are not told why. In an average ten-round contest, he states, 1,000 punches are exchanged. Earlier, Kaplan and Browder (1954) reported their findings of a four-year study of 1,043 professional boxers in New York. Ringside observations (one of the writers attended at the ringside once a week for three years), slow-motion photography, and electroencephalography were the three main procedures. Their conclusions are interesting. Clinical observation "failed to reveal any abnormal neurological features, even in those contenders who lost their bouts by a knock-out." Slow-motion cinematography "confirmed one major impression gained at the ringside, namely that most blows to the head were short of their mark or deflected by their opponent." Electroencephalography "primarily serves its purpose in professional fighters in the detection of grossly disorganized electroencephalograms." All the effects of cerebral concussion are reversible, state these doctors.

These New York investigations failed to disclose any examples of the punch-drunk syndrome. "It has never been proved to be a neurological syndrome peculiar to boxers and produced by boxing . . . a slick medical cliché" (McCown, 1959b).

But surely inquiries such as these do not tell us what happens to the brains of professional boxers in later years. One might just as well say that regular medical and radiological examination of young cigarette smokers for four or five years, failing to reveal any examples of pulmonary carcinoma, means that cigarette smoking does not cause that disease. The problem is the delay in the appearance of symptoms. Many doctors have seen the immediate consequences of boxing injuries, but it is the possibility of delayed effects which require study. I have personally seen one young amateur boxer die, the day after a fight, of intracranial haemorrhage following thrombosis of his carotid arteries, and another stricken with a permanent hemiplegia. Such accidents are fortunately rare, and a knowledge of them affords little assistance in any critical examination of the punch-drunk syndrome.

It is clear that we do not know what happens to the brain of a professional boxer. The three published necropsy reports of cerebral atrophy are a meagre pathological basis for the punch-drunk syndrome (Brandenburg and Hallervorden, 1954; Grahmann and Ule, 1957; Neuberger, Sinton, and Denst, 1959). These authors suggest that repeated injury may upset the normal colloid equilibrium of the brain and induce premature ageing. Holbourn's (1943) studies of the mechanics of head injuries led him to conclude that shear-strains set up by rotational forces were the probable cause of cerebral damage. The knock-out blow to the chin in boxing produces just such a rotational movement. Pudenz and Shelden (1946) were able to demonstrate swirling rotatory movements of the brain in experimental injuries in monkeys.

Evidence of this kind certainly suggests that repeated blows to the head may damage the brain, but much more requires to be known before the existence and pathological basis of the punch-drunk syndrome can be established. The diagnostic difficulties are illustrated in the cases forming the basis of this communication. In two of them (Cases 1 and 5) local opinion was that they had been punch-drunk for some years. But in Case 1 we do not know whether the absence of the septum pellucidum was congenital or acquired. In Case 5 the foci of cortical cerebral and cerebellar softening may have resulted from alcoholism.

Future studies should include observations of the condition of the septum pellucidum. Lesions of this membrane may sometimes be traumatic. It is not suggested that such lesions are functionally important, only that if they turned out to be commoner in boxers trauma could then be incriminated.

Summary

An account is given of five former professional boxers, four of whom developed a chronic cerebral disorder in later life.

Two (Cases 1 and 5) were both locally considered to be punch-drunk. However, the former had an absent septum pellucidum and the other was an alcoholic. Necropsy in the latter showed foci of softening in the cerebral and cerebellar cortex.

Two (Cases 2 and 3) suffered from cerebral atrophy. Both had cavum septi pellucidi, and in one (Case 2) the leaves of the septum seemed to be perforated.

One (Case 4) was not physically disabled, but his behaviour was violent, his cerebrospinal fluid protein was raised (60 mg./100 ml.), and air encephalography suggested that there was a perforation in his septum pellucidum.

The possible relationship between these lesions and boxing is discussed. There is a paucity of pathological observations of the so-called punch-drunk syndrome, but before the existence of the syndrome is denied, as in some quarters it has been, follow-up studies of former professional boxers will be necessary.

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STUDIES OF THE RETINAL CIRCULATION WITH FLUORESCEIN*

BY

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[WITH SPECIAL PLATE]

The retinal circulation is unique in being the only part of the vascular system available for direct inspection. Observation of the retina in systemic diseases like hypertension and diabetes, in which the small blood vessels are liable to damage, has become a widely accepted routine. Much can be learned from clinical ophthalmoscopy, which reveals features characteristic of various diseases affecting the retina and its circulation. Finer details have been revealed by pathological studies, particularly post-mortem injection of the retinal vessels (Ashton, 1951) and digestion studies (Kuwabara and Cogan, 1960). These techniques have demonstrated abnormalities in vessels too small to be observed during life, and have provided new information concerning the pathogenesis of exudative lesions of the retina.

A new technique has been described by Novotny and Alvis (1961) which promises to reveal during life some of the fine details previously seen only in necropsy studies. A retinal camera was modified to photograph the passage of intravenously injected fluorescein through the retinal vessels. This method shows details of the blood flow through the retinal vessels, including variable rates of flow in different vessels, and also shows spreading patches of fluorescein which seem to indicate increased vascular permeability in certain areas.

In this paper we report experience based on 60 studies of abnormalities of the retinal circulation using the fluorescein method modified in some minor respects.

Method

The apparatus used was basically the same as that described by Novotny and Alvis. A Zeiss retinal camera was modified for fluorescence photography by the insertion of a blue glass filter (Kodak Wratten No. 47B) in the common pathway of the incandescent and electronic flashlight beams, and a green filter (Kodak Wratten No. 58) immediately in front of the camera body. For normal photography the blue filter could be withdrawn by a control on the side of the camera, and this was also used for a quick check on the position of the eye between fluorescence photographs. This was not always necessary, as with good dark adaptation of the observer and the use of the brightest incandescent-light setting, the fundus could be visualized satisfactorily by blue light alone.

Fluorescence photographs were taken on Ilford HPS film (ASA rating 800) which was force-developed in

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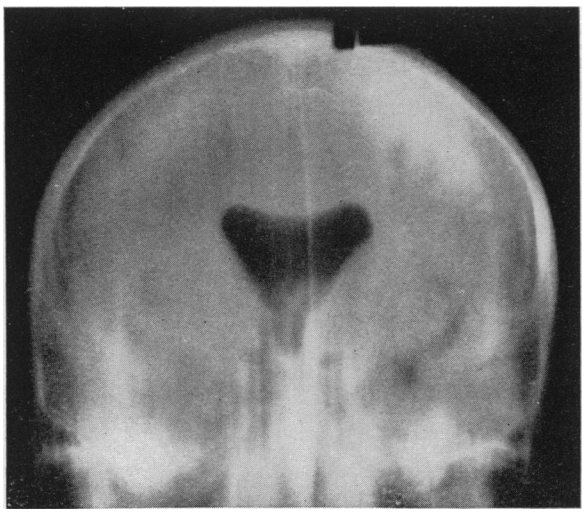

FIG. 1.—Case 1. Antero-posterior tomogram.

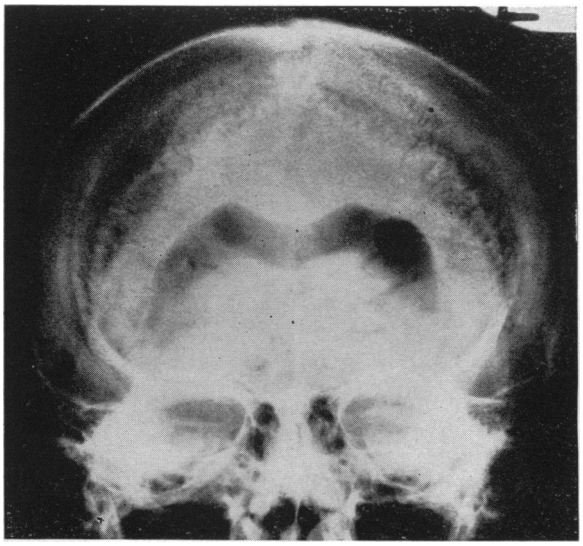

FIG. 2.—Case 1. Postero-anterior view.

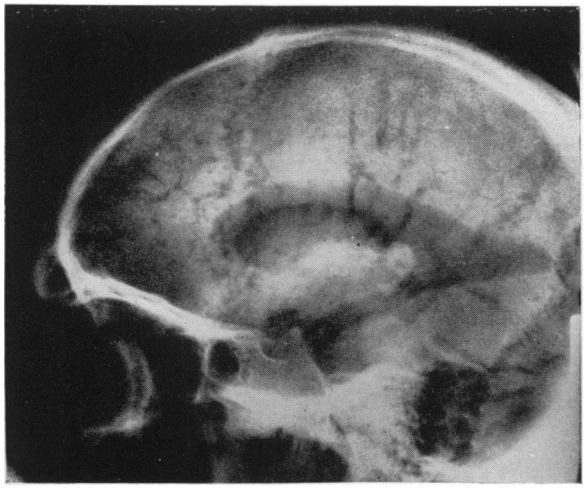

FIG. 3.—Case 1. Lateral horizontal view.

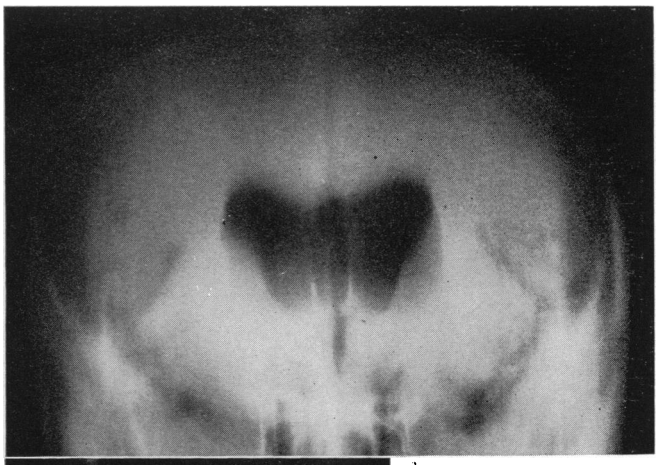

FIG. 4. — Case 2. Antero-posterior tomogram, showing dilated ventricles.

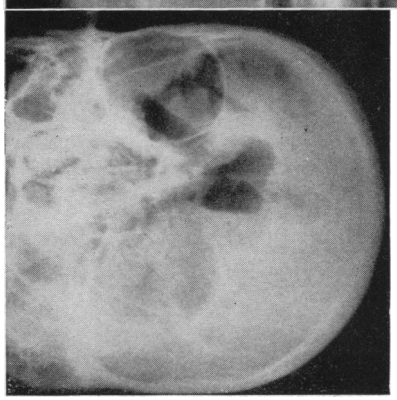

FIG. 5. — Case 2. Antero-posterior view, patient lying on his side.

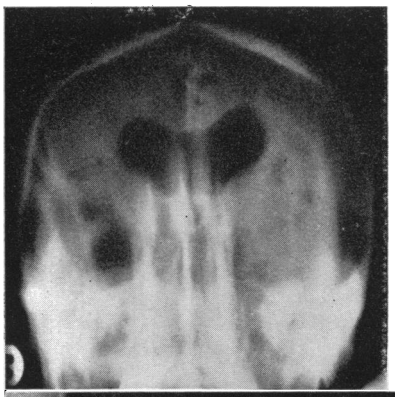

FIG. 6. — Case 3. Antero-posterior tomogram.

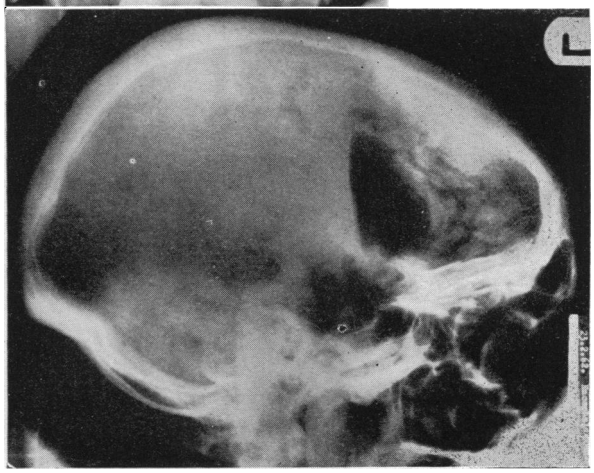

FIG. 7.—Case 3. Enlarged frontal horn.

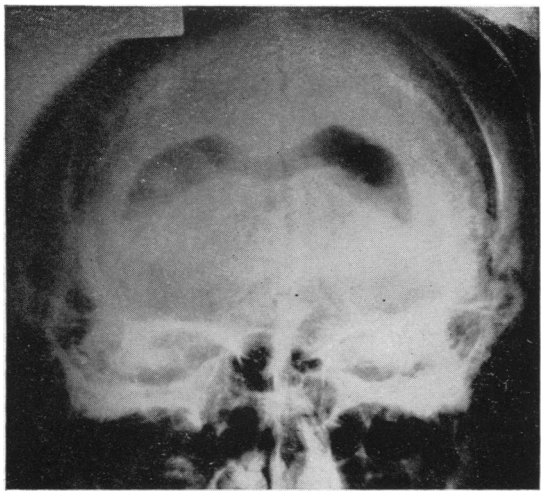

FIG. 8.—Case 4.

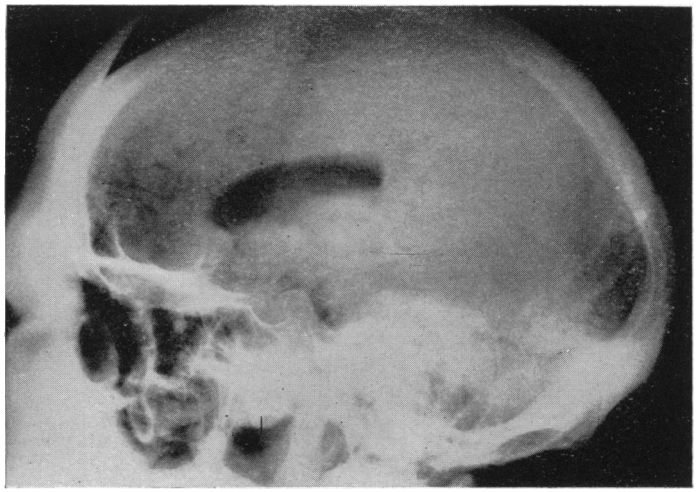

FIG. 10.—Case 4.

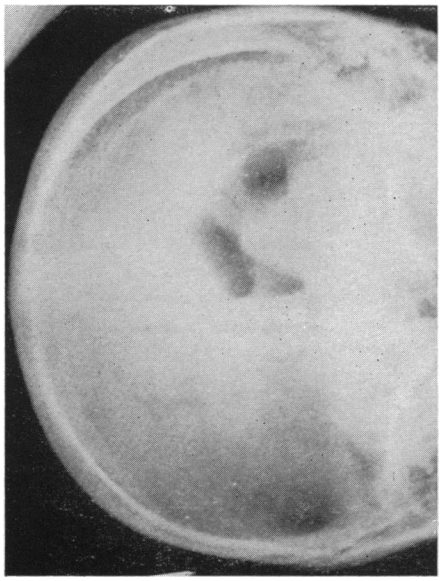

FIG. 9.—Case 4.

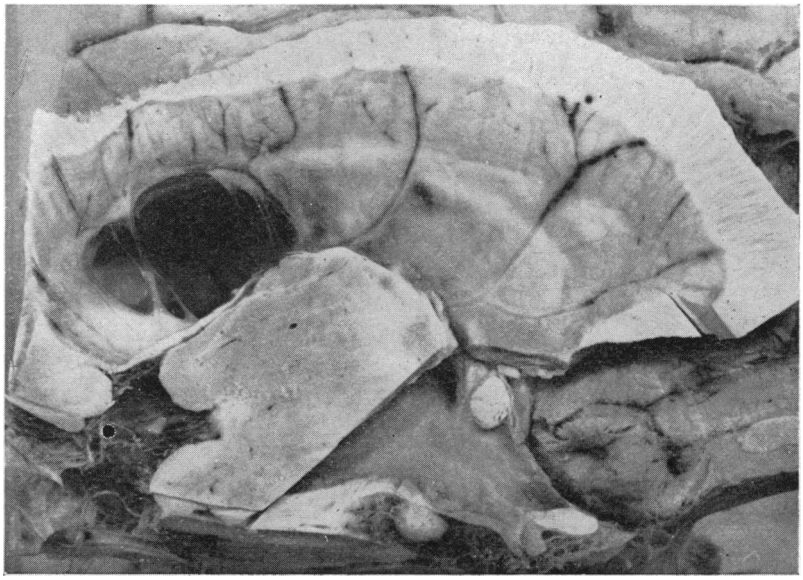

FIG. 11

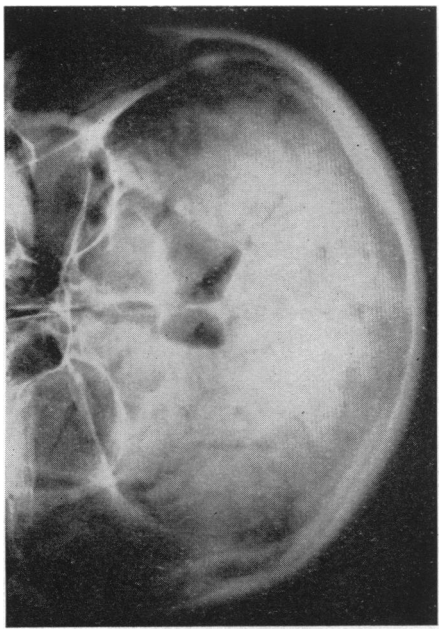

FIG. 12

FIG. 8.—Case 4. Postero-anterior view. Apparent confluence of the lateral ventricles in the midline. Note similarity to Fig. 2.

FIG. 9.—Case 4. Same view as in Fig. 5. No air trapped below septum pellucidum.

FIG. 10.—Case 4. Horizontal view, showing a dense shadow within that cast by the lateral ventricles.

FIG. 11.—Anatomical specimen showing absence of posterior third of septum pellucidum. Note remaining strands of tissue.

FIG. 12.—Normal air encephalogram. Antero-posterior view with patient lying on his side. Some of the air remains below the septum pellucidum in the lowermost ventricle.