# TRAUMATIC ENCEPHALOPATHY ('PUNCH DRUNK') OF PROFESSIONAL PUGILISTS\*

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THE result, immediate or remote, of repeated injuries to the brain of a professional boxer forms a study all in itself, and contrasts with the more usual sequelæ seen when patients have received only one injury in the course of industrial or other pursuits. The problem, moreover, forms one phase of the extremely complicated and highly controversial subject of head injury and its consequence. For purposes of description, the injuries received by pugilists in activities of their profession may be divided into those received during an actual bout, serious enough to cause death immediately or a few hours later, and those which more by their repetition than by their severity lead to slower development of disability during the fighter's career. It is with this latter group that this paper is concerned.

Martland, in 1928, drew attention to the various mental and physical states encountered in examination of former pugilists, and under the title of 'punch drunk' outlined some interesting clinical syndromes. He assumed that the condition afflicted, in mild or severe form, at least 50 per cent. of pugilists of a certain type who had kept at their profession over a certain period. It is not so common among successful pugilists who reach their eminence by rapidly disposing of their less expert adversaries, with but little injury to themselves. Quick, agile, clever boxers who guard themselves well and take little punishment seem to escape. Chiefly affected are the less expert but courageous men who take considerable injury in the hope of wearing out their opponents until they can get in a final knock-out blow, and those used for training, who are accustomed to be knocked down several times a day. Aggressive, hardy fighters who like to give the crowd an exhibition of blows, and who lead the fight to the end, are likely to suffer, and the degree of facial disfigurement is the index of the susceptibility to 'punch drunk.' According to Martland, the early symptoms of the disease are represented by occasional clumsiness of one foot, seen in walking to the corner of the ring, slight ataxia and periods of confusion; at this stage ability to fight may not be seriously impaired. Many cases are mild and do

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not progress, but in the more severe types the legs drag in walking, and tremors, dysarthria, deafness, physical slowing-up and even mental deterioration which requires commitment to an institution may supervene. It is thus possible that a pugilist may be only mildly affected, and may continue to fight to the end of his career, or he may be so disabled that he ultimately has to quit boxing and yet gets no worse in after life. Lastly, a progressive neurological syndrome may appear, putting an end to all fighting, and leading finally to mental or physical helplessness. It is well understood, moreover, that pugilists are not immune to the ravages of syphilis, alcohol, or even epidemic encephalitis, and certainly the incidence of these diseases is no lower among pugilists than in the general population. The point that Martland urged, however, was that the high frequency with which professional pugilists develop crippling disease of the central nervous system of one sort or another suggests a result of the repeated injuries to the brain that they received while carrying on the activities of their profession. Figures which disclose the frequency of disability among former pugilists unfortunately are not available, but the investigation that Martland made suggests that it is relatively high. In the hope of further elucidating the problem, some new material is presented as an argument for the existence of such a condition as 'punch drunk' or traumatic encephalopathy and illustrating some of its very variable clinical features.

# REPORT OF CASES

Case I.—A man, age 24, came to The Mayo Clinic on February 9, 1934, because of weakness of the legs and tremor of the hands. There was no family history of similar disease. He had had influenza in 1922; it was not severe, and he was in bed only a few days.

At the age of 15 he had commenced to box, at first as an amateur. He had become a professional pugilist at the age of 16, in 1926. In that year, in the course of a boxing match, he had received a blow over the left temple, which had knocked him down, and blood had run from his left ear. From 1926 to 1931, he had fought as a lightweight and had averaged about two fights a month. He had made no great headway in his profession, for he had lost as often as he had won and he had not improved his technique greatly. He was not a very good boxer, although he was willing to take considerable punishment in the hope of getting a final victory by knocking out his opponent or wearing him down. He had been knocked unconscious many times, but only for short intervals. One time he had finished eight rounds after being knocked down and had no subsequent memory of it. In 1929, he had been knocked down five times, but he had not been knocked unconscious; the referee stopped the fight. The patient usually made little effort to guard himself, and as a consequence he had a flattened nose and two 'cauliflower' ears. In 1931, he was told by his manager that the chances of his rising in his profession were slight, and, opportunities of getting good matches becoming less and less, he decided, in 1931, to retire from the ring and became a filling-station attendant. He felt well during that time. In April, 1932, the chance of making money from a bout presented itself and he re-entered the ring. He was knocked down twice in the third round, once by a blow over the right side of his jaw and the other time by a blow over the left eye and temple. He took the count of nine and then finished the fight of six rounds. Immediately after the fight, he felt as usual, but a few minutes later he took a drink of water, which he promptly vomited. He continued to vomit and retch, even on an empty stomach, and an hour after the fight, when attempting to go home, he reeled and staggered as if drunk. He managed to make his way home, and went to bed, but slept poorly because of nausea. Finally he slept and awakened at 10 a.m. When he attempted to arise he found himself very unsteady and was unable to balance himself. He was able to walk after a fashion, and actually had more difficulty in equilibrium than during the previous night. After consulting a physician, he returned to bed, where he stayed for three days, feeling nauseated and complaining of diplopia. He improved somewhat after this rest, and again he arose, but, while the diplopia had disappeared, he was still unsteady, although very much less so than previously. In two weeks he returned to his duty as a filling-station attendant, still slightly ataxic.

Two months later, in June, 1932, chance of another fight had come to him, and, in spite of his poor balance, he thought to train for it and overcome this difficulty. While in training, his legs seemed to get worse and he found that he tired very readily. He seemed to be fast enough on his feet in the ring at the beginning, but was unable to endure any considerable length of time, because of a sense of fatigue of his lower extremities and a tendency of them to drag. He had given up the idea of fighting.

From that time up to his admission at the clinic (1934) his condition had changed little. When I first saw him he still had trouble in walking, in that his feet seemed to drag. The right foot was worse than the left and he wore away the toes of his shoes. The sense of balance was not normal, but slightly better than it had been; yet he frequently staggered, more at times than at others. By conscious attention to walking he would pick up his feet better than he usually did, but he soon relapsed into a dragging gait. His mother complained that he was more nervous since his last fight, a little more forgetful of names and details, and that his hands shook. Under excitement, he tended to have a general tremor.

The patient was well developed and muscular, weighing 120 pounds (54.4 kg.) and showing obvious signs of his previous profession in the form of thickened auricles and flattened nose. His systolic blood pressure was 130 mm. of mercury, and his diastolic 76. He walked slowly and unsteadily. His lower limbs were stiff and slow, the right being the worse of the two. At the same time, he staggered on turning suddenly and was somewhat ataxic. He held the right arm flexed when walking, and stiffly at his side, and did not swing it like the other. He hopped clumsily on the right foot, but did better with the left. The toes of both shoes were well worn down. There was a coarse, pill-rolling tremor of both hands, more marked on the right. Slight tremor of the head was present, but speech was unaltered. Speed was diminished on the right side, in both the upper and lower extremity, especially for rapid movements like drumming with fingers on a table. All tendon reflexes were greatly exaggerated, but there was no Babinski's sign on plantar stimulation. Romberg's sign was positive, and slight, horizontal nystagmus was present. Facial expression was normal and labile. Urinalysis and examination of the blood disclosed nothing abnormal, and the flocculation test for syphilis performed on the blood was negative. X-ray studies of the skull gave no evidence of fracture or other abnormality.

## COMMENT

There is but little doubt that the severe beating received by this pugilist in his last fight was responsible for the chain of circumstances which followed. Although he finished the fight, the symptoms which appeared almost immediately thereafter suggested at least moderately severe injury to the brain, from which he made very far from complete recovery. It might also be

suspected that his previous experiences had altered him so that he was an easy victim in his last fight, but of this there is no evidence, and he attributed all his difficulties to his last and futile appearance in the ring. The unbroken chain of cause and effect is very evident in this case, and it is to be noted that in nearly two years the condition had not become worse nor was there much improvement beyond a certain point. Clinically, the picture was a mixed one, suggesting multiple lesions involving the cerebellar, corticospinal, and basal ganglia systems of motor control. No specific nervous syndrome appeared, such as Parkinson's disease, but rather a medley of scattered and incomplete lesions of the brain.

Case II.—A man, age 30 years, came to The Mayo Clinic on January 9, 1928, because of difficulty in walking and in speech. There was no history of any similar condition in his immediate family. He was married and had one child who was living and well. He had had practically no illnesses before the condition concerning which he made his complaint, although he admitted having had minor infections of the respiratory tract, which at no time had necessitated confinement to bed. He denied having had syphilis. He had commenced boxing at 16, in 1914, and shortly thereafter he had become a professional pugilist, fighting more or less continuously up to 1921. He had ranked as a second-rate boxer, and although he had demonstrated plenty of courage and had taken considerable punishment, he never had reached professional eminence. He had the usual broken nose and thickened ears of his trade.

On March 4, 1921, he was knocked out in a fight. He was unconscious during the count of ten, and unable to get up a few seconds later. He mentioned that he had received the blow on the left side of the lower jaw and that it was sufficient to put him out quickly. He walked out of the ring, however, rode home in a street-car, and, angered by his defeat, proceeded to get drunk, but finally went to bed. The next day he seemed normal, and then had started training for the next fight which was to take place 16 days later. During this period of training he noticed that, when he was skipping rope, his right leg tended to catch on the rope, but he took little notice of it at the time. In the subsequent three fights which he finished, he had been winner in the first, and had fought the other two to a draw. The fourth fight was in June, 1921, and it proved to be his last. He was knocked down by the first blow of his opponent, which was a moderately hard one, and when he attempted to get up he was dizzy, had a severe headache, and promptly gave up the fight. While walking home, he found that the difficulty with his right leg had become decidedly noticeable; both lower limbs felt numb and they dragged as he walked. He decided, after this, that he was no longer fit to fight in the ring, and became a labourer in a steel mill. During the first month that elapsed after his last fight, and while he was working in the mill, the stiffness and slowness of movement of his lower extremities increased. This was more marked on the right side, for, as time went on, he wore out two soles of the right shoe to one of the left. Walking became increasingly difficult; he noticed that his voice had become nasal, and that articulate speech was becoming more and more indistinct. He continued at the mill about 18 months, when he was discharged because his infirmity had become obvious.

In the latter part of 1923, movements of the right arm became stiff, clumsy, and awkward. Spasmodic contractions of the muscles of the right arm, right leg and back appeared and gradually increased so that in walking he felt a tendency to be pulled backward. He had occasional parietal headaches lasting from one to four hours, and his powers of mental concentration, attention and memory were considerably reduced.

Through the subsequent four years, his difficulties slowly and gradually increased. He experienced more and more difficulty in walking and in speech, as well as increasing spasms of the muscles of the extremities and trunk. At the time of his examination, however, he still was able to walk alone, and he could feed and dress himself.

The patient was well nourished, weighed 130 pounds (59 kg.) and there were no abnormal physical findings apart from those suggesting disease of the nervous system. Urinalysis, blood count, examination of eye grounds, and the Wassermann test revealed nothing unusual, and the same result was obtained from X-ray studies of the skull and cervical portion of the spinal column. Neurological examination gave a complex picture.

The man was of unusual muscular development, probably because of his profession and training, and this rendered more conspicuous the various contractions and spasms continuously at play in his muscles. In walking, his gait was unsteady and spastic; the spasticity was more marked on the right than on the left side. More conspicuous, however, were the continuous, irregular spasms of the muscles of the right arm and back. These were sufficiently violent to pull the man backward and to the right, with the right arm jerking and his hand slapping his thigh. These contractions were rapid, ever changing and quickly repeated. When the right arm was extended forward, fully supinated, continuous jerking occurred, tending to flex and pronate the arm. When, on the other hand, the arm was drawn behind the trunk in full pronation, these movements ceased. While the patient was seated the muscles of the back were continuously jerking, pulling him backward and to the right. The muscles of the neck and face were quiet. There was no paralysis, but all of the man's movements were slow and clumsy, except when he was running and jumping, which acts he performed, on the whole, better than slow walking. When he was asked to perform rapid movements of the fingers, these movements were slow on the right side, but in the left leg and foot the movements were equally slow and spastic. The tendon reflexes on the left side were exaggerated, and plantar stimulation on the left side produced an extensor reflex. Stroking the lips with a hard object produced a sucking reflex (Oppenheim's sign). Tests of coordination, applied to the upper and lower extremities, did not disclose marked dysmetria or dyssynergia, but all tests were interfered with by the irregular, spasmodic contractions. There was no nystagmus. Speech was very indistinct, slow, laboured, grating and nasal in tone, and the breath escaped through the nose while the man was talking, apparently because of some weakness of his soft palate. During animated conversation, he had the tendency to break into a silly guffaw. This, with the mouth wide open and with the tone of voice, seemed to suggest the spasmodic laugh seen in pseudobulbar palsy or progressive lenticular degeneration. Mentally the man seemed childish, and below par even when his social standing and previous education were discounted. He was written to and a reply was received dated March 22, 1934. In this letter he stated he was still able to get around, had not become any worse, and, if anything, there was a slight improvement during the six years that had elapsed since his examination at the clinic.

#### COMMENT

Again, as in Case I, this patient was a plucky and aggressive fighter, but of no great eminence in his profession; he was a lightweight, who started to fight when quite young, took plenty of injuries, and ended disastrously. However, the clear-cut chain of events existing between cause and effect is not so obvious in this case. All that is known is that after seven years of strenuous fighting and during the course of his career, between one bout

and the next, symptoms appeared insidiously, increased in severity, and put an end to his fighting. Even after he had left the ring and had tried other pursuits, his difficulties increased steadily up to the time he appeared at the clinic, seven years after his last fight. Argument could be advanced in this case that his trouble was due to a chronic phase of epidemic encephalitis and that the initial episode, in the form of a mild respiratory infection, had passed unnoticed. In Parkinson's syndrome of epidemic encephalitis the initial episode may be absent or so mild that it is even forgotten, and yet the disease appears and progresses steadily to complete disability. It is, however, begging the question to assume that this patient had epidemic encephalitis, for this is incapable of proof, and it is just as plausible and reasonable to assume that repeated injuries to his brain led to a progressive, bizarre syndrome of disease of the nervous system. Moreover, the clinical picture was exceedingly complicated and did not resemble at least the more usual sequelæ of epidemic encephalitis. There was, first, evident dystonia, producing grotesque muscular contractions, but the corticospinal motor system was affected also, as shown by the spasticity and Babinski phenomenon. There was also the dysarthria and the spasmodic laugh to account for, and altogether the picture was that of scattered and diffuse lesions of the brain, resembling no specific clinical syndrome. In the final analysis, and unsupported by necropsy, it can only be a matter of opinion that this patient's trouble was due to his pugilism, but in view of other cases reported a strong feeling remained that it was actually due to this.

Case III.—A man, age 28, came to The Mayo Clinic first on March 23, 1923, because of stiffness and dragging of his legs in walking, unsteadiness of gait, and indistinct speech. There was no family history of similar difficulty, and the patient had had no previous illnesses requiring confinement to bed. He admitted having had gonorrhæa several times, and that he had had a sore on his penis at the age of 17, the nature of which was uncertain.

He had started as a professional pugilist at the age of 18, and had fought steadily, up to the time of his admission, i.e, for ten years. He had had as many as three fights in one week, and five fights in one month; in one year he had fought 22 times. He had served in the army during the war and had boxed several times. While serving as a mess waiter, just before his discharge in 1919, he noticed that his left hand and foot were clumsy; he spilled glasses of water because the left foot hit the ground awkwardly and the hand shook. He thought nothing of it at the time. On discharge from the army he immediately had gone back to professional boxing, and had continued to hold a championship until he lost it because of an adverse decision. During the latter part of 1919, while walking with his friends, they jeered him because his left leg hit the ground with a slapping noise, and for the first time he realized there was something wrong. Nevertheless, this had not interfered with his ability as a boxer at that time. In 1920 episodes of staggering while walking in the street appeared, and his speech became thick, so that he was accused unjustly a number of times of being drunk. Just before one fight a ring attendant, after talking to the patient in his dressing room, insisted on calling the manager to stop the fight, because he thought the fighter was intoxicated. The patient had won that fight after a terrific battle. Gradually, during 1921 and 1922, the increasing stiffness of the left arm and leg and the unsteady gait became more obvious. He would be slow, awkward and clumsy in the first few rounds, would spar for time, then, becoming heated and loosened up under the influence of exercise and excitement, would finish as well as ever. The left arm would go into clonus, puzzling his opponent by its unexpected and grotesque trepidation, and then the right arm, which never had become affected, would do its deadly work. Nevertheless, remorselessly his disease advanced, speed and precision were slower in appearing with each bout, and a few days before the patient's first visit to the clinic his career ended in an inglorious fashion. He had been pitted against a boxer whom he easily had beaten before, and had expected to beat again. In the first few seconds of the first round, while still stiff and awkward, he was given a slight push, stumbled and fell and was unable even to get back on his feet again. While the crowd howled in derision his seconds threw in the towel and his last fight was over.

As a boxer he had been of the aggressive type, fast enough in action, but he had disdained to guard himself much, and had carried the battle into his opponent's quarters continuously. He stated that he hardly ever had felt the blows he received, never had been knocked out, but had received many terrible beatings, winning in the end by his superior stamina and ability to stand up under heavy punishment. He never had to go to bed after a fight, in spite of many cuts and bruises received around his face and head. He belonged to a certain school of prize fighters and was the only one affected among his crowd. As a possible explanation was the fact that they were all fast, clever boxers, hard to reach with a blow, and of them all he was by far the most disfigured and had a particular reputation for being a hard fighter. He had had innumerable Wassermann tests of his blood, all with negative results, and at least several examinations of spinal fluid, not one of which had disclosed evidence of syphilis.

When he was examined in 1923 his disability was obvious. His speech was thick, muffled and hard to understand; there was a rasping, laboured quality in the voice. His gait was spastic. The spasticity was marked on the left side, and he dragged his legs, wearing out the toe of his left shoe. There was no marked weakness. The grip of the left hand was weaker than that of the right, but the outstanding feature was the slowness and clumsiness of the fingers, hand and arm. In certain positions the left arm went into clonus. Patellar clonus was also present, and there were a few strokes of ankle clonus on the left. Tendon reflexes were everywhere enormously exaggerated, more so on the left, and on that side a Babinski phenomenon could be elicited. The jaw reflex was exaggerated, and sucking reflexes were present on stroking the lips. The man was unable to hop on the left leg, and on performing active movements, such as shadow-boxing, he was so stiff and clumsy that he nearly fell, and his left arm was slow and awkward. The right arm and hand seemed but little affected. There were no sensory changes, tremors or nystagmus; facial expression was mobile and pupillary reactions were normal. He seemed below par mentally; his general intelligence and his memory were somewhat reduced. Facial disfigurement was extreme; both ears were thickened and shapeless; his nose was flattened; his eyebrows were thickened and full of scars, and his lips were irregular and distorted; many front teeth were missing. General physical examination disclosed nothing more. The Kolmer modification of the Wassermann test, applied to the blood, gave negative results. On examination of the spinal fluid, the Wassermann reaction was negative; the Nonne test was negative; 1 small lymphocyte was present per cubic millimetre, and the colloidal benzoin test gave no curve.

A diagnosis was made of lateral sclerosis of the spinal cord, and some doubt was expressed as to the origin of the condition. Syphilis was suspected because of the history, and a course of antisyphilitic treatment was advised on empirical grounds. A very poor prognosis was given, since it was expected that progression of the disease could be expected. The patient was lost track of thereafter, until, for the purpose of the present investigation, he was written to, and in response he reappeared on

March 7, 1934, almost exactly eleven years after his first examination. To our surprise we found that time had dealt gently with him. He seemed in no way worse, and beyond acquiring a certain amount of obesity he presented an identically similar picture to that encountered on his first examination, and was, if anything, somewhat improved, discounting the increased weight and lack of training. His disturbance of speech and gait were as conspicuous as before, but he had not deteriorated any more, physically or mentally. He had taken no antisyphilitic treatment in the meantime, and had added to his collection a few more negative Wassermann tests on his blood and spinal fluid. He had been engaged in various occupations, mainly sedentary, had been moderately successful, and seemed at peace with the world.

#### COMMENT

Here the picture is somewhat different from that encountered in the other two cases. The patient, a high-grade pugilist, developed symptoms during a period of four years, coincident with his heaviest fighting. The symptoms slowly progressed up to his final, ignominious failure in the ring, and then, for eleven years thereafter, his condition remained much the same as it was when he ceased fighting. The picture was also different from that presented in Cases I and II; it was mainly that of injury to the corticospinal motor tracts on both sides; hence the original impression of lateral sclerosis. The man's frank admission that he had contracted venereal disease further led to the presumption that he had syphilis, but this was never proved, either serologically or by any clinical finding that was specific for syphilis of the central nervous system. It is reasonable to exclude this disease in its entirety, and more logical to blame the innumerable injuries to the brain received at the height of the patient's career.

Jokl and Guttman have recently reviewed the subject of symptoms referable to the central nervous system presented by pugilists. They divide the symptoms into three classes: those appearing in the course of the actual bout; those coming on some hours or days thereafter, and those appearing in the later life of the pugilist. The latter they divide into mental and physical. Mental deterioration, they think, can afflict patients who are predisposed to it even before they enter the ring, but they also think a definite dementia affects former pugilists as a result of cerebral trauma received during their careers. On the physical side, Jokl and Guttmann describe changes in tendon reflexes, dysarthria, disturbances of coordination and anomalies of speech.

## SUMMARY AND CONCLUSIONS

Three cases of chronic disease of the nervous system, affecting professional pugilists, have been described. In the first, the maximal functional disability appeared immediately after a fight, improved somewhat during the next few weeks, but thereafter remained at a standstill. In the second case the progressively crippling syndrome appeared insidiously during the

last few fights of the patient's career. His difficulties had progressed up to the time of his examination four years later, but apparently had not become worse, and were, if anything, improved in the subsequent six years. third patient also gave evidence of the insidious development of an affliction of the central nervous system appearing five years before he ceased to fight and progressing up to the time of his leaving the ring. However, after the patient's fighting career was over his condition remained stationary or perhaps improved slightly. The clinical picture in each case was very complex and it differed in the one case from the others. The only agreement, however, was in the suggestion of diffuse or scattered lesions of the brain affecting different systems at one and the same time. It is assumed that these patients' difficulties resulted from repeated injuries to the brain received during their pugilistic careers. If probabilities are to be discussed, the first patient was without reasonable doubt injured during his last fight and his subsequent difficulties resulted from it. In the case of the third patient, his difficulties appeared during his fighting career but stopped progressing as soon as he left the ring. The probability here, that his disease was connected with his occupation, is good. The case of the second patient must remain in doubt, for there was not the clear-cut association between his occupation and his disability. Since postmortem study is lacking, the exact pathological mechanism of these cases is to date unknown. The fortuitous occurrence of epidemic encephalitis or syphilis of the nervous system during the pugilist's career cannot be excluded as a cause of the difficulty. However, the frequency of occurrence of conditions of this kind as reported by others among people who followed the profession of pugilism makes it seem very likely that their profession led to their ultimate disablement. It is to be hoped that in the future more statistical data, backed up by pathological studies, may put these conclusions on a firm basis.

# REFERENCES

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