important, because if there is a strict medical control of amateur boxers the possibility of extensive and repeated neuronal destruction will not exist and the argument indicting boxing on account of the cerebral end-results will therefore be groundless and unnecessary. Thus according to the London Amateur Boxing Association laws, boxers who are repeatedly knocked out, who suffer lengthy amnesias on several occasions, or who are severely concussed are excluded from further contests.

There is another aspect of the medical service, and this is to explore all possible measures that will help to safeguard the non-professional boxer. As well as our report on the use of the E.E.G. as a prophylactic aid, other authors have published their results (Ardito, 1954; Soeder and Arndt, 1954; Larsson *et al.*, 1954; Medved, 1955; Pampus and Grote, 1956). Kaplan and Browder (1954) made an extensive study of professional boxers, and they, like most other authors and ourselves, found little or no evidence of permanent brain changes. Records taken soon after a bout in which the boxer received a lot of head blows were usually normal, and a few men who were followed for up to three years whilst boxing regularly developed no E.E.G. abnormality. Permanent cerebral deficits, although they no doubt occur, seem to be very difficult to detect by the methods of investigation at present available, and are in any case probably rare. Kaplan and Browder (1954), when evaluating the relative merits of the information obtained from these investigations, arrange them in the following order of usefulness: past medical history, physical fitness, past boxing performances, and finally the E.E.G. With this we would agree, and it is obvious that a decision concerning a boy's boxing future can be reached only by considering all the available evidence.

At the moment, all the possible information concerning boxing head blows must be amassed, and observations on the amnesia of the concussed boxer is of considerable importance. The retrograde and post-traumatic memory losses are both indications of the severity of the blow (Russell and Nathan, 1946), but they must be assessed with reference to events observed by onlookers. Thus, a lengthy amnesia without the external appearances of loss of consciousness should be considered equivalent to a state where the boxer is obviously unconscious.

The grading of head blows received in the ring is not easy, and we have attempted to provide a classification that seems to include most cases of concussion. It is possible that as we acquire more knowledge some modification may be necessary, but at the moment the recording of both objective and subjective information is justified.

Much more information on boxing amnesia, however, is required, and, as well as the immediate retrograde type, the behaviour of more distant pre-traumatic memories may be important (Williams and Zangwill, 1952). It seems possible that assessment of the total traumatic amnesia will be more important than the neurological examination, the E.E.G., or other tests in detecting the first signs of the deleterious effects of boxing upon the brain—if such will ever result from amateur boxing.

The increase of medical interest in amateur boxing that is evident all over the world is of considerable importance. It is helping to eradicate the prejudices and misconceptions that dominate most discussions on the subject. At the same time, it will improve and popularize the medical welfare of youths who indulge in boxing as a recreation and in particular will protect them from chronic cerebral damage, if this can be proved to be a consequence of amateur boxing. The medical problems of the amateurs are under constant review and new knowledge acquired from the sport or from elsewhere will be made use of where appropriate.

With regard to the more fundamental objections to boxing as a sport, we have nothing to add to previous statements (Blonstein and Clarke, 1954), except to comment upon the interesting fact that the emotions said to be displayed in the ring and condemned by the opponents of boxing are often evident in their belligerent verbosity.

Summary

Boxing injuries encountered in 3,000 London Amateur Boxing Association contests during 1955–6 are assessed.

Only 29 boxers (0.58%) were severely concussed or knocked out more than once. The results of neurological and electroencephalographic examinations were normal in all. Other injuries were very few.

A classification of boxing head injuries is suggested and the importance of traumatic amnesia stressed.

Doubt is cast upon the possibility of chronic cerebral changes in amateur boxers who are protected by an adequate medical service.

In view of the limitations of clinical, electroencephalographic, and other investigations the evaluation of memory loss may be of great importance as a prophylactic aid.

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A FATAL INJURY AT BOXING (TRAUMATIC DECEREBRATE RIGIDITY)

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A fatal outcome from a punch on the head at boxing is fortunately a rare event, and when it does occur a sportsman who was fighting fit has been lost. The clinical and post-mortem findings in these fatal cases provide a continuous set of observations of the victim from before the beginning of the contest to the post-mortem examination. As the objective of a combatant at boxing is to punch the opponent on the head with sufficient force to drop him to the mat and cause him to remain there till he is counted out by the referee, it is evident that the victim of a "knock-out" drops because he has suffered a concussion of the brain. It is expected that the concussion will last just long enough for the count, and that recovery will be full and complete after this.

In the occasional fatal case the questions arise: has the victim died of concussion? and has he sustained additional injuries to the brain which caused his death? These are questions which could be decided by a post-mortem examination. The fatal accident also provides

the opportunity for investigating the mechanics of a punch delivered by a gloved hand on the head of an opponent who was prepared for the attack, in which the direction and the force of the punch are observed, and the condition of the victim is observed before, at the time of, and after the blow. If concussion be the cause of death, here is a complete picture of the condition. If intracranial haemorrhages have resulted from the blow, the part that these play in determining the fatal outcome can be decided. These features were well presented in the case under review.

Case History

On June 21, 1956, at an amateur boxing meeting open to boxers who had not won a trophy at an intermediate or open meeting, two entrants met for a bantam-weight—118 lb. (53.5 kg.) and under—contest. The victim of the "knock-out," Raymond Perera, was 114 lb. (51.7 kg.) and his opponent 116 lb. (52.6 kg.). Both contestants were 20 years old. The judge, Mr. A. B. Henricus, gave me his observations. The knock-out was delivered shortly after the first minute of the first round. Up to this stage the deceased, Perera, had been leading "lefts" at his opponent and the blow which knocked him out was the first struck by his opponent. It was a right cross which caught Perera on the left side of his chin. Perera appeared to be stunned by the impact, and gradually went dizzier, and slowly slumped down till his bottom reached the canvas. Then he rolled over to one side. He did not appear to be unconscious, and he was raised by his two seconds and helped to walk to his corner.

Mr. A. I. Obeysekera, who was the time-keeper at this meeting, was watching this match between rounds. He observed that when Perera reached his chair he was lying with his limbs fully extended and his jaws clenched, and that he was breathing deeply and audibly like a man sound asleep. To Mr. Obeysekera these appearances were unreal and so unlike those of the victim of a knock-out that he wondered whether the victim was shamming. He had noticed that the victim had never spoken from the time of the knock-out and that he still remained rigidly extended when being lifted into the stretcher for transport to hospital. The knock-out had occurred at 6.30 p.m. I was on duty at 7 p.m. in the casualty ward of the General Hospital, Colombo, when Perera was brought in. He lay on his stretcher breathing deeply as if in deep sleep, his lower limbs were rigidly extended at the hips and knees, and his upper limbs lay by the side of the body rigidly extended at the elbows, with the forearms fully pronated and the fists clenched. Passive movements were resisted by the rigidity, and if the resistance was overcome by force there was movement of the limb, restoring it to its original position. Perera was unconscious, and made no response to a pin tickling the inside of the nostril or pricking the skin of an eyelid.

The left pupil was widely dilated, the right pupil was small. There were no external injuries, no bruises, and no haemorrhage from the nostrils, mouth, or chin. The temperature was 97.4° F. (36.3° C.), pulse full and regular 104, and respirations 20.

At 8 p.m. Mr. C. P. De Fonseka made a trephine hole in the left temporal fossa, and found the dura mater bulged with subdural blood. Incision of the dura mater evacuated 2 oz. (57 ml.) of fluid blood. A second trephine hole in the right temporal fossa showed no extradural or subdural blood. At 11 p.m. the pulse was 88, respirations 38, and temperature 102° F. (38.9° C.). Perera's condition continued to deteriorate and he died at 1.30 a.m. on June 22.

I obtained permission of the coroner to make a post-mortem examination on June 22 at 11 a.m. On turning down the scalp there were diffuse films of coagulated blood on the subpicranial surface of the scalp and on the pericranial surface of the skull extending upwards from the temporal fossae from the surgical incisions in the scalp.

When the vault of the skull was removed, the dura mater covering the whole of the convex surface of the left cerebral

hemisphere was blue from subdural blood, and when the dura mater was incised a thin film of fluid subdural blood covering the convex surface of the left cerebral hemisphere flowed away. The blood was pooled posteriorly, where it bathed the left occipital lobe. The brain was not compressed by the blood. There was no subdural blood over the right cerebral hemisphere. There was a film of subarachnoid blood coating the pre- and post-central convolutions of both cerebral hemispheres extending a little in front of and behind these convolutions as well.

On the left side this film of subarachnoid blood continued into the Sylvian fissure over the island of Reil, and over the outer surface of the left temporal lobe. The medial and inferior surfaces of the cerebral hemispheres had no films of subarachnoid blood. There were no cerebral lacerations. There was about 3 oz. (85 ml.) of dark blood in the posterior fossa, but the cerebellum and the brain stem showed no subarachnoid films of blood and no lacerations, and it is likely that this blood had flowed into the posterior fossa from above.

Sections of the cerebral hemispheres showed no intracerebral haemorrhages or lacerations, but the white matter of both hemispheres was studded with the pinhead orifices of dilated vessels. There were no haemorrhages at these orifices. Sections of the brain stem and the cerebellum showed no haemorrhage or laceration at these sites.

Discussion

The decerebrate rigidity of the victim of this bout presented some striking features. That it came on with the receipt of the blow was evident from the observation that the victim appeared to be stunned and to get dizzier, and that he then slowly slumped down till his bottom touched the canvas, an unusual fall for a knock-out blow which would be explicable on the basis that the decerebrate rigidity had broken the fall. Then, too, the "automatic walk" back to the corner, when he had been picked up and supported by his two seconds, is another phenomenon referable to the state of decerebrate rigidity. The poker-back posture when he was laid down was clear evidence of the state of decerebrate rigidity, as was the maintenance of this posture till the time of death. Experimental transections of the mid-brain have established that decerebrate rigidity is the result of a lesion between the vestibular and the red nuclei. The post-mortem examination showed that there was no visible lesion in the mid-brain, but it did establish that there had been an appreciable displacement of the cerebral hemispheres within the brain case. The subarachnoid haemorrhages on the convex surfaces of both cerebral hemispheres across their waists between their frontal and occipital poles is clearly the result of an upward drive of both cerebral hemispheres against the cranial vault from the blow on the chin being transmitted upwards through the condyles of the mandible. The diffuse subdural blood covering the convex and inferior surfaces of the left cerebral hemispheres must have been from a traction rupture of veins in the subdural space passing from the dura mater to the inferior surface and the lower convex surface of the left cerebral hemisphere. As the punch was delivered on the left of the chin this would account for the greater upward shift of the left cerebral hemisphere rupturing the veins in the subdural space on this side.

The post-mortem findings afford clear evidence of an upward shift of both cerebral hemispheres, and it would be logical to deduce that this had determined the strain in the mid-brain between the vestibular and the red nuclei which had caused the condition of decerebrate rigidity.

In this case the site of the strain in the mid-brain is postulated with precision because of its causing the condition of decerebrate rigidity. The theory that concussion is the result of a strain in the mid-brain just above the red nuclei cannot be postulated with the same certainty, but the evidence of this case in which the strain occurred at a slightly lower level is good evidence for the mid-brain lesion of concussion.

Brain Injuries at Boxing

Boxing is a well-established sport, and the rarity of fatal accidents at boxing and the absence of apparent ill effects in combatants encourage its continuance. Although the head may well receive many punches during a bout, the brain escapes damage. What is the reason for this? It must be that the positioning and tensing of the head and neck, when it is about to receive a punch, prevents the brain from being displaced. In the occasional instance where this protective position is ineffective, the brain does shift within the brain case, and the effects of this are to be assessed by examining the fatal case. The present case details such an examination. The evidence in this case permitted a reconstruction of the mechanics of the injury, and this reconstruction throws light on the mechanics of concussion.

Summary

The case is described of a boxer aged 20 who was knocked out by a blow on the chin and died in consequence. Between the knock-out and the intracranial operation the patient was in decerebrate rigidity.

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ANTIGENIC ACTIVITY OF BRITISH
POLIOMYELITIS VACCINE

A REPORT TO THE COMMITTEE ON LABORATORY INVESTIGATIONS OF POLIOMYELITIS OF THE MEDICAL RESEARCH COUNCIL*

During May and June, 1956, three batches of poliomyelitis vaccine were used for the inoculation of 200,000 children in Britain under the scheme organized by the Ministry of Health. The antigenic activity of these three batches was studied in a group of children who were shown to have no detectable antibodies to any of the three types of poliomyelitis virus before their course of inoculations. The group consisted of 196 children between the ages of 1 and 11 years, in five different areas—London, Manchester, Central Scotland, Sheffield, and Southend-on-Sea.

Each batch of vaccine was also tested for antigenic activity in monkeys.

Vaccine

The three batches—designated 4, 5, and 6—were prepared by the Glaxo Laboratories under the direction of Dr. W. Wood from virus strains Brunenders (type 1), MEF-1 (type 2), and Saukett (type 3) by the formalin-inactivation method introduced by Salk. The vaccine differed in its type 1 component from that produced in America, the Mahoney strain having been replaced by a partially attenuated variant of the Brunhilde strain (Enders *et al.*, 1952; Sabin, 1955). Gear (1956) has called this the Brunenders strain.

*Children were immunized and sera collected by Dr. A. Dudgeon, Dr. J. C. W. MacFarlane, Dr. D. O'Brien, and Dr. A. M. Peach (London); Dr. P. B. Stones and the medical staff of the Health Department (Manchester); Dr. N. R. Grist, Dr. W. M. Janieson, Dr. R. C. McLeod, Dr. B. R. Nisbet, and Dr. E. Neil Reid (Scotland); Dr. J. Rendle-Short, Professor C. H. Stuart-Harris, and Dr. D. Tyrrell (Sheffield); Dr. J. Greenhalgh, Dr. J. Stevenson Logan, Dr. A. D. Macrae, Dr. Gladys Neill, Dr. R. Pilsworth, and Dr. J. C. Preston (Southend-on-Sea).

Laboratory investigations were made by Miss A. J. Ferguson, Dr. F. F. Perkins, Mrs. K. Russell, and Dr. J. O'H. Tobin, of the Biological Standards Control Laboratory, Hampstead.

The results were analysed and the report prepared by Dr. F. T. Perkins and Dr. J. O'H. Tobin

Titration of Sera

Serial dilutions of serum in Hanks basic salt solution were mixed with an equal volume (0.25 ml.) of virus suspension in Parker 199 solution containing approximately 50 TCID₅₀ of the required type of virus. The mixtures were held at 37° C. for three hours, and then added to tubes containing 6- to 8-day-old monkey kidney cell cultures; two tubes were used for each serum dilution. The inoculated tubes were incubated at 37° C. in roller drums for six days, when the final readings of the cytopathic effect of virus were made and the titre (PD₅₀) of the serum determined. Titres are given as the dilution of serum in the serum-virus mixtures, as recommended by the Committee on Laboratory Investigations of Poliomyelitis.

All sera were titrated, using Brunenders, MEF-1, and Saukett as the test viruses. Twenty post-immunization sera were also titrated for type 1 antibody, using Mahoney as the test virus. They gave similar results to those obtained with Brunenders, the geometric mean titres being 1/90 with Brunenders and 1/70 with Mahoney.

As controls the three British Standard poliomyelitis antisera (to be published) were always titrated in parallel in each set of tests and gave consistent results throughout. Each standard serum has been assigned a potency of 10 units per ml. and, by the method of titration employed in this study, gave on the average a titre of 1/480, 1/806, and 1/960 for types 1, 2, and 3 respectively.

Antigenic Activity in Children

Of the 196 children selected for the study, the majority had their pre-immunization sera taken on the day the first dose of vaccine was given and the remainder within a month before the first dose. The sera were shown to be negative at a titre of 1/8 to all three types of poliomyelitis virus. Seventy-two of the children were injected with batch 4, 66 with batch 5, 53 with batch 6, and 5 with a mixture of batches. Each child received two injections, each of 1 ml., at an interval of three to four weeks. The injections were given intramuscularly into the left deltoid; no untoward reactions were observed in any of the children. Serum was taken on the average two weeks after the second dose and titrated for antibody to each of the three types. Those sera showing no antibody at 1/8 were retested at 1/2 and 1/4.

Table I gives the results of the three batches of vaccine together in each area and in all areas combined; the combined results are also given in the figure. The results clearly show that almost all children responded; of the 190 whose sera were fully examined, all except two responded to all three types. It is noted that the distribution of type I antibody titres in Manchester differed from that in the other areas; of the 33 sera from Manchester, 9

Antibody titres in children after immunization with poliomyelitis vaccine.