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Short report

Clinical neurological examination, neuropsychology, electroencephalography and computed tomographic head scanning in active amateur boxers

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SUMMARY Twenty active amateur boxers were studied seeking evidence of neurological dysfunction and, if present, the best method for detecting it. Seven of these boxers had an abnormal clinical neurological examination, eight an abnormal EEG and nine of 15 examined had abnormal neuropsychometry. The CT scan was abnormal in only one. An abnormal clinical examination correlated significantly ($p < 0.05$) with an increasing number of fights, and an abnormal EEG with decreasing age ($p < 0.05$). In several of the neuropsychometric tests, the boxers were significantly worse than controls ($p < 0.05$). Neuropsychometry was the best method for detecting neurological dysfunction.

Boxing remains controversial. In its recent report, the British Medical Association (BMA) collected evidence showing that, in both professional and amateur boxers, there is clinical, radiological and neuropsychological evidence of permanent brain damage.[1] In some the damage is severe.

The professional boxing authorities maintain that the BMA's evidence concerns brain damage which occurred before there was adequate medical supervision. In the most recent study however in which most of the boxers had fought after 1960 that is, the modern era, the majority had definite evidence of brain damage.[2]

The amateur boxing authorities maintain that the damage sustained by professional boxers would not, or could not, occur in their sport. There is certainly evidence to support this view,[3] although another study using electroencephalography (EEG), computed tomographic head (CT) scanning and neuropsychometry showed significant abnormalities in amateur boxers.[4] Because of the continuing controversy we decided to study this matter further.

The evidence for brain damage in professional boxers is conclusive and we, therefore, wanted to examine a very different group, active amateur boxers. Before embarking on a proper controlled study, however, we felt it necessary first of all to find out if there is evidence of abnormal neuronal dysfunction in this group and secondly the best method of detecting it. We have, therefore, examined prospectively an unselected group of active amateur boxers.

Patients and methods

We obtained a list of active amateur boxers from Club Secretaries and from the Scottish Amateur Boxing Association's Medical Officer. We then wrote to the boxers outlining our interest in examining them, and inviting them to participate. Other boxers on hearing about the study volunteered to come. We excluded anyone with known exposure to organic solvents, previous head injury, heavy drinking and any other known reason for brain atrophy.

Twenty boxers attended the Institute of Neurological Sciences, Glasgow and had a full clinical, neurological and neuropsychological examination. An EEG and a CT scan

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were also carried out.

The clinical examination was carried out by one of us (IDM). Particular attention was paid to general demeanour, quality of speech, pupillary size and reflexes, eye movements and convergence, postural control and co-ordination of limb movements and of gait.

The EEG was recorded on a 16 channel machine using the standard international 10/20 electrode position and the recording was continued throughout 3 minutes of hyperventilation. EEG interpretation was on the basis of straightforward visual analysis by one of us (IDM), based on conventional EEG criteria. We noted excess slow wave activity, recognising that this abnormality was non-specific. The EEGs were also examined independently by a specialist in EEG.

CT scans were performed on a General Electric 1010 head scanner and the views taken included the posterior fossa. We paid particular attention to ventricular size and configuration and to the possibility of cortical atrophy.

Neuropsychological function was assessed by means of clinical psychometric procedures and computer administered tests. The choice of measures was dictated by our assumption that boxing may cause damage of the kind found in minor head injury,[5 6] for example, deficits in learning and memory, and various aspects of attentional performance. These were investigated by simple memory span (digit span), simple and more complex word learning (the three paired word learning test of Inglis[7] and word learning from the Wechsler Memory Scale[8]). Recall of complex prose was tested by the "logical memory" sub-test of the Wechsler Memory Scale, and recall of complex visual information by means of the Rey figure test,[9] and recall of matrix patterns generated by an Apple 11+ microcomputer. Information processing was tested by means of the Paced Auditory Serial Addition Test (PASAT),[10] and a simple and 4-choice reaction time using a procedure which allowed movement times and decision times to be distinguished.[11] Finally, the ability to process rapid change in the visual modality was measured using a procedure which required detection of stimulus onset.[12]

During the study each investigator was unaware of the results of the other tests.

Results

The 20 boxers ranged in age from 18 to 49 years and the number of fights ranged from four to 200 (table).

Clinical neurological examination Neurological abnormalities were noted in seven (35%) (table). The most significant of these included extensor plantar responses, and impairment of rapid alternating movement of the forearms and hands. Abnormal neurological examination correlated significantly with increasing number of fights ($p < 0.05$ Mann-Whitney).

Electroencephalography An abnormal EEG was recorded in eight (table). Boxers number 6 and 17 showed dominant 7 Hz activity, abnormal for their ages. Several showed local slow wave activity unrelated to any relevant medical history. Two others

Details of boxers examined. Increasing number of fights correlates significantly with abnormal clinical examination ($p < 0.05$ Mann-Whitney). Abnormal EEG correlates with increasing age ($p < 0.05$ Mann Whitney)

Boxer	*Age years*	*No fights*	*Clinical examination*	*EEG*	*CT scan*	*Neuropsychometry*
1	18	4	Normal	Bilateral slow activity	Normal	Poor visual memory
2	21	7	Normal	Normal	Normal	Normal
3	35	12	Normal	Normal	Normal	Normal
4	33	17	Normal	Normal	Normal	Normal
5	30	20	Normal	Local slow activity (R) Hemisphere		
6	20	21	Normal	7 Hz Dominant Bilateral Discharges	Normal	Poor visual memory Slow reaction time
7	25	28	Normal	Normal	Normal	Poor attention
8	49	30	Normal	Normal	Normal	Normal
9	26	31	Normal	Normal	Normal	—
10	35	40	Normal	Normal	Normal	Normal
11	39	40	Pupil inequality	Normal	Normal	Poor verbal memory and attentional disturbance Slow reaction time
12	21	55	Normal	Normal	Normal	—
13	22	65	Poor rapid alternating movements	Normal	Normal	Poor verbal and visual memory. Slow reaction time
14	21	70	Head turn	Theta and delta activity right hemisphere	Normal	Poor attention
15	19	76	Poor rapid alternating movements	Minor disturbance (R) temporal	Normal	Poor verbal and visual memory. Slow reaction time
16	21	85	Normal	Slow activity posteriorly	Normal	Normal
17	19	95	Extensor plantar responses	7 Hz Dominant	Normal	—
18	32	140	Slow pupil reaction	Normal	Dilated ventricles No cortical atrophy	Poor verbal and visual memory. Slow reaction time
19	20	200	Extensor plantar responses	Fast transients Left temporal	Normal	—
20	21	200	Normal	Normal	Normal	Poor verbal memory

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showed fast transients or discharges but had no history of seizures. The specialist in EEG concurred with the first examiner's interpretation of abnormality in every case and added one further abnormal record to the list.

Of the eight boxers with abnormalities of the EEG four had an abnormal clinical examination. Abnormality of the EEG correlated significantly with age ($p < 0.05$ Mann-Whitney); the younger the boxer, the more likely he was to have an abnormal EEG. There was no correlation between an abnormal EEG and the number of fights.

CT scanning Nineteen of the 20 patients had CT scans that were normal for their ages. One boxer had a thick skull vault with dilatation of the lateral and third ventricles without any evidence of cortical atrophy. This could represent either central atrophy or a form of arrested hydrocephalus.

Neuropsychology Nine of the boxers examined had poor performance on two or more of the clinical measures. Three cases, Nos 11, 15 and 18 performed at a level on all six of the clinical measures that on clinical examination would suggest severe impairment. A further two patients scored at this level on four of the measures.

Using *t* tests the boxers were compared with a control group of orthopaedic out-patients with limb fractures. The boxers were significantly poorer on the Inglis Word Learning Test ($p < 0.03$), and on the copy and immediate recall of the Rey Figure. They did not differ significantly however on the Wechsler Memory Word Learning Test, or on digit span nor story recall. On the Inglis test the variance of the two groups differed significantly as two of the boxers scored extremely poorly on this test. Indeed this variance was a feature of the boxers' group, in that whereas most of them performed normally on a given test, a small subgroup (not always the same subgroup) performed extremely badly on each test. Unfortunately normal controls were not available for the PASAT, but it was noticeable that at the slow speed of presentation, four boxers had very marked problems scoring 60% or fewer and all four had problems on at least three other tests.

The results from the computer administered measures support and extend the findings from the clinical measures. On visual memory and detection of visual change, boxers performed normally, although they nevertheless showed a trend towards lower performance on the memory tests; four boxers had lower scores on one or both components of the memory tests than any of a control group of patients consulting at a local Health Centre. Using an analysis of variance to compare the median reaction times of the boxers with the reaction times of a group of university students and staff of a similar mean age, the boxers had significantly ($p < 0.05$) faster movement times than students. Simple reaction times of boxers and students were not different, but the boxers had significantly slower decision times on 4-choice reaction time ($p < 0.05$). This finding is similar to that reported by Van Zomeren[13] in an investigation of head injured patients. The pattern of results indicates that the slower decision times of boxers are not due to perceptual or motor factors, but have a central locus.

Discussion

Recent studies using the same investigation as ourselves have shown evidence of neuronal dysfunction in boxers. These studies were either in professional boxers of some standing or in retired amateur boxers.[4] We are more concerned with the early detection of neuronal dysfunction and, therefore, studied active amateur boxers. We have shown that in a group of apparently healthy active young men, there is clinical, electroencephalographic and neuropsychological evidence of abnormal brain function.

We must emphasise that it is not possible to conclude from our data that the abnormalities we have found are the result of boxing. It may be that any group of young men examined in this way would have similar findings. In addition our study group may be unrepresentative; it comprised only those who accepted the invitation to be examined and possibly they did so because they had complaints and were concerned. Finally it could be that men who take up boxing come from a population who already have such abnormalities. Definitive conclusions therefore are not possible.

What is the best method for detecting abnormal function or more importantly brain damage? In the Finnish study,[4] neuropsychometry, the clinical examination and the EEG were equally sensitive, the CT scan rarely being abnormal. In our study although the EEG was abnormal in eight (40%) this abnormality did not correlate with an increasing number of fights. The EEG is accepted to be outside normal limits in 10–20% of the normal population.[13] In our study an abnormal EEG did correlate with decreasing age. This is in keeping with the fact that in the young population with less mature brains the EEG is more likely to be abnormal. Therefore, although the EEG does detect abnormalities these are not necessarily due to brain damage. A single EEG, therefore, is not the best method of diagnosing brain damage. On the other hand a series of EEGs showing an emerging abnormality would be of significance.

It is not surprising that we did not find any case of brain atrophy on CT scanning. CT scanning only detects severe degrees of damage, which explains the well recognised abnormalities seen in professional

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boxers.[2] A normal brain scan, therefore, does not exclude significant brain damage and cannot be used for assessing it.

The high number of abnormalities on clinical neurological examination (35%) is surprising. Signs uncovered by an experienced neurologist are always noteworthy, but caution should be applied to some of the minor neurological signs when they are searched for so intensely. None of the present subjects could be regarded as in any way physically disabled, rather the reverse. Only one had symptoms. Thus the great majority of the motor system disorders we found were quite asymptomatic. Again only by serial examination would it be possible to show that these signs were the forerunners of clinical disability.

The best method for detecting neurological abnormalities and possible brain damage is neuropsychometry. In our study where we were able to compare the boxers' neuropsychological performance with a control group, we found that the boxers in some tests were significantly poorer than the controls. In addition, there were some boxers who had clinically significant abnormalities.

This is a preliminary study which has shown that there is neurological dysfunction in amateur boxers and the best method for detecting it is neuropsychometry. As we have already stressed, however, it would be wrong at this stage to attribute these abnormalities to boxing. A controlled study is necessary and we are currently carrying one out comparing neuropsychometry in active amateur boxers with that in their peers.

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