A CHARACTERISTIC FORM OF OVERACTIVE BEHAVIOUR IN BRAIN DAMAGED CHILDREN

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BEHAVIOUR disorders resulting from localized lesions of the brain have attracted increasing attention recently. With the rapid progress being made in neurosurgical treatment there is a prospect that their accurate diagnosis may become more than an academic exercise. In the present paper a description will be presented of characteristic overactive behaviour which appears, in the majority of cases, to be due to cortical damage, frequently involving the temporal lobe, and sustained either at the time of birth or in later childhood.

The behaviour symptoms exhibited were remarkably constant throughout the series of twenty-five children studied. There was marked restlessness, distractibility and an inability to concentrate for more than a few seconds at a time. The affected children showed an apparent compulsive need to touch every object which they saw, and to suck or chew them. Outbursts of aggressive and irresponsible behaviour occurred frequently, and the patients seemed to be incapable of responding to reprimand or chastisement for longer than a few minutes. The majority suffered from epilepsy or showed other evidence suggesting the presence of cerebral damage on examination.

SELECTION OF PATIENTS, AND METHODS OF EXAMINATION

Thirty-one patients with the type of behaviour described above have been seen in the past three years in the Royal Hospital for Sick Children, Edinburgh, and in a number of local schools, residential homes and institutions. The majority were referred on account of behaviour disorders, epilepsy, neurological symptoms, or because of failure to make progress at school. In six patients the histories were inadequate for analysis, or the facilities for detailed examination were lacking. These cases are therefore excluded from the series of twenty-five patients whose findings will be described.

Detailed histories of the family backgrounds of the patients were taken, special attention being paid to the presence of any psychiatric or neurological symptoms amongst parents or siblings.

Mothers were closely questioned about the patient's prenatal, natal and neonatal history, and whenever possible their answers were supplemented by reports from attending doctors and by notes from Maternity Hospitals. A careful history was obtained of the child's physical development, and any illness he had suffered. Descriptions of the child's behaviour at different ages were obtained from parents, other relatives, teachers, local doctors, and from the notes of any hospitals to which he had been admitted.

The children were observed at play for as long as possible on a number of occasions, preferably in different environments and with different people present. Some children, for example, were examined at home, at school, and in the In-patient and Out-patient departments of the Department of Psychological Medicine in the Royal Hospital for Sick Children. Every child was submitted

to a general medical and detailed neurological examination. Whenever the child was felt to be co-operative enough for helpful results to be obtainable, intelligence testing and electroencephalography were attempted. A number of patients had been more fully investigated by air encephalography and arteriography in other hospitals, and the results of these investigations were obtained.

Observations

Of the twenty-five patients, sixteen were male, and nine were female. Their ages varied from four to twelve years at the time of examination. The majority had first been referred to paediatricians for examination shortly after beginning school, or on reaching school age. In all the cases there was evidence in the histories, or on examination, which suggested the presence of cerebral damage. In thirteen cases this appeared to have been sustained at the time of birth ("Congenital cases"), and in twelve it had probably occurred as a result of intracranial infection, vascular disorder or haemorrhage after delivery ("Acquired cases"). The histories and results of examination are summarized in the accompanying table. As far as possible the description of behaviour symptoms is recorded in the parent's or guardian's own words.

Behaviour

In the congenital cases overactivity was not usually complained of until the children were between twelve and twenty months of age. Six patients were noted to be rather drowsy or lethargic in the first six or nine months of life, and seven were thought to have shown normal behaviour. Only gradually did symptoms of abnormal behaviour appear, after this time. In the acquired cases there was also a latent period following the initial intracranial infection or accident before overactivity was apparent. In eight there was a period of between six weeks and four months of apparent lethargy and in four a similar period of normal behaviour prior to overactivity becoming apparent.

Following the period of normal behaviour or lethargy the children became more and more active, and the parents complained that they were always restless and never tired by play. They began to take an unusually intense and sustained interest in features of their immediate environment. This interest was usually shown as a marked tendency to crawl or run rapidly to every object within reach, handle it and whenever possible bring it to the mouth to suck, lick or chew. Having explored it the object would be thrown away, only to be replaced immediately by another. Activity of this type could persist for many hours at a time, day after day, without the child tiring or seeming to become familiar with the objects he had handled. A single empty box, for example, might be explored with great urgency some twenty or thirty times a day for weeks on end, until its paint was licked off, or it was chewed beyond recognition. Failure to benefit by experience was shown by the frequency with which jets of steam or fires had been handled repeatedly in spite of burns being caused. No object or person could hold the child's interest for more than a matter of seconds, and the distractibility was such that even meals were interrupted repeatedly because of the child's compulsive desire to handle and suck things he had already investigated many times that day.

The marked desire to suck, bite or chew presented a major problem in some of the more severely affected patients. Some mothers had found that the amount of destruction caused to clothes and bedding was lessened if the child

was provided with a piece of cloth (Cases 6, 14, 16) or string (Cases 9, 16, 21) or a handkerchief to chew. But in two cases it proved impossible to keep the children in clothes at all. One had to be put to bed naked between bags made of rubber, and another naked on the springs of a spring mattress without bedclothes, but with an electric radiator placed high on the wall out of reach to keep him warm.

Scolding or beating were effective in modifying behaviour for only a minute or two, if at all. Even the children who knew what was considered right or wrong failed to resist the impulse to touch, put to the mouth and tear whatever had attracted their attention. When frustrated they were liable to rages, and often showed much strength and ferocity in aggressive behaviour of great intensity. Similar outbursts were a feature of the behaviour of most patients from time to time without there being any apparent provocation for it. In these aggressive phases the child would appear to be attacking his surroundings at random, kicking, hitting and biting everything in sight, whether animate or inanimate, animal or human. The majority of the patients had been refused permission to play with neighbouring contemporaries by the time they were two or three years of age.

The patients appeared to show a lack of affection for their parents and siblings. Five mothers complained in almost identical terms that they were "treated like pieces of furniture", to be touched and sucked or bitten whenever the child felt the urge to do so. Younger brothers and sisters were often brutally handled by the patients without apparent provocation. Six of the patients were said to have sudden "outbursts of love" when they would rush to one or other parent, sometimes practically throwing themselves at them and demand to be fondled and treated like infants.

Fear was absent in most of the patients. Two children, neither of whom could swim, had been rescued from drowning, one on two occasions. Attacks on adults who were total strangers and on older children were frequent. Characteristically the patients were those who could be dared to feats of courage with the greatest ease, and mothers complained repeatedly of the frequency with which their clothes were torn, and cuts and bruises needing medical attention were sustained.

By the time they went to school overactivity was a major problem in the patients. Teachers found that their restlessness, inability to concentrate, and their marked distractibility together with aggressive outbursts made education quite impossible in many cases, not only for the patients but for the rest of the class as well. The majority of children had been in at least two schools by the time they were seven, and periods of exclusion from school were often noted.

Marked fluctuations of mood and of the degree of overactivity were present in the majority of cases. These fluctuations were present from day to day and from week to week. It was found that the periods of intense overactivity tended to diminish in frequency, duration and severity in most cases after the age of seven or eight years. Two patients who had previously been considered to be ineducable on account of their overactivity so improved in behaviour without specific treatment by the age of twelve that full-time schooling was possible (Cases 11 and 23).

In most cases sleep was very deep and it was found very difficult to rouse the children from it. Its duration was usually between six and eight hours, and parents frequently asked if this could be considered enough in view of the amount of energy their children seemed to use in the day.

Epilepsy

Epileptic attacks of various types were present in thirteen of the twenty-five patients and were absent in twelve. Five had grand mal convulsions without any apparent localizing features (Cases 5, 9, 14, 21, 22 and 25). In Case 21 the onset of an attack had never been witnessed, and in Case 22 convulsions were infrequent and only occurred when the child was febrile. In Case 25 the patient also suffered from petit mal. Her attacks of grand mal were atypical but showed no definite localizing features. Case 14 suffered from both grand mal and psychomotor attacks. The latter occurred three or four times a day. During them the child seemed to be quite disoriented and would run about waving her arms for from one to three minutes alternately crying and laughing. She usually passed urine during attacks, and slept or was drowsy after them. In two patients with grand mal (Cases 4 and 13) each fit was preceded by a period of one or two minutes in which the child was disoriented and hallucinated. Both cried out in fear during this time. Case 4 persistently complained of bears, ghosts or burglars coming to attack him from the right hand side, and would curl up in his bed in great terror immediately before losing consciousness and showing clonic movements of the limbs. In Case 24 the epileptic attacks, which were very typically of grand mal type were constantly preceded by sniffing and chewing movements for about twenty seconds.

In three of the acquired cases (15, 16 and 17) the attacks were frankly Jacksonian in type. In the latter case consciousness was impaired but not lost, whilst in the other two consciousness was lost for five or ten minutes. In Cases 15 and 17 the patients had warning of attacks and would run to their mothers, apparently very afraid, which was very unusual behaviour for them. In Case 15 the child became hallucinated for a few seconds before each attack and would cry out in terror, "They are coming to get me", or some similar phrase. Owing to the severe mental defect in Case 17 it is difficult to be sure that the child was in fact hallucinated, but the mother was convinced that "He seems to see things" immediately before his attacks. In Cases 15 and 17 clonic movements began in the face, in Case 16 in the paretic right hand. In Case 3 the attacks were confined to the right side, but were not typically Jacksonian in type.

The frequency of grand mal or Jacksonian attacks varied greatly from patient to patient from an average of one a year in Case 21 to three or four a day in Case 13. The occurrence of convulsions appeared to have no great effect on behaviour afterwards except in Cases 9, 14 and 22, whose mothers all thought that behaviour was less overactive for a day or two after each attack.

Neurological Findings

The neurological findings are summarized in the table. It will be noted that in two patients no definite neurological abnormalities were found (Cases 6 and 13) though the histories were compatible with the occurrence of cerebral birth injury in each case, and in the latter the electroencephalogram was grossly abnormal. Seventeen cases showed hemiplegia, ten on the right and seven on the left. Case 21 showed bilateral hemiplegia. Spastic diplegia was present in two patients (Cases 9 and 10), both of whom had ataxia of cerebellar type. Case 11 had moderately severe bilateral choreo-athetosis, apparently of congenital origin. In Case 19 there was ataxia of cerebellar type associated with defective vision, slight increase of the tone in the right limbs and an equivocal right plantar response, the left plantar response being flexor. In Case 22 there was slight increase of tone in the right side associated with aphasia, but the plantar

responses were flexor bilaterally. Neither of these children had significant paresis of the limbs.

Intelligence Testing

Intelligence testing was attempted in the majority of cases, but to obtain reliable estimates of intellectual endowment was extremely difficult on account of their poor co-operation and concentration. Of the nineteen children in whom formal testing was attempted ten were considered to be of at least average intelligence. Seven were "dull and backward" or high grade defectives, and two were severely defective. Six were untestable or probably severely mentally defective. School performance appeared to bear little relationship to the level of intelligence estimated as a result of testing. This seemed to be because the children could be induced to concentrate a little better for the relatively short time of the test than they could be in a busy classroom.

Electroencephalography

Nineteen patients were subjected to electroencephalography, but the recordings were unsatisfactory in three owing to their marked restlessness. In the other sixteen cases the records were within normal limits in two (Cases 6 and 10). In nine cases the recordings were outwith normal limits, but the abnormalities were present bilaterally. In Cases 4, 6 and 13 generalized delta activity of moderate amplitude was present bilaterally, but was most marked over the left temporal region in Case 4, and over the right parietal region in Case 6. In Case 13 the maximum activity was in the right temporoparietal region. In Case 18 there was generalized high amplitude activity most marked on the left.

Treatment

Phenobarbitone had been given to fourteen patients in the series, eleven of whom had epileptic attacks. Of the latter eight showed a reduction of the number, or abolition of the epileptic attacks following administration of the drug, and in three there was little or no difference in the number of attacks or in their severity. Of the fourteen patients who had been given phenobarbitone two showed diminished overactivity following its administration (Cases 19 and 21). In four patients no significant change in behaviour was noted. In eight patients there was a definite exacerbation of the overactivity, especially of the tendency to aggressive outbursts. In two (Cases 4 and 13) this exacerbation had been treated by increasing the dosage of phenobarbitone, and an apparently still more marked behaviour disturbance had resulted. A dosage as small as gr. $\frac{1}{4}$ twice a day in two five years old children (Cases 13 and 22) was sufficient to increase the severity of overactive behaviour.

Phenytoin sodium had been given to seven patients, all of whom suffered from epilepsy. In four the attacks were abolished or reduced in number, and in three they were unaffected by the drug. Behaviour was unaffected in four patients, possibly slightly improved in two, and exacerbated in one.

Amphetamine sulphate had been given to nine patients, four of whom had grand mal or Jacksonian epilepsy. In a dosage of from 10 to 30 milligrams in the day it resulted in a diminution of the number of epileptic attacks in two cases (13 and 15). In the former the frequency of psychomotor attacks was also reduced. In two patients epileptic attacks were unaffected (Cases 4 and 24). The overactivity was unaffected in four patients, slightly diminished in one, and greatly lessened in four. In all four in whom there was marked improvement

distractibility lessened and there was a gain in the ability to concentrate. Thus school work improved dramatically in two cases (8 and 11).

Primidone had been given to ten patients, of whom five suffered from epilepsy. In four the attacks were abolished or much lessened in frequency. In one (Case 3) there has not been time to assess its effect on the infrequent convulsions. In seven patients there has been a dramatic lessening of the overactivity, a marked improvement in the span of concentration and a reduction in the severity and frequency of aggressive outbursts. The improvement has been evident within two or three days of giving the drug in a dosage of 125 milligrams twice a day in all the seven cases, and has been maintained on this dosage for periods of up to nine months. In one of the three patients who failed to respond to primidone (Case 8) the overactivity was somewhat diminished by treatment with reserpine (Serpasil), 0·25 mg. three times a day, but a more satisfactory response was obtained with chlorpromazine, 25 mg. three times a day.

The home management of the patients was a matter of great difficulty because of their intense distractibility and tendency to have aggressive outbursts whenever they were frustrated. In general, parents who were able to adopt a rather firm but kindly attitude, and whose discipline was strict, appeared to be more successful in producing relatively amenable behaviour than did those who regarded the disorder as being beyond the child's control. Though psychotherapy increased insight into the nature of their disability in a number of the more intelligent patients it did not by itself appear to result in significant improvement in behaviour.

Aetiology

Cases were grouped into congenital and acquired categories on the basis of their birth history, their milestones and the presence or absence of evidence suggesting that there had been a change in behaviour following a later episode of intracranial trauma, infection or cerebral anoxia.

Detailed medical notes of the births were available in nine of the thirteen congenital cases. In Case 10, who was adopted at the age of three weeks, no details of the birth could be obtained but she was said to have "had a very rough time of it during delivery", and at the time of adoption was unusually drowsy and difficult to feed. In Cases 7, 8 and 9 full medical details of the births were not available, but in all three delivery was known to have been abnormal.

Prolonged apnoea after birth was present in eight cases and in none of these had labour and delivery been normal. In Case 4 the child breathed immediately after birth, but the history of severe coal gas poisoning of the mother twenty-six hours before the birth, after which foetal heart sounds could not be heard and foetal movements were absent was felt to suggest the presence of foetal anoxia. It is impossible in retrospect to attempt to assess the degree of trauma and anoxia to which each infant was exposed.

Full details were obtained of the illnesses and accidents which appeared to have resulted in changes in behaviour and neurological abnormalities in all twelve of the patients in the acquired group. All but one (Case 21) had been admitted to hospital on account of the causative conditions. With the exception of Case 20 who was knocked down at the age of eight years, and Case 19 who suffered from very severe whooping cough at the age of five years, all the acquired cases were under the age of three at this time, and eight were under the age of two years.

In six cases the causative disorder was diagnosed as cortical thrombophlebitis. In five patients this was a complication of acute or chronic middle ear

disease (Cases 14, 16, 17, 24 and 25). In Case 17 an extensive cerebral abscess resulted, which destroyed much of the left temporal and parietal lobes. In Case 16 thrombosis of the internal and external jugular veins occurred and air encephalography revealed gross left cerebral atrophy. In Case 14 air encephalography revealed marked bilateral enlargement of the lateral ventricles and cerebral atrophy which was more marked on the left than the right. In Case 22 very severe dehydration resulted from gastro-enteritis which complicated an attack of tonsillitis. Recurrent convulsions with impairment of consciousness were present, and following investigation cerebral thrombophlebitis was diagnosed. In two cases (23 and 21) there were histories suggestive of the occurrence of cerebral thrombophlebitis, but this diagnosis was not made at the time of the illness, and it is impossible to be certain about it retrospectively.

In Cases 15 and 19 changes in behaviour occurred following severe attacks of whooping cough, complicated by fits and impairment of consciousness in both. In Case 18 the patient developed tuberculous meningitis at the age of 14 months. This was successfully treated with streptomycin, but a relapse occurred eight months after the initial attack, and following recovery she became progressively overactive. Case 20 was unconscious for one week following head injury. Severe widespread cerebral contusions most marked over the left temporal lobe were noted at exploratory operation following the accident.

Discussion

Overactive behaviour of the type shown by the patients in the present series has been described by a number of authors (Bakwin and Bakwin, 1953; Peterman, 1953). The most detailed account is that of Ounsted (1955). This author found 70 patients who exhibited this form of behaviour amongst over 800 children referred on account of epileptic attacks. The behaviour was characterized by increased distractibility, a short attention span, fluctuating mood with underlying euphoria, aggressive outbursts, diminution or absence of spontaneously affectionate behaviour, lack of shyness and absence of fear. The children were apparently tireless and failed to respond to reprimand or punishment. Their intelligence varied from high average to grossly defective. The majority of patients showed no definite neurological lesion, but fifteen of them showed spastic hemiplegia. A wide range of normal and abnormal electroencephalographic patterns were noted. Phenobarbitone tended to exacerbate the behaviour disorder in most patients but resulted in a reduction in the number and severity of epileptic attacks. Amphetamine was effective in improving the behaviour symptoms in a proportion of cases, and caused a reduction in the frequency and severity of fits. Primidone was the most frequently successful drug in controlling both the fits and the behaviour symptoms.

Ounsted did not comment, however, on the very marked oral tendencies exhibited by the majority of patients with this form of overactive behaviour. A tendency to touch everything in sight and then suck, bite or chew it in order to investigate it further was one of the most striking and consistent features of the patients in this series. It is also worth stressing that epilepsy is by no means a constant finding in patients with overactive behaviour of this type, though in the majority of cases the histories and findings on examination indicate that brain damage has occurred. In the present series as many as twelve of the twenty-five cases had no convulsions. The tendency for there to be spontaneous remission of the overactivity in a proportion of cases is also noteworthy.

A number of authors have postulated that the form of overactivity which

has been described is most frequently the result of subcortical brain damage and that it often follows anoxia at the time of birth (Balf, 1952; Ounsted, 1955). However the frequency with which Jacksonian or unilateral convulsions, hemiplegia, aphasia and homonymous visual field defects occur in the present series suggests strongly that cortical lesions are usually present, and that they involve the temporal lobes in at least a proportion of cases. The visual and gustatory auras which were present in at least four of the cases with epilepsy provide some supporting evidence in favour of this conclusion.

The finding that only two of the patients showed electroencephalographic foci in the region of the temporal lobes is not against this hypothesis, for it has been stressed recently that unilateral temporal lobe lesions, especially those involving the hippocampus, may give rise to widespread bilateral dysrhythmia over the surface of the brain (Gastaut, Naquet and Roger, 1952).

The high proportion of congenital cases with a history of neonatal apnoea or other paranatal anoxia is of interest. It has been shown that anoxia at the time of birth or later is not infrequently followed by the appearance of localized areas of cortical necrosis and scars, as well as the more commonly occurring striatal damage (Courville, 1953; Penfield and Erickson, 1941). The particular liability of children, especially those under the age of three years to suffer from cortical thrombophlebitis as a result of acute or chronic otitis media and other infections in the region of the head and neck has long been known. The occurrence of cerebral abscess, particularly in the region of the temporal lobe is a well-recognized complication (Symonds, 1927; Ford, 1952).

There is some interesting experimental evidence that induced lesions of the temporal lobes in animals may result in overactive behaviour, similar in some respects to that observed in the patients in the present series. Following bilateral temporal lobotomy involving the uncus or hippocampal gyrus in monkeys it was found that the animals became markedly overactive (Klüver and Bucy, 1939). These authors found that the animals showed "psychic blindness" manifest as a tendency to approach animate and inanimate objects without hesitation, even when previously they had been afraid of them. There was an apparently compulsive desire to contact every object in sight, handle it and put it to the mouth. Oral tendencies were very marked and every object which could be reached was sucked or chewed and swallowed if edible. The authors noted the presence of "hypermetamorphosis" or evidence which suggested that behaviour was being controlled by an irresistible impulse or series of impulses. Emotional changes sometimes occurred, usually tending towards a lessening of affection for the opposite sex.

In the cat alterations of behaviour were observed after damaging the hippocampal gyrus experimentally by Gastaut, Vigouroux and Naquet (1952). These authors found that the animals showed chewing movements and overactivity of the muscles of the mouth associated with excessive salivation. There was a tendency for the cats to be unusually alert and active in exploring their environment, especially on the side opposite to that on which the lesions were made. Sometimes unusual states of rage and fear were noted.

To lay too much stress on the results of these animal experiments, especially when they involve observations on behaviour is obviously dangerous. There does, however, appear to be a remarkable similarity in some of the features described as being typical of the behaviour of the animals after experimentally induced lesions of the temporal lobes, especially those involving the hippocampus to that found in the patients described, the majority of whom showed other evidence of cortical damage.

Summary and Conclusions

The histories and clinical findings of twenty-five children showing a characteristic type of overactive behaviour are described. The behaviour is typified by an increase in distractability, poor attention span, an apparently irresistible urge to touch and chew every object which is seen, a diminished capacity for spontaneous affectionate behaviour, aggressive outbursts, an absence of fear and a failure to respond to reprimand or chastisement. Neurological findings indicating the presence of cortical lesions were present in the majority of cases and thirteen, or about half, suffered from epilepsy. In most patients phenobarbitone resulted in an exacerbation of behaviour symptoms, though the drug appeared to diminish the epileptic manifestations. Amphetamine and primidone were successful in controlling both behaviour symptoms and epileptic manifestations in a high proportion of the patients to whom they were given.

Most of the patients had histories suggesting the occurrence of cerebral injury. In thirteen this appeared to have been sustained at the time of birth, most commonly as a result of paranatal anoxia. In twelve cases it was acquired as a result of trauma or anoxia or of intracranial infections of which cerebral thrombophlebitis was the most common. All but two of the twelve acquired cases were under the age of three years at the time of their causative illness.

The histories and neurological findings, especially the frequency with which aphasia or disproportionately severe retardation of speech development, homonymous visual field defects and Jacksonian or epileptic attacks with visual or gustatory auras occurred suggest that the majority of the patients suffered from lesions of the cerebral cortex involving the temporal lobes in a proportion of the patients. Abnormalities of behaviour showing a resemblance to the symptoms present in the patients in the series have been produced experimentally in animals by temporal lobotomy.

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