Patterns of decerebration in infants and children : defects in homeostasis and sequelae

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SUMMARY Sixty-four infants and children showing signs of decerebrate rigidity admitted to a paediatric unit have been studied. Cases of head injury, myelomeningocele, and tumours were excluded from the series. The aetiological factors causing decerebration in the remainder fell into four main groups: infections, hypoxia, metabolic disease, and intracranial haemorrhage. Increased intracranial pressure was diagnosed in 87%. Defects in homeostasis occurred in 75%, respiratory abnormalities were present in 66%, cardiovascular in 33%, hypothermia in 30%, and hyponatraemia in 17%. Early recognition and treatment of raised intracranial pressure and defects in homeostasis are of the utmost importance if morbidity and mortality are to be minimized. There was a 31% mortality from the acute illness: 30% of the survivors were normal at follow-up examination; the remainder showed varying degrees of handicap. The severity of decerebration showed no correlation with aetiology or prognosis. The study shows that a wide range of disorders can lead to the clinical picture of decerebration in the young child, and that the prognosis is probably much better than in adults showing the same symptoms and signs.

Severe brain damage may occur in the course of many metabolic and infectious diseases affecting the brain in infants and young children in whom the acute stage of brain injury may be manifest clinically as convulsions, coma, and states of decerebrate and decorticate rigidity (generalized extensor hypertonus). In this paper the clinical findings of 64 children who showed a clinical picture of acute decerebration are described. The need for wider appreciation of the significance of acute decerebration and its emergency medical treatment is emphasized.

DEFINITION

Two major patterns of generalized extensor hypertonus in the human as a result of lesions of the brain have been described. In both there is generalized extension of the trunk, and of the lower limbs, which are thrown into a position of extension and internal rotation of the hips, extension of the knees, and plantar flexion of the feet, often with 'scissoring'. There is a gross increase of muscular tone, associated with exaggerated stretch reflexes and extensor plantar

responses. No matter in which position the child is placed the increase of muscle tone and abnormalities of posture persist (Sherrington, 1898; Kinnier Wilson, 1920; Walshe, 1923a, b; Fulton, 1943).

In the first and commoner type which may be termed, after Sherrington, 'true decerebration' extensor rigidity in the legs is associated with adduction and internal rotation of the shoulders, extension of the elbows, pronation of the forearms with flexion of the wrists and digits, the thumb often being held beneath the fingers in the palms of the hands. In the second type, termed more accurately by Sherrington, 'decortication', there is semi-flexion adduction and internal rotation at the shoulders, semi-flexion or flexion of the elbows with flexion of the wrists and digits, the thumbs again being held beneath the flexed fingers as in the first type. For the sake of simplicity we shall refer to both these types of acute extensor hypertonus as 'acute decerebration' in this article.

In decerebrate rigidity of both types there is an exaggeration of the so-called 'primitive reflexes' including the rooting, lip, and sucking responses

(Peiper, 1964). The grasp reflexes, the symmetrical and asymmetrical tonic neck reflexes, and the trunk incurvation reflex are easily elicited.

There is a marked similarity in the postures assumed by decerebrate babies and babies in the dystonic stages of diplegia and dyskinetic cerebral palsy, but in the latter changes of the position of the child in space tend to modify the child's posture and muscle tone (Ingram, 1964).

METHODS

Sixty-four patients were studied between 1968 and 1970. The majority of patients were seen in the general medical and surgical wards in the Royal Hospital for Sick Children, Edinburgh, and in the nurseries of the Simpson Memorial Maternity Pavilion, Royal Infirmary, Edinburgh, as soon as they showed neurological abnormalities. Thereafter they were seen frequently while in hospital. The survivors were examined regularly as outpatients. Twenty of the 64 patients died between 24 hours and nine months after the initial illness. Of these, 13 came to necropsy.

Patients suffering from the following categories of neurological disease who showed decerebrate rigidity were excluded from consideration: known head injuries; intracranial tumours; spina bifida with or without hydrocephalus; mild transient neonatal decerebration of the type which commonly follows hypoxia; apparent decerebration in the newborn the result of hypocalcaemia and/or hypomagnesaemia (Brown, 1970). Neonates with persistent decerebration of more than 24 hours duration after birth or postnatal hypoxia, were included.

RESULTS

AGE RANGE The ages of the patients studied varied from 1 day to 10 years. Sixty of the 64 patients were aged less than 4 years (Table 1).

TABLE 1
AGE AND SEX DISTRIBUTION OF CASES

Age (yr)	Male	Female	Total
Under 6/12	24	12	36
1	3	2	5
2	3	1	4
3	3	5	8
4	4	3	7
Over 4	4	0	4

SEX RATIO There were 41 boys and 23 girls, a ratio of almost two to one.

PREVIOUS HISTORY We tried to determine if there were any indication of abnormality before the illness in which the episode of decerebration had occurred. There was strong presumptive evidence in 53 cases (83%) that the children had been normal. Three were known to have shown neurological symptoms and/or signs previously, and in the remaining eight the earlier histories were too short or too inadequate for any reliable opinion to be made as to the child's previous normality or abnormality.

CLASSIFICATION OF PATIENTS Decerebrate rigidity is almost always the result of severe, and usually acute, raised intracranial pressure (87%), metabolic disturbance (5%), or diffuse encephalopathic disease (8%), however these are caused. In a significant proportion of the patients suffering from raised intracranial pressure, cerebral oedema was thought to be the cause (51%), while hydrocephalus and space occupation accounted for equal numbers of the remainder.

It will be seen (Table 2) that a large number of underlying disorders may result in the clinical picture of decerebrate rigidity. Moreover, a number of these may produce decerebration in different ways (Table 2).

CLINICAL OBSERVATIONS

PREMONITORY SIGNS AND ONSET OF DECEREBRATE RIGIDITY A number of typical neurological changes may occur in young children shortly before decerebrate rigidity appears—for example, neck retraction, strabismus, and lessening of awareness. Cycling movements of the lower limbs, which are occasionally unilateral, are common premonitory signs. 'Doggy paddling' movements in the arms are the equivalent in the upper limbs of the cycling movements in the legs, but are less commonly seen.

At this stage it is common to find that there is an exaggeration of the symmetrical and asymmetrical tonic neck reflexes, the Perez reflex (extension of the spine, flexion of the legs, and

TABLE 2

AETIOLOGY OF DECEREBRATE RIGIDITY IN 64 CHILDREN

	Cases (no.)
Intracranial haemorrhage (30%)	
Subdural haemorrhage—battered baby	5
—other causes	5
Intraventricular haemorrhage	6
Arteriovenous malformation	1
Other	2
Infections (30%)	
Meningitis	8
Encephalitis	6
Abscess	1
Cortical thrombophlebitis	1*
Septicaemia (with intravascular coag.)	2
Fatty liver encephalopathy	1
Hypoxia (18%)	
Cardiac arrest (anaesthetic)	3
Neonatal hypoxia	6
Drowning	1
Post convulsion	2
Metabolic (14%)	
Hypernatraemic dehydration (G. cnt.)	3
Alkalotic tetany	2
Kernicterus	1
Burns encephalopathy	2
Water intoxication (transfusion)	1
Other (8%)	
Birth trauma	3
Congenital malformation	1
Sagittal sinus injury	1

* Plus two in meningitic group.

extension of the head on pressure stimulation over the dorsal spinous processes), and the trunk incurvation response. These features should be regarded as danger signals, for they are often rapidly followed in a matter of minutes, or at most in one to two hours, by the appearance of decerebrate rigidity in a high proportion of patients.

Decerebrate rigidity is usually first evident in the lower limbs, which extend at the hips and knees with the feet plantar flexed, toes abducted, and great toes hyperextended as in the spontaneous extensor plantar response (spontaneous Babinski response). The pattern may then develop into one or other of the full pictures of decerebrate rigidity described above.

In some patients the first evidence of extensor hypertonus occurs in the neck and proceeds in a cephalo-caudal direction, so that head retraction is closely followed by extension and hyper-

tonus in the trunk and upper limbs and then in the lower limbs.

Once the picture of decerebrate rigidity is established, the Perez reflex, the trunk incurvation reflex, cycling movements, and the asymmetrical tonic neck reflexes are temporarily inhibited by the severe extensor rigidity of the muscles, but may reappear as soon as it lessens.

In the established picture of decerebrate rigidity a number of stimuli cause an increase in the extensor pattern and in muscle tone. These include exerting gentle pressure on the nose, which tends to cause increased hyperextension of the great toe (termed unofficially in our clinic the nasal Babinski response or snout reflex). Perineal stimulation may have a similar effect. Occasionally, the severity of the rigidity may be observed to vary during the respiratory cycle, becoming more marked on inspiration, and lessening on expiration.

SEVERITY OF DECEREBRATE RIGIDITY It is possible to recognize varying degrees of severity in decerebrate rigidity. (1) The term 'mild decerebrate rigidity' is applied when the child shows neck retraction with exaggerated extensor reflexes and generalized rigidity only on stimulation such as handling. This rigidity does not alter with changes of position in space. (2) Moderately severe decerebrate rigidity is considered to exist when there is neck retraction with persistent extensor postures of the lower limbs. Alternatively, there may be neck retraction with involvement of the trunk and upper limbs, the lower limbs being affected to a lesser extent. (3) Severe decerebrate rigidity is marked by opisthotonos with gross extension of the lower limbs and usually also the upper limbs, though there may be semi-flexion at the elbows as noted above (Fig. 1a, b, c).

In the present series of 64 patients, two were classified in the mild category (1), 17 in the moderate category (2), and 45 in the category termed severe (3). (cf. Appendix).

Neither the cause of the decerebration nor the prognosis can be determined on the basis of the severity of the decerebration. For example, in case 61, water intoxication produced a picture of total decerebration, and in case 20 a large and ultimately fatal subdural haematoma produced only mild to moderate extensor hypertonus (Table 2).

(a)

(b)

(c)

FIG. 1. *Clinical pattern of extensor hypertonus.* (a, top) *Mild.* (b, centre) *Moderate.* (c, bottom) *Severe.*

AWARENESS AND IMPAIRMENT OF CONSCIOUSNESS

It is difficult to assess the level of consciousness in young infants. Instead it is necessary to use criteria of alertness or level of arousal, as suggested by Prechtl and Beintema (1964). In older children, above the age of 6 months, depression of consciousness may be assessed approximately

in the same way as adults. The stages marking the progressive depression of the level of consciousness are of particular value in such patients (Wylie and Churchill-Davidson, 1965).

It was found that most older children in the series showed depression of consciousness when intracranial pressure was significantly raised. In

contrast, a number of the young infants lay in a totally opisthotonic posture and yet could suck a bottle and even smile at the appearance of their mothers. The level of alertness and arousal was thought to be normal in 10 infants. Sixteen infants were classified as being extremely apathetic, a stage which was considered to be the equivalent of being unconscious in the older child. Six children showed impairment of consciousness and 32 were unconscious, using the criteria suggested above. There was a striking dissociation in some patients between the severe degree of decerebrate rigidity and the level of consciousness (Halsey and Downie, 1966).

SIGNS OF INCREASED INTRACRANIAL PRESSURE A high proportion of patients had signs of raised intracranial pressure. This was diagnosed either on the basis of abnormalities of the fundi or changes in the pupils and eye movements as

oedema was present in only 24 of the 56 children with eye signs suggesting the presence of raised intracranial pressure, and was absent in 57%. Severe cerebral oedema, confirmed at necropsy, was found to be compatible with normal fundal appearances. Decerebration due to diffuse encephalopathic lesions or metabolic causes was always associated with normal fundal appearances and usually also with normal pupils, though abnormal eye movements were found in some patients in all three of the major categories (Table 3).

SEIZURES Seizures occurred in 42 (64%) of the patients in the series during the illness associated with the appearance of decerebrate rigidity. The seizures were focal in 21 (32%), of grand mal type (or of the generalized type seen in infancy), in 17 patients (26%), and essentially tonic in type in 13 (20%). Nine patients had seizures of more than one type.

ABNORMALITIES OF RESPIRATION Forty-three patients (66%) showed abnormalities of respiration while in the state of decerebrate rigidity. In two patients (cases 44, 45) the respiratory disorder was considered to be the primary cause of brain dysfunction (see below), but in the other 41 there was no evidence of primary respiratory disease and the abnormalities of respiratory function were considered to be of neurogenic origin.

One infant (case 44) who suffered from an *E. coli* septicaemia, required intermittent positive pressure respiration. The hyperventilation which resulted reduced his plasma CO₂ to 25 mmHg with an acute alkalosis and he showed a picture of complete decerebration with opisthotonos and generalized fits of the multifocal infantile type. Adjustment of the ventilator completely reversed the clinical picture. The baby ceased to convulse and the decerebrate rigidity disappeared. Another infant was hyperventilated during bronchoscopy. He showed a picture of decerebrate rigidity which lasted for some hours, though recovery was complete (case 45).

The respiratory abnormalities which were encountered in association with decerebrate rigidity are shown in Table 4.

ABNORMALITIES OF CARDIOVASCULAR FUNCTION Bradycardia, irregularity of the heart rate and

TABLE 3
OCULAR FINDINGS ASSOCIATED WITH ACUTE DECEREBRATION
IN INFANTS AND CHILDREN

	Cases	
	(no.)	(%)
Fundi		
Normal	32	50
Papilloedema	24	37.5
Retinal haemorrhage	10	15.6
Pupils		
Normal	32	50
Unequal	5	7.8
Pinpoint	7	11
Dilated	20	31
Ocular movements		
Normal	22	34.4
'Sunset' sign	6	9.4
Ophthalmoplegia	13	20
Doll's eye response	14	21.9
VI nerve palsy (isolated)	2	3
Conjugate spasm	7	11

(vertical 2)

listed in Table 3, or because the sutures were splayed, the fontanelle tense, or the occipito-frontal circumference was increasing. The latter signs were present in 28 patients (44% of the series).

It will be noted from Table 3 that papill-

TABLE 4
RESPIRATORY ABNORMALITIES IN INFANTS AND CHILDREN
WITH ACUTE DECEREBRATION

	Cases	
	(no.)	(%)
Cyanotic attacks or cyanosis	14	21
Apnoeic attacks or respiratory arrest	21	32
Tachypnoea (over 70/min)	8	12
Bradypnoea	6	9
Cheyne-Stokes respiration	3	5
Laryngeal stridor	5	8

cardiac arrest, all known to result from raised intracranial pressure, were encountered in a number of patients. Bradycardia was found in six (cases 1, 8, 9, 11, 13, 35), though in one case (case 9) the rate slowed markedly to 20 per minute. In contrast, tachycardia of more than 200 per minute was noted in six patients and in another five the heart rate was between 170 and 200 per minute.

The electrocardiogram (ECG) may be abnormal with sinus bradycardia, sinus arrest or varying heart block and yet it may revert dramatically back to normal if the intracranial pressure is reduced, as by aspirating the lateral ventricle through the anterior fontanelle.

One child (case 9), who also showed bradycardia, had five episodes of cardiac arrest which were associated with other evidence of autonomic over-activity—intense salivation, sweating, and borborygmi which were audible without a stethoscope. Injection of atropine intravenously immediately reversed the cardiovascular ab-

TABLE 5
DEFECTS IN HOMEOSTASIS ASSOCIATED WITH ACUTE DECEREBRATION IN INFANTS AND CHILDREN

Defect	Cases	
	(no.)	(%)
Hypothermia: temperature 35° C (95° F) or less	19	29.7
Hyperthermia: temperature 40° C (104° F) or over	7	10.5
Hyponatraemia: sodium 130 m-equiv/l. or less		
lowest 110 m-equiv/l.	11	17
Hypoglycaemia	6	9.4
Hyperglycaemia: over 200 mg/100 ml.	2	3
Failure to thrive: ?diencephalic syndrome	2	3

normalities. Six other patients (cases 1, 5, 7, 13, 14, 17) suffered from cardiac arrest, apparently secondary to an increase of intracranial tension, associated with decerebrate rigidity. In another three, cardiac arrest occurred during anaesthesia in previously healthy children. In two (cases 9 and 13) of the 10 patients who suffered from episodes of cardiac arrest, the heart stopped beating on more than one occasion. Two of the children who suffered from cardiac arrest, apparently as a result of raised intracranial pressure (cases 13 and 62) associated with hydrocephalus, showed no significant change in their neurological findings on later examination.

TABLE 6
SEQUELAE OF ACUTE DECEREBRATION IN INFANTS AND CHILDREN

Sequelae	Cases	
	(no.)	(%)
Dead	20	31
Normal	19	29.5
Severe handicap	16	24
Mental retardation	15	
Blind	2	
Deaf	1	
Epileptic	5	
Tetraplegic or dystonic	10	
Hemiplegia	6	
Mild handicap	7	11.5
Ataxia	5	
Hyperkinesia	3	
Mild hemiparesis	1	
Abnormal before acute episode (no change)	2	3

TEMPERATURE REGULATION Some other defects in homeostasis are summarized in Table 5. Hypothermia, defined as a rectal temperature of 35° C (95° F) or less, was present in 19 cases (30%) during their acute illnesses. A temperature of 40° C (104° F) or more was noted in seven patients, and in one (case 1) the temperature was 41.7° C (107° F).

Marked fluctuations in temperature as wide as 29°–41.7° C (85°–107° F) were seen in one 24 hour period. In two patients loss of temperature control persisted—in case 31 for a period of eight months until he died, and in the other case, who suffered from permanent neurological damage after his episode of decerebration, it was present one year later (case 12) (Fig. 2).

FIG. 2. Loss of temperature control with poikilothermia in an infant after severe asphyxial brain damage, associated with decerebration.

CEREBROSPINAL FLUID In 78% of patients a lumbar puncture was performed, because the risk of missing the presence of meningitis was considered to be greater than that of causing coning of the brain, even when there was evidence of increased intracranial pressure. Intravenous mannitol (5–7 ml./kg) was given 20 minutes before the lumbar puncture in order to lessen the dangers of the procedure, but in spite of this the clinical conditions of three patients deteriorated after the puncture.

ELECTROLYTES AND OSMOLALITY Hyponatraemia was considered to be present when the serum sodium concentration was 130 m-equiv/l. or less. This was found in 11 patients (17%) and in two it was only 110 m-equiv/l. (cases 26 and 64).

Water intoxication is one cause of cerebral oedema and in case 60 the administration of dextrose solution produced the syndrome, the serum osmolality falling acutely to 256 m-osmol/kg (the normal range being 280–295 m-osmol/kg). The osmolality was rapidly restored to

normal by the infusion of saline and the child's decerebration disappeared.

The fact that control of osmolality may be impaired in severe brain damage was illustrated by an infant (case 21) who was admitted to hospital suffering from *Haemophilus influenzae* meningitis. He was treated with antibiotics from the time of admission, but 24 hours later he started to have convulsions and developed severe decerebrate rigidity with tachycardia of over 200 per minute, bilateral papilloedema, and fixed dilated pupils. The serum osmolality was found to be low at 255 m-osmol/kg and the osmolality of the urine high at 578 m-osmol/kg. It was felt that the child had cerebral oedema due to water intoxication probably due to inappropriate secretion of antidiuretic hormone. He was treated by the infusion of hypertonic saline (10%) and dexamethasone. This therapy resulted in cessation of the fits, the disappearance of decerebration, and resolution of the papilloedema. On subsequent follow-up examination the child was found to be neurologically normal.

In three other patients too rapid correction of hyponatraemia by intravenous fluid was thought to be responsible for brain swelling accompanied by decerebrate rigidity.

CONTROL OF BLOOD SUGAR Hypoglycaemia (defined from term onwards as a blood glucose of less than 30 mg/100 ml.) was present in six patients, in one of whom the blood glucose was found to be only 5 mg/100 ml. Hyperglycaemia, defined as a blood glucose level of 200 mg/100 ml. or more, was noted in two other patients (cases 3 and 43).

TREATMENT

The patients were given many different treatments during the study because of the many different causes of decerebration and associated abnormalities. Vagal over-activity manifest as cardiac irregularity, bradycardia, or cardiac arrest was corrected by the administration of atropine in two cases (cases 9 and 13), but might have been considered in others. Hypertonic saline was administered in two patients who showed hypo-osmolality. Glucose was given intravenously for the correction of hypoglycaemia in six patients. Attempts to aspirate subdural haematoma through the lateral angles of the anterior fontanelle were made in 14 patients. Raised intracranial pressure associated with hydrocephalus was lowered by removal of cerebrospinal fluid from the lateral ventricle by ventricular tap in 13 patients. Cerebral oedema was treated by a combination of mannitol 20%, 5–7 ml./kg (16 cases), and dexamethasone given parenterally in 30 cases. In newborn infants dexamethasone alone was used. It is our practice to nurse these patients in the sitting position providing an adequate airway can be maintained. Neurosurgical intervention was needed in 15 cases (20%).

FOLLOW-UP STUDIES All the surviving patients were seen as outpatients after they had been discharged from hospital. Twenty children (31%) had died during their acute illness and 19 (30%) were considered to be completely healthy, normal children on later follow-up. Two patients were known to have had evidence of neurological handicap before their acute episode. Their neurological findings were not altered on subse-

quent follow-up examination, although one of them had suffered cardiac arrest during the acute episode (case 13). Seven children (11.5%) were found to manifest ataxia, hyperkinetic behaviour disturbances, mild hemiparesis, or paralytic squints. Sixteen of the patients (24%) showed severe mental and physical handicaps, usually associated with a tetraplegic type of cerebral palsy (Table 6).

Forty-one children suffered from epileptic seizures when acutely ill; in only five did fits occur afterwards.

PATHOLOGICAL FINDINGS Necropsy was carried out in 13 of the 20 patients who died. Many different pathological abnormalities were found. Unequal swelling of the hemispheres had caused shifts of the brain across the midline; shifts downwards were found with tentorial herniation; lateral compression of the brain-stem and infarction of the occipital lobes due to compression of a posterior cerebral artery and coning at the foramen magnum were all seen. In one patient necrosis of the herniated cerebellar tonsils resulted in cerebellar tissue being found in the lumbar theca. Hydrocephalus, subdural haematoma, and encephalitis were confirmed in patients in whom this had been the provisional antemortem diagnosis. In three infants the brain was found to be softened, pale, swollen, and almost liquid, so marked was the cerebral infarction.

DISCUSSION

Sherrington found that after a section of the brain at an intercollicular level there was rigidity in the trunk or all four limbs, with extension of the neck and trunk, adduction and internal rotation of the shoulders, extension of the elbows, pronation of the forearms, flexion of the wrists and fingers—usually over the adducted thumbs—adduction and internal rotation of the hips, extension of the knees, and plantar flexion of the feet so that 'scissoring' of the lower limbs frequently occurred (Sherrington, 1906, 1947). This type of rigidity has been attributed to release of the activity of alpha nerve fibres which control muscle tone in experimental animals, though interpretation is more difficult in the

human (Rushworth, 1964). The second type of decerebrate rigidity described by Denny-Brown (1966) in which there is flexion at the shoulders and elbows so that the upper limbs tend to be held across the chest tends to occur when lesions of the brain are more diffuse in nature, and particularly when the basal ganglia and cerebellum are involved. In animals it has been attributed to the release predominantly of the gamma efferent system in the spinal cord.

The clinical pictures of the two types of decerebrate rigidity are usually quite distinct, but the explanation as to why one or other should occur is impossible to make in terms of presumed underlying pathological change in the brain (Denny-Brown, 1962). Both types may be abolished by deep anaesthesia and the posture and rigidity of the limbs are altered in the gamma type if they are de-afferented. A number of authors have considered that the brain-stem is more often damaged in patients in whom the upper limbs show flexor patterns (Walshe, 1923a; Granit, 1955; Denny-Brown, 1962).

The two types of decerebrate rigidity described must be distinguished from other states in which there is extension or hyperextension of the neck and trunk, and extension of the limbs with rigid increase of tone. In some diseases of the corpus striatum, for example, transient or long-lasting posture of this type may be found—the so-called dystonic syndrome described by Denny-Brown (1963). In this syndrome posture is modifiable by changes of position and by alteration of the position of the limbs. Dystonic postures of this type are commonly found in the early stages of the development of dyskinetic or diplegic cerebral palsy in severely affected patients (Ingram, 1964). They may also be observed in the course of prenatal and early postnatal development, before higher cerebral control of motor function has developed.

Thus decerebrate and dystonic postures may be explained in developmental terms. By 12 weeks of gestation the foetus shows well-developed flexor tone with recoil and adduction and flexion of the limbs, if the corresponding dermatomes are stroked. This response appears to be predominantly spinal in origin. By the 18th week of gestation flexor tone has become inhibited and extensor reflexes appear. The premature baby born at 28 weeks' gestation has

a tendency to mass extension of the trunk, crossed extension of the limbs, trunk incurvation, and tonic neck reflexes. Almost any abrupt stimulation or change in position is liable to produce sudden rigid extension of the neck and trunk and of the limbs, especially the lower limbs with hyperextension of the great toes. Similar extensor patterns have been described in anencephalic infants and have been witnessed personally (Walshe, 1923a; Peiper, 1964).

Between 30 and 40 weeks of gestation there is again an increase in flexor tone, and by term the child's posture is one of semi-flexion of the neck, trunk, and limbs rather similar to that seen between 12 and 18 weeks. After the first month of extrauterine life another extensor phase occurs and reaches its maximum at the age of 12 to 16 weeks (Ingram, 1970). When babies at this stage of motor development are placed in the supine position supported by a hand beneath the back, the posture assumed may be very similar to that found in decerebrate rigidity or in the dystonic syndrome as described by Denny-Brown. It is important to distinguish babies who show a well-marked, but physiological, stage of 'extensor tonus' from babies showing abnormalities of posture and tone attributable to brain disease.

As seen in Table 2 a wide variety of pathological conditions may be associated with decerebrate rigidity. The commonest of these is raised intracranial pressure with associated compression of the mid brain due to coning at the tentorial orifices (Johnson and Yates, 1956). This neuropathological state is most often caused by swelling of the brain secondary to head injury, tumours of the posterior fossa, or hydrocephalus.

Most of the other causes of decerebrate rigidity produce their effects by causing cerebral oedema and raised intracranial pressure. These are found in lead poisoning (Wechsler, 1963), diabetic ketoacidosis (Young and Bradley, 1967), hypernatraemic dehydration (Morris-Jones, 1967), and in cases of pseudo-tumour cerebri (Greer, 1967). Brain swelling can occur in young infants after very trivial trauma and minor hypoxic episodes. It may be associated with the use of certain drugs such as tetracyclines, steroid withdrawal, and vitamin intoxications. There is what appears to be a specific condition causing toxic encephalopathy associated with fatty degeneration of the liver, hypoglycaemia, and

severe metabolic acidosis which occurs predominantly in young children under 5 years of age described by Reye, Morgan, and Baral (1963) and Simpson (1966). Minor scalds may be associated with severe brain oedema and decerebrate rigidity with loss of homeostasis in children under 5 years of age (there were two cases in the present series, nos. 1 and 2) (Emery and Reid, 1962).

The other causes of extensor hypertonus in the child which produce decerebrate postures include diffuse encephalopathic diseases of the mid brain which may occur in encephalitis, tumours, hypoxia or ischaemic lesions. Pure metabolic disturbances may also have a direct effect on the brain-stem causing release of decerebrate rigidity without any associated swelling. This is seen in severe kernicterus (Eastham and Jancar, 1968), maple syrup urine disease (Martin and Norman, 1967), hepatic failure (Conomy and Swash, 1968), hypocalcaemia, hypomagnesaemia (Brown, 1970), and in alkalosis or phenothiazine poisoning. Intracranial haemorrhage accounted for 30% of the present series. The battered baby suffering from intracranial haemorrhage is one of the diagnoses which must always be borne in mind in any infant who presents with unexplained extensor hypertonus.

HOMEOSTATIC CONTROL As indicated above, a breakdown in homeostatic control may be associated with decerebrate rigidity. Such an association may involve abnormalities of cardio-respiratory control, and regulation of osmolality, serum sodium, blood glucose, temperature, and pH in which it is presumed that the disturbance in homeostasis is secondary to the disease process which also results in extensor hypertonus. A possible mechanism would be the pressure effect on the hypothalamus of a swollen brain or of a dilated third ventricle in hydrocephalus. In decerebrate rigidity there is frequently an imbalance between the sympathetic and parasympathetic components of the autonomic nervous system and this may account for the pinpoint or fixed dilated pupils, the bradycardia or tachycardia, the hypo- or hyperglycaemia, the hypo- or hyperthermia, and the other autonomic

disturbances often found (Robertson and Pollard, 1955; Appenzeller, 1970).

Abnormalities of the function of the autonomic nervous system on the circulation may cause hypotension or hypertension and this may be associated with pulmonary oedema (Chapman, Livingston, Livingston, and Sweet, 1950; Paine, Smith, and Howard, 1952; Hilton, 1966; Ducker, 1968). In young infants an odd syndrome of tachycardia, enlargement of the liver, and peripheral oedema may occur secondary to lesions of the brain and has been known as cerebral heart failure (Gray and Prosser, 1967).

Abnormalities of cardiac rate or rhythm and associated disorders of the circulation may be secondary to diseases of the nervous system and may also cause brain damage from ischaemia. Thirty-three per cent of the present series showed some abnormality of the cardiovascular system. There may be vagal stimulation with resultant bradycardia, sinus arrest or nodal escape, and typical changes in the ECG. Alternatively, severe tachycardia (which may impair diastolic filling) can also be due to central sympathetic overactivity. Before bradycardia becomes apparent, there may be marked exaggeration of the normal sinus arrhythmia and irregularities of the pulse may also be due to varying degrees of heart block. Changes in the ECG may occur after subarachnoid haemorrhage when large T-waves, left bundle branch block, U-waves, and prolongation of the Q-T interval may be seen (Shuster, 1960; Srivastava, and Robson, 1964).

Respiratory difficulties were present in 66% of cases. Disorders of pulmonary ventilation may occur when either hyperventilation producing alkalosis and a reduction in cerebral flow (Plum and Swanson, 1959) or hypoventilation is present. Hypoventilation is more common and is associated with retention of carbon dioxide and acidosis. This is dangerous because the retention of carbon dioxide may cause cerebral venous dilatation, further increasing intracranial pressure. Apnoeic attacks or respiratory arrest were seen most often after a period of bradypnoea (McNealy and Plum, 1962). Tachypnoea, bradypnoea, Cheyne-Stokes, or gasping respirations all occurred. Between the two different phases of Cheyne-Stokes respiration, pupillary size, conjugate eye deviation, and plantar responses could all vary. Laryngeal stridor,

recorded by a number of earlier workers, was found in five patients in the present series (Morley, 1969). The acid base status of the CSF was not studied in the present series of cases, but it is known that respiratory abnormality and encephalopathy may be associated with changes in CSF acid-base balance (Posner, Swanson, and Plum, 1965).

In view of the high incidence of respiratory disorders associated with decerebrate rigidity in the present series the adequacy of ventilation should be routinely monitored either as minute volume, end tidal CO_2 , or by sequential blood gas studies. The airway must at all times be kept patent. Early tracheotomy in children with decerebrate state has been recommended (Robertson and Pollard, 1955). This is not without hazard in small children and we found the use of Jackson Rees nasotracheal intubation satisfactory. There is no place for the use of respiratory stimulant drugs in these cases as they may precipitate status epilepticus. Intermittent positive pressure ventilation was required in 20% of cases in the present series because of ventilatory failure.

Temperature regulation depends on hypothalamic function and hypothalamic connections, regulation of secretion of thyrotropic hormone, skin blood flow, shivering and sweating, and an intact autonomic nervous system (Roddie, 1969). Three abnormalities may occur: hypothermia, hyperthermia, and poikilothermia. Hyperthermia has a recognized association with hyperglycaemia and pinpoint pupils in pontine lesions (Brain and Walton, 1969) but hypothermia is said to be more common in diseases of the central nervous system. In the present series, 30% of the cases had a temperature of 35°C (95°F) or less at some point in the acute stage of the disease. Hypothermia is important in the very small infant, as hypoglycaemia, acidosis, and an increase in oxygen consumption may all occur as a result.

The autonomic nervous system is involved in blood glucose control; the sympathetic component causes breakdown of the liver glycogen and inhibition of insulin release (Buchanan, 1969). Through the vagus nerve the parasympathetic nervous system stimulates insulin release (Levine and Haft, 1970). It is established that hyperglycaemia can occur in neurological disease

and that hypoglycaemia may cause neurological disease (Ingram, Stark, and Blackburn, 1967; Neligan, 1969). The role of neurological disease in causing hypoglycaemia is more controversial (Cornblath and Schwartz, 1966; Wilson, 1968). In the present series hypoglycaemia often appeared multifactorial. In four of the cases (cases 6, 33, 58, 64) hypoglycaemia was felt to be secondary to neurological disease (see Appendix).

There appear to be two types of hyponatraemia which may complicate diseases of the brain (Nyhan and Cooke, 1956; Mangos and Lobeck, 1964). The first is manifest as a reduction of the total body sodium and fluid volume so that the patient is clinically dehydrated. The more common type found in neurological disease is associated with hyponatraemia with normal hydration or even oedema. This is most often due to water intoxication and may be due to inappropriate secretion of antidiuretic hormone. In the unconscious child the latter type may also be due to overtransfusion with hypo-osmolar fluids. The need for care in maintaining the fluid and electrolyte balance in unconscious patients can hardly be overestimated, for hyponatraemia or hypernatraemia may cause further neurological damage. Attempts to control hyperosmolar dehydration too rapidly—for example, in hypernatraemia—may result in brain swelling and oedema with decerebration, and this was responsible for the syndrome in three patients. Another condition which must be borne in mind in any acute neurological illness associated with disorders of fluid electrolyte balance is diabetes insipidus. Unsuspected diabetes insipidus may present with hypernatraemic dehydration and be the cause of brain damage.

In the management of a child showing decerebrate rigidity, the first aim must be to obtain an accurate diagnosis of the cause of his neurological abnormality. Prompt treatment of the underlying cause is essential as, for example, in the case of acute bacterial meningitis. In addition systemic treatment of any brain swelling, whatever the cause, must be early and energetic. Whenever raised pressure within the skull is suspected, the patient should be sat up in order to lessen intracranial venous pressure. Hydrocephalus should be relieved by tapping of the lateral ventricle when the fontanelle is open. The

use of osmotic diuretics, steroid drugs, controlled hyperventilation, hypothermia, and hypotension has been reviewed recently by Shenkin (1970). Complicating convulsions may be treated by injecting diazepam with phenytoin intravenously. Disordered activity of homeostatic mechanisms may greatly aggravate the underlying neurological disease and cause death. The pulse rate, temperature, respiration, blood glucose, osmolality of the blood, acid base status, serum electrolytes, and urinary output must all be monitored and any abnormality corrected at an early stage. Neurosurgical consultation should be sought so that any amenable intracranial lesions can be treated early and effectively.

The appearance of decerebrate rigidity is always a serious sign, but with early and appropriate treatment the prognosis is less grave than formerly. Of 64 patients in the present series, 31% died and 36% of the 44 survivors had significant severe handicap. Forty per cent, however, of the total number of children had either no handicap or only a mild to moderate one. Some authors have suggested that the prognosis for patients showing decerebrate rigidity is better in childhood than adults, even after severe traumatic head injury. A mortality of 86% was found in a group of adults studied by Pia (1957). Since the criteria used in the selection of the present series were different from the criteria used in other series, direct comparisons cannot be made with the findings of Robertson and Pollard (1955), Brendler and Selverstone (1970), Gutterman and Shenkin (1970). The mortality for children showing decerebration will depend very greatly upon the causes of the syndrome and the treatment they are given.

We are grateful to Professor J. O. Forfar and the consultant paediatricians at the Royal Hospital for Sick Children, Edinburgh, and the Simpson Memorial Maternity Pavilion for ready access to patients admitted under their care, and to Professor J. O. Forfar for the care with which he read and suggested modifications to the manuscript. We are also grateful to Dr. A. Baine who performed the post mortem examinations and Mr. George Pollock for his interest and support in the investigation of handicapping conditions in childhood.

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APPENDIX TABLE
CLINICAL DETAILS OF 64 PATIENTS

Case no.	Age (yr)	Sex	Aetiology	Severity	Outcome
1	2	F	Burns encephalopathy	Sev.	Dead
2	2	M	Burns encephalopathy	Sev.	Normal
3	2	F	Battered baby	Sev.	Dead
4	10/12	M	Battered baby	Sev.	Blind, epilepsy, mental retardation, hemiplegia
5	3	F	Encephalitis	Sev.	Dead
6	3	M	Encephalitis	Sev.	Dead
7	3	F	Encephalitis	Sev.	Dead
8	4	M	Post convulsion	Mod.	Normal
9	3	F	Intracranial haemorrhage	Sev.	Dead
10	3	M	Anaesthetic cardiac arrest	Sev.	Mental retardation, cerebral palsy
11	2	F	Meningitis ?battered baby	Sev.	Dead
12	2	F	Meningitis	Sev.	Severe mental retardation, cerebral palsy—died 1 yr later
13	3½	M	Intracranial haemorrhage	Sev.	Mild diplegia and hemiparesis unchanged
14	3	M	Encephalitis	Sev.	Dead
15	5	M	Drowning	Mod.	Normal
16	5/12	M	Subdural haematoma ?cause	Mod.	Normal
17	10	M	A.V. malformation	Sev.	Dead
18	15/12	M	Post convulsion	Sev.	Normal
19	2½	M	Hypernatraemic dehydration	Sev.	Hyperkinesia, mild hemiparesis
20	8/12	M	Reye's syndrome	Mod.	Mental retardation, tetraplegia
21	6/12	F	Meningitis	Sev.	Normal
22	9/52	F	Battered baby	Sev.	Dead
23	Neonate	M	Meningitis	Sev.	Dead
24	1/12	M	Congenital CNS malformation	Sev.	Dead
25	Neonate	M	Intraventricular haemorrhage	Mod.	Dead
26	"	M	Intraventricular haemorrhage	Sev.	Mental retardation, tetraplegia, epilepsy
27	6/12	F	Subdural haematoma	Mod.	Normal
28	2/12	M	Subdural haematoma, birth injury	Mod.	Mild ataxia
29	6/12	M	Meningitis	Mod.	Hyperkinetic behaviour
30	7/52	M	Intraventricular haemorrhage	Mod.	Dead
31	Neonate	F	Neonatal hypoxia	Mod.	Dead
32	6/52	F	Hypernatraemia dehydration	Sev.	Ataxia hyperkinesia
33	3/12	M	Cerebral abscess	Mod.	Mental retardation, tetraplegia
34	Neonate	F	Neonatal hypoxia	Sev.	Microcephaly, slow devel. hemiparesis
35	2/12	M	Hypernatraemic dehydration	Sev.	Recovered, died in 2nd attack 7/12 later
36	10/52	F	Septicaemia	Sev.	Normal
37	2/52	M	Encephalitis	Sev.	Dead
38	3/52	M	Kernicterus	Sev.	Dystonic C.P. deaf
39	5/12	M	Meningitis	Mild	Normal
40	4/12	M	Battered baby	Mod.	Mental retardation, hemiplegia
41	2	M	Meningitis	Sev.	Mild ataxia
42	2/12	F	Intraventricular haemorrhage	Sev.	Mild ataxia, diplegia
43	2/12	F	Battered baby	Sev.	Mental retardation, tetraplegia
44	6/52	M	Septicaemia alkalosis	Sev.	Dead
45	1/12	F	Hyperventilation alkalosis	Sev.	Normal
46	4/12	M	Subdural haem. ?cause	Sev.	Normal
47	1½	M	Subdural haem. ?traumatic	Mild	Normal
48	1	M	Anaesthetic cardiac arrest	Sev.	Mental retardation, blind, dystonic tetraplegia
49	1½	F	Intraventricular haemorrhage	Sev.	Mental retardation, cerebral palsy
50	2/12	M	Encephalitis	Sev.	Normal
51	Neonate	M	Neonatal asphyxia	Sev.	Normal
52	"	M	Birth injury, sagittal sinus thrombosis	Sev.	Mental retardation, cerebral palsy
53	"	F	Birth injury	Mod.	R hemiparesis, slow devel.
54	"	F	Septicaemia	Mod.	Normal
55	"	M	Intraventricular haemorrhage	Mod.	Mild ataxia
56	"	M	Birth injury	Sev.	Dead
57	"	M	Neonatal hypoxia	Sev.	Normal
58	"	F	Neonatal hypoxia	Sev.	Microcephaly, mental retardation, epilepsy, diplegia
59	"	M	Birth injury	Mod.	Normal
60	"	M	Over-transfusion (dextrose)	Sev.	Normal
61	2/12	F	Meningitis	Mod.	Mental retardation, hemiplegia
62	6	M	Anaesthetic cardiac arrest	Sev.	Ataxia hemiparesis—unchanged
63	2	F	Haem. encephalopathy (D.I.C.)	Sev.	Normal
64	Neonate	M	Neonatal hypoxia	Sev.	Microcephaly, mental retardation, hemiplegia