Decerebrate rigidity in acute head injury

ALBINO BRICOLO, M.D., SERGIO TURAZZI, M.D., ALBERTO ALEXANDRE, M.D., AND NICOLA RIZZUTO, M.D.

Department of Neurosurgery, City Hospital of Verona and Department of Neurology, University of Padua in Verona, Verona, Italy

A comprehensive study of the motor patterns, usually grouped under the heading "decerebrate rigidity," was carried out in a series of 800 patients with severe head injuries. The incidence of these manifestations was 39.6%, and when they were present chances of survival were reduced from 79.4% to 28.1%. Clinical and electromyographic investigations revealed heterogeneous and unstable motor manifestations that did not fit into the classical groups of experimental models of decerebrate rigidity. Combinations of extensor and flexor attitudes and/or responses were frequently found in the same patient, but could be separated into homogeneous groups. Each recognized postural pattern had its own distinct neurological signs and prognosis. Age did not significantly affect the outcome; however, intracranial expanding lesions (73.5%), impairment of the brain-stem oculomotor system (49.8%), and deep coma (88.9%) all contributed to an unfavorable course. Surgical treatment was effective when performed for intracranial hematomas and in patients with incomplete extensor rigidity. Good recovery was achieved in 16% of decerebrate patients, while 12.1% survived in prolonged coma or with severe disabilities.

All clinical and neuropathological data suggest that extensor motor abnormalities in the acute phase of cerebral traumatic disease do not always conclusively indicate structural brain-stem damage. A critical analysis of so-called "decerebrate rigidity" (rejecting in some instances its Sherringtonian implications) may allow for a more accurate clinical assessment of the severity of head injury.

KEY WORDS • decerebrate rigidity • head injury • neuropathology • brain-stem lesions • coma • prognosis • electropolygraphy •

Terms such as "decerebration," and "decerebrate rigidity," are broadly used to describe the clinical picture characterized by extensor hypertonic phenomena of the skeletal muscles encountered especially in patients with acute severe head injuries. These popular terms embrace various motor abnormalities that do not fit either the original experimental concept of decerebrate rigidity (which implies the disconnection between the telencephalon and brain stem) or the clinical criteria for the assessment of the decerebrate state in man recognized early in this century. From the analogy between human postural alterations and those obtained in animals by Sherrington these terms have come to be considered as indications of brain-stem damage. In 1945, Mollaret and Bertrand sharply criticized the habit of transferring to man the physiological concept of experimental decerebration.

Daily observation of severely injured patients led us to consider the postural abnormalities usually grouped under the term "decerebration" as a very heterogeneous spectrum of motor manifestations indicative of different degrees of severity of the clinical condition. We have made clinical, electro-
Decerebrate rigidity in acute head injury

encephalographic, neurological, and neuropathological studies in 800 patients with head injuries. The collected data, some of which were evaluated by means of statistical calculations, are reported here.

The basic aims of this report are 1) to pinpoint the most definable postural patterns and their association with other neurological signs; 2) to assess the present significance of these phenomena; 3) to appraise the effectiveness of the various methods of management; and 4) to attempt a correlation with the neuropathological findings.

Clinical Material

Management of Patients

This study includes 800 consecutive unselected patients with severe head injuries admitted to the Department of Neurosurgery of the Verona Hospital from January, 1973, to August, 1975. These 800 patients represent 21% of the total 3800 head injury cases admitted during the same period. Most of the injuries were caused by traffic accidents. Immediately or very shortly after injury all the patients in this series were unconscious for 24 hours or longer, with evidence of anatomical or functional impairment of the central nervous system (CNS).

The patient's course from the time of injury was carefully reconstructed. Upon admission and periodically thereafter, we carried out accurate and complete neurological examinations to ascertain the degree of impairment of consciousness (light, mild, and deep coma), and several neurological syndromes (hemispheric, diencephalic, uncal, mesencephalopontine, and bulbar) that indicate different levels of brain dysfunction. In addition, electromyographic (EMG) recordings were initiated as soon as possible and repeated frequently over the following days. They provided many important elements both as complements of clinical evaluation and as reliable yardsticks in the localization of anatomico-functional cerebral lesions.

Immediately after admission each patient was examined by a neurosurgeon. Efficient respiratory function was promptly restored and possible shock was properly treated. Carotid angiography was carried out when necessary and was a routine and urgent procedure in all decerebrate patients. When indicated by angiographic studies and the clinical situation, surgery was quickly carried out; this included such procedures as removal of hematomas or other intracranial expanding lesions, external decompression, reduction of temporal herniation, tentorial split, and hemicraniectomy.

Routine management in the intensive care unit included endotracheal intubation, administration of steroids and dehydrating agents such as mannitol, blood transfusions, adequate hydration, high caloric intake, antibiotics, and anticonvulsant medication. Artificial ventilation was given to more than two-thirds of the decerebrate patients, and their arterial blood gas levels were constantly controlled. Chlorpromazine, benzodiazepine, phenobarbitone, and the like were used to attenuate seizures and autonomic emergency reactions, and to avoid resistance to the ventilator. Hypothermia was applied only in special cases.

Collection of Data

Of the various motor manifestations developed by these patients, our attention was directed to those extensor attitudes caused by exaggerated contraction of the anti-gravitative muscles. Their clinical appearance, their reactivity to several types of stimuli, any longitudinal modifications of associated neurological signs, and vegetative disturbances were noted in detail.

Almost all these patients underwent electromyographic (EMG) recordings obtained by silver discs or pin surface electrodes placed symmetrically on the skin above the muscles at a distance of 3 to 6 cm. The recordings were made on a 16- or 8-channel Galileo electroencephalograph* with a time constant of 0.1 second and at a paper speed of 7.5 to 15 mm/sec. Identical calibration for the symmetrical muscles was established to prevent saturation of the channel when contractions reached a maximum. Besides EMG of flexor and extensor muscles of the four limbs, respiration and electrocardiogram (EKG) were also frequently recorded. The recordings were generally carried out for a sufficiently long time to provide information on the spontaneous course of postural alterations and on their reaction to various stimuli. Thereafter recordings were made one or more times a

*Galileo electroencephalograph manufactured by Officine Galileo, Florence, Italy.
TABLE 1
Incidence of motor signs and mortality rate in 800 patients

<table>
<thead>
<tr>
<th>Motor Signs</th>
<th>Cases</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>decerebrate rigidity</td>
<td>317</td>
<td>39.6</td>
</tr>
<tr>
<td>full</td>
<td>109</td>
<td>34.4</td>
</tr>
<tr>
<td>unilateral</td>
<td>37</td>
<td>11.7</td>
</tr>
<tr>
<td>alternating</td>
<td>125</td>
<td>39.4</td>
</tr>
<tr>
<td>combined</td>
<td>20</td>
<td>6.3</td>
</tr>
<tr>
<td>mixed</td>
<td>5</td>
<td>1.6</td>
</tr>
<tr>
<td>decorticate</td>
<td>21</td>
<td>6.6</td>
</tr>
<tr>
<td>other motor signs</td>
<td>483</td>
<td>60.4</td>
</tr>
<tr>
<td>absent</td>
<td>367</td>
<td>75.9</td>
</tr>
<tr>
<td>hemiplegia</td>
<td>78</td>
<td>16.2</td>
</tr>
<tr>
<td>tetraplegia</td>
<td>38</td>
<td>7.9</td>
</tr>
<tr>
<td>total</td>
<td>800</td>
<td>100</td>
</tr>
</tbody>
</table>

*Figures are at a 95% confidence level.

A. Bricolo, S. Turazzi, A. Alexandre and N. Rizzuto

Results

Clinical and Electropolygraphic Patterns

Of the 800 consecutive patients with major head injuries who suffered impairment of consciousness for 24 hours or more and associated neurological signs, 317 (39.6%) developed decerebrate rigidity during the acute stage of the disease. The clinical picture of decerebrate rigidity was extremely varied and often unstable, but diligent clinical observation and electropolygraphic recordings permitted the detection of some motor manifestations sufficiently homogeneous and recurrent to be grouped into certain postural patterns. Table 1 summarizes our series according to the motor signs and their mortality rate. For practical reasons and without any attempt to introduce new terminology, we have subdivided decerebrate rigidity into the
Decerebrate rigidity in acute head injury

Fig. 1. Some clinical patterns of extensor hypertonus. a. Full decerebrate rigidity. b. Decorticate rigidity. c. Combined decerebrate rigidity. d. Mixed decerebration.

following types: full, unilateral, alternating, combined and mixed decerebrate rigidity, and decorticate rigidity.

Full Decerebrate Rigidity. Full decerebrate rigidity was observed in 109 patients (34.4%). The symptoms consisted of clenching of the jaw and extension of the four limbs, the upper limbs more than the lower; the arms were also adducted and internally rotated, the shoulders lifted, and the feet in plantar flexion (Fig. 1a). Opisthotonus was seldom associated and its presence was accompanied by a more sustained and generalized extensor hypertonus. The EMG tracings showed a more or less continuous tonic activity of the antigravitative muscles. Prolonged electro-polygraphic recording showed postural extensor synergies, expressed by spontaneous or provoked seizures. Despite the presence of typical extensor posturing, in many cases a strong global contraction of both the extensor and flexor muscles was recorded (Fig. 2). Even if not apparent clinically, episodic or continued activity of the extensor muscles was detected by prolonged EMG recordings. As the clinical and neurological pictures worsened, EMG tracings recorded a progressive extinction of the extensor activity up
A. Bricolo, S. Turazzi, A. Alexandre and N. Rizzuto

Fig. 2. Full decerebrate rigidity. Upper: Electromyographic recording during a spontaneous fit. Note generalized activity of all extensor and flexor muscles. R, right; S, sternocleidus; L, left; B, biceps; T, triceps; Q, quadriceps. Lower: Sections of the mesencephalon (a) and pons (b) demonstrating only ischemic necrosis of the ventrolateral part of the pons. Sections embedded in paraffin; Spielmeyer technique.

to the appearance of some flexor activity in the terminal phase. The EEG pattern was not significantly modified by spontaneous or induced decerebrate seizures (Fig. 3).

Unilateral Decerebrate Rigidity. Unilateral decerebrate rigidity was observed in 37 patients (11.7%) who developed extensor motor activity of the limbs on one side and spontaneous or evoked normal mobility on the opposite side. Electromyographic recordings confirmed the clinical evidence.

Decorticate Rigidity. Twenty-one patients (6.6%) showed the typical picture of decorticate rigidity, with triple flexion of the upper limbs, adducted on the trunk, and clenched fists, while the lower limbs were hyperextended (Fig. 1b). This attitude remained unchanged for a long time in the acute course, appearing as a very stable postural pattern. The EMG recordings showed sustained activity of the extensor muscles in the legs and of the flexors in the arms. Simultaneous hyperactivity of extensor muscles in the arms was also sometimes observed.

Combined Decerebrate Rigidity. Twenty patients with severe head injuries (6.3%) developed a more complex postural picture, consisting of extensor rigidity on one side and a decorticate attitude on the other (Fig. 1c). Painful stimuli or spontaneous seizures increased such posturing without modifying the pattern.

Mixed Decerebrate Rigidity. Only five patients (1.6%) showed mixed decerebrate

J. Neurosurg. / Volume 47 / November, 1977
Decerebrate rigidity in acute head injury

![Electroencephalographic and electromyographic recordings during a spontaneous extensor fit.](image)

Fig. 3. Electroencephalographic and electromyographic recordings during a spontaneous extensor fit. Note no changes of the slow high-voltage EEG activity. R, right; F, frontal; C, central; O, occipital; L, left; B, biceps; T, triceps.

rigidity described by Plum and Posner as flexor rigidity of the lower limbs and hyperextension of the upper limbs. This pattern, which may escape inattentive clinical observation, emerges more clearly as a reaction to painful stimuli (Fig. 1d). The EMG, consistent with the clinical appearance, confirmed that the flexor hypertonus of the lower limbs was not stable, but mostly reactive.

**Alternating Decerebrate Rigidity.** The largest group of patients (125, or 39.4%) showed a greatly variable postural attitude, either spontaneously or evoked, consisting of extensor or flexor rigidity in the same limb. This mixture of motor abnormalities was apparently more influenced than the other types by metabolic and respiratory disturbances, as well as by the nature and site of the stimulus. Prolonged stimulus often evoked an immediate extensor response followed by a flexor reaction; sometimes flexion preceded extension. Electromyography derived from the same limb tonic discharges of extensor antigravitative muscles intermingled with discharges of flexor antagonist muscles (Fig. 4). The EEG pattern was not modified by the occurrence of these motor reactions. In only one case did bilateral hypersynchronous discharges appear on the EEG tracing 0.5 to 1 second before decorticate or decerebrate activity, either spontaneously or evoked by even the slightest stimulus.

**Other Motor Abnormalities.** In a small number of patients with the above-mentioned motor abnormalities a Magnus-de Kleijn reflex was evoked by turning the head. However, EMG recordings of many patients showed modified distribution of the postural tonus as a result of head turning, even when clinical evidence was lacking.

Tetraplegia was sometimes observed when no lesions of the cervical spinal cord were present. It was often seen in the terminal stage of rostrocaudal deterioration when decerebrate rigidity had disappeared. Hemiparesis was the only motor alteration in 78 patients.

In the present series of patients, a breakdown of the function of the autonomic nervous system almost always occurred. Abnormalities of cardiac rate or rhythm, tachycardia or bradycardia, systemic blood pressure and temperature disturbances, sweating, tachypnea, and periodic or irregular breathing, were commonly observed. Since these disturbances were modified by therapeutic techniques, their parameters have not been considered in the present investigation. A comprehensive study of the various disorders of autonomic, metabolic, endo-
Consciousness and Ocular Signs

Of the 317 patients with decerebrate rigidity, 282 (89% ± 3.4 SD) suffered deep coma, and 158 (50.2% ± 5.6 SD) showed brain-stem ocular motility dysfunction (Fig. 5). Of the 109 patients with full decerebrate rigidity, 76.1% (± 8 SD) showed signs of dysfunction of both brain-stem reflex ocular motility and pupillary reaction to light and pain; in 16.5% oculomotor control was either preserved or impaired only at the hemispheric level; in 7.4% unilateral third cranial nerve palsy was observed. Of these 109 patients, 103 (94.4% ± 4.2 SD) were in deep coma; the six remaining patients regained a good level of consciousness, even if spontaneous or evoked motor rigidity persisted. Five of these developed no brain-stem signs; one harbored a unilateral third cranial nerve lesion.

Of the 125 patients with alternating decerebrate rigidity, 40% exhibited impairment of the reflex oculomotor system and 88.8% were in deep coma.

The brain-stem oculomotor system was damaged in 32.2% of the 37 patients with uni-
Decerebrate rigidity in acute head injury

lateral decerebration and 89.1% were in deep coma. In eight cases a unilateral lesion of the third cranial nerve completed the classical picture of uncal syndrome. In 30% of the 20 patients with combined decerebrate rigidity there were brain-stem oculomotor disorders; 80% of the 20 cases were in deep coma.

Of the 21 patients with decorticate rigidity, 14 (66.6% ± 20 SD) were in deep coma. Only two patients had involvement of the brain-stem oculomotor system (9.5% ± 12.8 SD).

All five patients with mixed decerebrate rigidity developed both impairment of the reflex oculomotor system and deep coma. Because of the small number of patients, this finding cannot be used for any reliable mathematical calculations.

The statistical analysis of all the above data demonstrated that deep coma percentages differ significantly (probability range > 99%) from the mean values only in full decerebration and decortication, being the highest in the former and the lowest in the latter. As regards ocular signs, the following mathematical deductions can be drawn with a 99.9% probability: their incidence in full decerebrate rigidity is significantly higher than that in decorticate rigidity and in alternating, unilateral, and combined decerebration taken together. Moreover, in cases with full decerebration, ocular disturbances have a significantly different incidence from the other two groups.

Angiographic Findings

All 317 patients with motor extensor activity underwent carotid angiography at least once. Of these, 233 (73.5%) were found to have intracranial traumatic expanding lesions, with a mortality rate of 75.9%, which was higher than that of the 84 cases with no intracranial expanding lesions (60.7%).

A comparison with the incidence of positive angiography in 445 severe head injuries with no motor abnormalities is not possible, because in 278 cases angiography was not necessary and was not carried out. Nevertheless, of 167 neuroradiological examinations performed, 108 gave indication for surgery, and assuming that an insignificant number of intracranial expanding lesions in other patients may have escaped notice, the rate of intracranial expanding lesions in head-injured patients with no posture alterations is 24.2%. In this group as well, the patients with negative angiography had better clinical results (25% mortality) than those with positive angiography (36.3% mortality).

Figure 6 shows the incidence of supratentorial expanding lesions according to motor patterns. The relatively low percentage of the groups with mixed (40%), combined (60%), and full decerebrate rigidity (68.8%), appear at the statistical analysis significantly different (confidence level 95%) from those with alternating decerebrate (78.4%) unilateral decerebrate (78.3%), and decorticate rigidity (80.9%).

Surgical Treatment

Only 146 of 233 patients with angiographic indications were surgically treated; the others were treated conservatively because of multiple or bilateral expanding lesions, excessively small lesions, precarious condition due to extracerebral factors, or
clinical improvement. Operated patients had a mortality rate of 76%, similar to that of the unoperated ones with abnormal angiographic findings (75.8%). On the contrary, in the group with severe head injuries with no extensor motor abnormalities, but with intracranial expanding lesions, operated patients had a lower mortality rate (37.5%) than unoperated (43.7%). The difference in progress between surgically and nonsurgically treated subjects is not constant in each postural pattern (Fig. 7). The survival rate appears not to be significantly modified by surgery in most postural groups, except in unilateral decerebrate and decorticate rigidity groups. The operated patients in the former groups had higher survival rate (57.9%) than unoperated ones (40%); the contrary is shown in the latter groups (46.5% against 67.7%). The results of surgery are greatly influenced by the nature of the lesion: mortality is high in cases of cerebral contusions (80.9%) and when they are accompanied by hematomas (87.3%), and is lower in cases with only intracranial hematomas (54.3%).

Age

The 800 patients with severe head injuries ranged in age from 3 months to 86 years. The mean age of decerebrate patients was 32 years, and that of non-decerebrate patients was 36 years. Figure 8 summarizes the distribution of extensor hypertonus in each age group; the occurrence varies from 28% to 49%. The only significant difference between the various age groups is an unexpectedly marked increase for the fourth decade. The only postural pattern associated with a particular age group is alternating decerebrate rigidity, which affects 72% of infants.

The relationship between age and mortality in decerebrate and non-decerebrate patients is reported in Fig. 9. In patients of all ages without decerebrate rigidity, the mortality
Decerebrate rigidity in acute head injury

The incidence of extensor rigidity in the fourth decade is significantly higher than that of any other age group.

Rate progressively increases as a function of age. The mortality rate in decerebrate patients is constant and almost independent of age. Of the survivors, three-quarters of those with good recovery were under 40 years of age. It is our impression that severe neurological deficits affect younger patients, while mental and psychological disabilities are most frequent in elderly patients.

Outcome

The group of 317 patients with decerebrate rigidity showed the highest mortality rate (71.9%) as compared with that of the total group of 800 patients with major head injuries (44.7%) and with that of patients with no decerebrate rigidity (20.6%).

Figure 10 shows the patient's course according to the type of extensor rigidity he developed. The mortality rates of patients with decorticate rigidity (57%) and combined (55%) and unilateral decerebrate rigidity (54%) significantly differ from the average rate for all severe head injuries (44.7%). A higher mortality rate (70.4%) was found in those patients who developed alternating decerebrate rigidity, that is, flexor-extensor reactivity of the upper limbs and stable extensor posturing of the lower limbs. A still higher mortality rate (84.4%) was observed in the 109 patients who manifested full decerebrate rigidity. All five patients with mixed decerebrate rigidity died.

In the groups of patients with the highest mortality rates, signs of brain-stem involvement were frequently noted to be associated with posture abnormalities. Among the few who developed full or alternating decerebrate rigidity but showed no signs of brain-stem involvement, nearly all survived. On the other hand, patients with combined, unilateral decerebrate, or decorticate rigidity who died had an altered brain-stem oculomotor system. The relationship of fatalities in patients with extensor postural abnormal-

Fig. 8. Percentage of patients with extensor rigidity (crosshatched area) in various age groups. The horizontal dotted lines indicate mean values; the vertical bars are 95% confidence ranges. The incidence of extensor rigidity in the fourth decade is significantly higher than that of any other age group.

Fig. 9. Relationship between age and mortality in decerebrate (thick line) and non-decerebrate (thin line) patients. The vertical bars and dotted lines are 95% confidence ranges. In decerebrate patients age does not affect the mortality rate, whereas in non-decerebrate cases it does to a significant degree.
A. Bricolo, S. Turazzi, A. Alexandre and N. Rizzuto

FIG. 10. Mortality rate (cross-hatched areas) in the total group of severe head injuries and according to motor signs. The vertical bars are 95% confidence ranges.

100 90 80 70 60 50 40 30 20 10
MORTALITY RATE

FIG. 11. Relationship in decerebrate patients between mortality rate and presence or absence of two combining indicants: deep coma and oculomotor disorders. Vertical bars are 95% confidence ranges.

Clinicopathological Correlations

Seventeen of our 31 patients who were studied at autopsy from a neuropathological point of view had shown full decerebrate rigidity, six alternating, three combined, one unilateral, and one mixed decerebrate rigidity. Three patients had developed tetraplegia.

Anoxic neocortical damage was found in all 31 patients in addition to contusions or lacerations. Hemorrhages or anoxic damage to the hemispheric white matter or basal ganglia were frequently associated (Fig. 12). Lesions of the corpus callosum were found in 18 cases. All patients showed signs of raised ICP consisting of pressure necrosis of parahippocampal and/or cingulate gyri, some-

ities to the presence of deep coma and/or brain-stem signs is illustrated in Fig. 11.

If these patients are subdivided into homogeneous classes depending on the clinical indications, the probable range of mortality progressively increases according to the following sequence of combinations: 1) of 35 decerebrate patients in mild coma, eight died (22.8% ± 14 SD); 2) of 159 decerebrate patients with no brain-stem oculomotor disorders, 77 died (48.4% ± 7.8 SD); 3) of 277 decerebrate patients in deep coma, 215 died (77.6% ± 5 SD); 4) of 158 decerebrate patients with fixed gaze, 146 died (95.4% ± 3.4 SD); 5) of 149 decerebrate patients in deep coma with fixed gaze, 144 died (96.6% ± 2.8 SD). No mathematical correlations can be made in these mortality samples, as they have been derived from groups of overlapping patients.

Of the 89 surviving patients (28% of all decerebrate patients) 51 (16%) were considered to have recovered in that they have resumed their former activities. The fewest of these patients are from the full decerebrate rigidity group. Of the remaining 38 survivors, 13 are in prolonged, presumably irreversible coma, while 25 are left with severe disabilities.

The relationship between the duration of decerebrate rigidity and outcome was also investigated. Survivors in a vegetative state or with severe disabilities had gone through extensor posturing for more than 15 days. In the present series one might say that the duration of decerebrate rigidity does not significantly affect chances of survival.
Decerebrate rigidity in acute head injury

FIG. 12. Neuropathological findings in a case of full decerebrate rigidity with 5 days of survival. Upper: Representative sections of cerebral hemispheres showing diffuse hemorrhagic lesions. Lower: The brain stem at the mesencephalic and pontine level, showing no evidence of structural damage. The tearing of the left cerebral peduncle is an artifact. Sections embedded in paraffin; Spielmeyer technique.

Times associated with softening of the medial occipital region. Pressure necrosis of the cerebellar tonsils due to compression against the foramen magnum was observed in seven cases; in one of these infarction of the posteroinferior part of a cerebellar hemisphere was also found. Damage to the superior cerebellar peduncles was observed in six cases.

Of the 17 patients with full decerebrate rigidity, 14 (82.3%) presented gross brain-stem lesions due to raised ICP, consisting of hemorrhages and/or foci of softening of the mesencephalon and upperpons at the lateral or medial part of these structures. In two of these 17 patients foci of spongy necrosis in the peripheral part of the cerebral peduncles and/or of the ventral pons were seen only at microscopic examination (Fig. 2a and b).

Five of our six patients with alternating decerebrate rigidity had hemorrhagic lesions and/or foci of softening in the mesencephalon and upper pons. In one case histopathological examination revealed only a small marginal necrosis of the cerebral peduncles associated with an ischemic necrosis of the lateral part of the base of the pons.

Severe lesions consisting of hemorrhages or areas of softening were detected in the brain stem of two of our three patients with combined decerebrate rigidity. The third case showed no brain-stem lesions at gross ex-
amination. The patient with unilateral decerebrate rigidity showed large hemorrhages and areas of softening in the mesencephalon and pons. The patient with mixed decerebrate rigidity had a large hemorrhagic lesion in the pons at the level of the superior cerebellar peduncle. Transtentorial herniation and mild neocortical contusion were also found. The three patients who had developed tetraplegia following trauma had hemorrhagic lesions in the mesencephalon and throughout the pons.

**Discussion**

Although decerebrate rigidity has long been known to affect patients with head injuries, its significance remains to some extent undefined. This is because most studies in this field have considered decerebrate rigidity simply as a clinical sign that may be present, and have taken for granted its Sherringtonian implication. Thus several studies have emphasized the relationship between decerebrate rigidity and brain stem compression, and the possibility of relief by means of proper neurosurgical procedures. However, the literature does not provide assessment of the incidence of decerebrate rigidity, its clinical features, and its role in epidemiology of severe head injury. Our study was performed on a large number of patients with head injury treated with the standard techniques available in a specialized unit, to assess the actual significance of postural abnormalities in the natural history of cerebral traumatic diseases. Our patients may be considered representative cases of major head injuries since our results are similar to those reported by Jennett, et al. The rate of decerebrate rigidity was 40%, and this proportion was constant throughout the various age groups with no preference for infants and children, as others have asserted.

The most striking finding was that only one-third of the motor abnormalities usually grouped under the term “decerebrate rigidity” fitted the classical picture and even in these cases the decerebrate pattern was seldom stable and spontaneous. Still more rare was the classical pattern of decorticate rigidity. The abnormality most commonly found consisted of a combination of extensor and flexor attitudes or responses. Decerebrate rigidity following head injury includes variable and complex clinical and EMG manifestations that differ from the stereotyped clinical picture. These observations have encouraged us and others to recognize different patterns and varying degrees of severity in decerebrate rigidity.

The only feature that might justify common classification was extensor response. But these postural alterations cannot be properly classified as “decerebrate rigidity” since this term implies, among other things, a stable extensor attitude. Moreover, the argument for subdividing this phenomenon on the basis of clinical and EMG criteria is supported by the different incidence of neurological signs. Each postural pattern has its own neurological signs, thus constituting various clinical conditions not consistent with the same level of impaired neurological function.

Therefore, the practice of simply designating as “decerebrate rigidity” any extensor manifestation in the acute stage of head injury must be critically reassessed; the commonly accepted concept that in man decerebrate rigidity is the clinical expression of a physiological-anatomical result of brain-stem transection must be rejected. The latter statement is supported by the coexistence of full and stable decerebrate rigidity with preservation of consciousness (also reported by Halsey and Downie) and integrity of reflex ocular motility, factors indicating an unimpaired upper brain stem.

Decerebrate rigidity in man has had such a negative prognosis as to be considered a point of no return. Progress in neurosurgery and resuscitation has abolished the concept of irreversibility of decerebrate rigidity, but some authors still consider it a grave clinical phenomenon. Our results confirm that, despite the present improved methods of treatment, the unfavorable prognosis persists; the mortality rate increases from 20.6% to 72% in patients who develop extensor abnormalities after severe head injury. However, our study, unlike others, shows that outcome may depend to some extent on the type of motor abnormalities involved.

After mixed decerebrate rigidity, which is always fatal, the most unfavorable patterns were full and alternating decerebrate rigidity with respective mortality rates of 84% and 70%, far more than that of unilateral, combined, and decorticate rigidity, whose combined mortality rate was about 55%.
Decerebrate rigidity in acute head injury

Associated neurological signs allow better assessment of prognosis for individuals within each group. Ocular signs are the most reliable features in predicting outcome. Fixed gaze and/or fixed pupils in comatose patients are closely related to a fatal outcome, no matter what postural pattern is present. Furthermore, an unimpaired brain stem oculomotor system, although associated with severe postural alterations, implies good prognosis for survival. Likewise, both preservation and mild impairment of consciousness were related to high chances of recovery.

The relationship always found in patients with head injuries between increasing age and higher mortality rate is irrelevant in those with decerebrate posturing. These data, in some aspects agreeing with those of Carlsson, et al., and Jennett, et al., show that the outcome is less closely related to age than is often asserted. This is probably because in non-decerebrate patients extracranial complications play the most important role in the fatalities, while in decerebrate patients the severe brain damage itself endangers life. This opinion is further sustained by the high frequency in decerebrate cases of intracranial expanding lesions that adversely affect recovery.

The chances for survival of these patients seem not to be significantly improved by the removal of intracranial expanding lesions. Yet, surgical treatment is very effective when performed for intracranial hematomas and/or in patients with incomplete decerebrate patterns. It is likely that in these cases the beneficial effect of surgery depends on relief of brain-stem compression and distortion due to lateral or central transtentorial herniations. Many aspects of some of these situations recall the experimental temporal lobe herniations in cats and monkeys reported by Jennett and Stern, who produced a reversible contralateral decerebrate rigidity without midbrain tegmental hemorrhages.

The unfavorable results of surgical treatment of intracranial complications in patients with complete decerebrate or decorticate rigidity suggest that the expanding lesion is not the only determining factor in the neurological picture. In these cases diffuse brain damage may be the major prognostic factor, rather than purely mechanical factors, which have been emphasized for a long time by other authors. Furthermore, the response of the human brain to injury, consisting of local and global cerebral blood flow and metabolism alterations, fluid content, and ICP changes, must be taken into account. The better progress of patients in whom such factors seem to play the main role emphasizes the importance of medical treatment when surgery is not required. Computerized tomography scanning further assists in the definition of neurosurgical indications.

The quality of survival following severe head injury, as emphasized by many authors, is of crucial importance in order to justify the great amount of money and human resources spent in the management of neurosurgical intensive care. The patient most involved in this question is the one who has decerebrate rigidity, since recent reports indicate that for such patients over 20 to 30 years of age there is no recovery. In our series 16% of such patients recovered sufficiently to resume most of their former activities; therefore the abandonment of these patients appears unacceptable. Although the particular pattern of decerebration and some other clinical features may be strongly predictive of an unfavorable outcome, the impossibility of survival cannot be determined early.

The fact that mortality occurs in some but not all decerebrate groups, as well as the spectrum of associated neurological signs and occurrence of severe mental disability suggests that the various decerebrate patterns are not sustained by a homogeneous neuropathological set of lesions. It is important to stress, according to Ommaya and Gennarelli, that even experimental models lack complete description of tri-dimensional distribution of all cerebral traumatic lesions at known time intervals after head injury. As a consequence, correlations between type, severity, and site of lesion and clinical patterns after head injury are not well recognized. Particularly where decerebrate rigidity is concerned, neither experimental models, nor blunt head injuries, nor anatomico-clinical observations in man could reproduce the results of experimental neurophysiology that indicate that the brain-stem transection is the substrate of this clinical phenomenon.

Our neuropathological findings confirm that a lesion produced by blunt head injury is
not confined strictly to the brain stem; if it were a comparison with the classical experimental models would be possible. We found instead structural telencephalic damage variably associated with lesions of both subcortical and brain-stem structures. Intracranial hypertension was another constant feature in our patients. Since postural and movement control is performed at every level of the nervous system by continuous integration and feedback, no definitive conclusions can be drawn from our study on the precise role played by each lesion in producing the postural alterations.

In the majority of cases extensor hyperreflexia coexisted with secondary hemorrhages and/or ischemic necrosis within the rostral brain stem. These data, in agreement with the opinions expressed by some other investigators, confirm the important role played by intracranial hypertension in producing both brain-stem lesions and decerebrate rigidity. However, the most important finding from our data is that not all patients with decerebrate rigidity had structural lesions in the brain-stem areas that were responsible for regulation of postural tone. The absence of this close association in five cases provides further proof that, at least during the acute stage of head injury, decerebrate rigidity does not conclusively indicate brain-stem structural damage. The postural extensor manifestations observed in our five patients may merely be related to an impairment of midbrain functions as a consequence of transtentorial herniation. The experience of complete reversibility of the decerebrate condition following decompression of the brain stem, both in experimental and clinical practice, argues strongly for this mechanism.

It is, however, difficult to accept the idea that a brain-stem lesion may be the common cause for the different posture groups of decerebrate rigidity. Rather, they may be explained in terms of various levels and degrees of anatomico-functional brain impairment in the light of Denny-Brown’s proposition that “removal of one competing factor in the control of movement at any level results in overaction of the others.” Thus, “decorticate rigidity” and “mixed decerebration” might be an expression of dysfunction of opposite levels of performance of motor integration. The former closely resembles the thalamic or decorticate animal sustained by a cerebral dysfunction confined rostrally at the diencephalic or cortico-subcortical level; the latter recalls the behavior of the retrocollicular animal, due to a lesion down to the pons at the trigeminal level.

The remaining and most frequent patterns of decerebrate rigidity in humans following head injury only at times reproduce the profound and pervasive extensor rigidity resulting from the classical investigations of Sherrington, due to a transection of the brain stem caudal to the red nucleus but rostral to Deiter’s nucleus of the vestibular complex. In addition, our observation of the overlapping of extreme rigidity and opisthotonus shows a close analogy with the experimental results obtained by ablation of the anterior lobe of the cerebellum in decerebrate cats, or by ligation of the carotid and basilar arteries. The not uncommon detection in neuropathological studies of lesions of the cerebellum, which are likely to produce a loss of the well known cerebellar inhibitory influence on postural tone, helps to explain these clinical features.

The dramatic character of decerebrate rigidity in animals and the critical level of transection required to produce it seem to be rarely duplicated by human neurotraumatology, which tends to produce clinical syndromes due to more complex and diffuse brain damage. In the light of recent neuropathological investigations, which suggest that primary brain-stem injury does not exist in isolation, one may not exclude the possibility that the lesions extending beyond the boundaries of the brain stem proper might play by themselves the pathogenetic role in producing decerebrate posture. Many clinicopathological and experimental observations, recently reviewed by Feldman and Sahrman, support this view stressing that the extensor posturing is not always indicative of a brain-stem lesion. Moreover, Snider and Woolsey reported the production of extensor rigidity by simultaneous lesions in pericruciate areas of the cerebral cortex and ablation of anterior lobe of the cerebellum in the cat. The assumption that a brain-stem lesion is responsible for extensor decerebrate-like postures following head injury is moderated by our data which suggest that the localizing value of decerebrate rigidity is overestimated.
Decerebrate rigidity in acute head injury

At this point one remembers the statement Clovis Vincent made at the Neurological Society in Paris in 1925: "Actuellement, il ne semble pas qu'on ait le droit d'appliquer à une rigidité humaine le nom de rigidité "décérébrée." On ne peut même pas l'appeler "rigidité type Sherrington," car on ne sait pas si cette rigidité est possible chez l'homme." ("Actually, it does not appear that one has the right to apply to a human posture the term 'decerebrate' rigidity. One is not even able to call it 'Sherrington-type' rigidity for one does not know if this posture is possible in man.")

Summary and Conclusions

1. The motor manifestations of exaggerated extensor tonus which frequently follow severe head injury cannot be classified simply; a complete description of the abnormal postures and responses in individual cases is required. Nevertheless, we feel it is valuable to attempt to classify these motor abnormalities into several homogeneous patterns.

2. Despite the proper modern neurosurgical treatment and intensive-care facilities, the phenomena of decerebrate rigidity are important unfavorable signs because they indicate severe and diffuse anatomico-functional brain damage and a serious clinical condition with increased metabolic demands.

3. Although the brain stem plays a very important role in motor regulation, the extensor postures usually called "decerebrate rigidity" are not always caused by brain-stem damage. Much of the collected data in humans tends to emphasize the importance of the entire amount of brain damage beyond the brain stem itself.

4. The localizing value of extensor motor abnormalities in patients with severe head injuries is not proven. The use of terms such as "decerebration" and "decerebrate state," because of their Sherringtonian implication, should be restricted to define a clinical condition due to severe midbrain dysfunction. This is implied when a stable and complete extensor posture combines with deep coma and brain-stem ocular signs.

Acknowledgments

The authors wish to thank Dr. Franco Marcolini, data processing manager of the City Hospital of Verona, for his invaluable advice on data processing and statistical methodology, and Mrs. Francesca Pistorelli for her assistance in preparation of the manuscript. The cooperation of the nursing staff of the Intensive-Care Section is gratefully acknowledged.

References


32. Fisher RA, Yates F: Statistical Tables for Biological, Agricultural and Medical Research. Edinburgh/London: Oliver and Boyd, 1953


47. Jennett B, Stern WE: Tentorial herniation, the
Decerebrate rigidity in acute head injury


73. Pompeiano O: Personal communication, 1976

74. Rademaker GGJ: Die bedeutung der roten Kerne und des Übrigen Mittlehirns für Muskeltonus, Körperstellung und Labyrinthreflexe. Berlin: Springer-Verlag, 1926


79. Sherrington CS: The Integrative Action of the Nervous System. New Haven, Conn: Yale
87. Vincent CI: Discussion. Rev Neurol 2:115, 1925

This paper was presented in part at the Annual Meeting of the Italian Neurosurgical Society, Rome, 1975.
This study was supported by the N.H. Marastoni Foundation of Verona and by Consiglio Nazionale Ricerche Grant 74.00270.04.
Address reprint requests to: Albino Bricolo, M.D., Department of Neurosurgery, City Hospital, Verona, Italy.