TREATMENT OF DECEREBRATE ACTIVITY FOLLOWING SEVERE CRANIOCEREBRAL TRAUMA: REPORT OF 2 CASES WITH DETAILED PHYSIOLOGICAL AND METABOLIC OBSERVATIONS

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Decerebrate rigidity following severe craniocerebral trauma is an ominous sign which is associated with a high mortality. It may be the result of specific injury to the brain stem or secondary to tentorial herniation from increased intracranial pressure due to an expanding surface hemorrhage or cerebral edema. Although the mortality rate is high, the decerebrate activity is potentially reversible and survival with good return of function possible[1-4]. The prompt evacuation of an expanding intracranial mass have resulted in recovery in those patients whose etiology was related to surgically amendable lesions[4,5]. In the non-operative cases MacIver reported a marked decrease in mortality rate when vigorous management was instituted to restore a stable physiological and metabolic state as well as prevention of pulmonary complications[2]. Although the clinical syndrome of decerebrate activity, hyperventilation, hyperpyrexia and tachycardia are well recognized, physiological and metabolic abnormalities which presumably account for the high mortality have not been well documented.

Two cases are presented who developed documented profound physiological and metabolic derangements during the decerebrate state which in themselves might have been deliterous to the recovery of the brain and potentially lethal. Control of the decerebrate activity restored homeostatic balance spontaneously. The detailed observations

are presented to demonstrate the interdependent relationship of the brain injury with alterations in homeostatic mechanisms leading to profound physiological and metabolic derangements. These in turn perpetuate a vicious cycle. The insights gained on the possible mechanisms of the abnormalities observed and the therapeutic implications derived may offer another consideration of therapeutic regimen in the management of severe CLOSED head trauma.

Case #1 A.M.

This 22 year old white male, involved in an automobile accident, was admitted to the neurosurgical service on 12/4/68. There was no evidence of skull fracture and a right carotid arteriogram was normal. He was in deep coma, responding only with decerebrate activity with marked muscular rigidity, opisthotonous and hyperventilation following painful stimulation. His blood pressure was 140/60; pulse was 80 per minute and regular during a quiet state. An endotracheal tube was inserted. The patient was treated with Solumedrol and oxygen. Twelve hours later he was transferred to the Shock Trauma Unit for evaluation and management.

His initial observations on admission to the Shock Trauma Unit were (Table I): Blood Pressure - 120/80 mmHg, CVP - 1.0 mmHg, pulse 168 beats per minute, respirations - 36 per minute and temperature - 101°F.

His arterial blood gas was: pH - 7.23, PCO_2 - 27 mmHg, PO_2 76 mmHg and base excess of (-16) MEQ. His central venous PO_2 was 19 mmHg. His

hemoglobin was 13.1 gm% and hematocrite was 38 vol.%. His serum osmolality was 300 mosm. The initial cardiac output was 8.47 L/minute. The A-V difference was 12.47 ml/100 of blood. His estimated oxygen consumption was 1040 cc/minute.

His primary problem was repeated episodes of decerebrate activity with hyperventilation. He was also hypoxemic, hypocapneic and demonstrated severe metabolic acidosis. There were marked hemodynamic swings related to his decerebrate activity. A tracheostomy was performed to improve tracheal toilet and he was placed on mechanical assisted ventilation. Despite his improvement in oxygenation, he continued to have repeated episodes of decerebrate activity with profound physiological and metabolic abnormalities for the first 24 hours. He was then sedated with morphine and cooled to a temperature of 98^{o}F. with a cooling blanket. His decerebrate activity dramatically subsided and his physiological and metabolic abnormalities corrected spontaneously. He required intermittent sedation for the next 6 days, to control his decerebrate activity. He was transferred to the neurological service on 12/22/68 for further care. Although his neurological status improved slowly, responding to verbal stimuli by the 5th week. He suddenly expired following a klebsiella septicemia, 7 1/2 weeks post injury.

Case #2 W.T.

This 9 year old negro male was admitted to the Shock Trauma Unit on 2/16/70 because of a head injury sustained when he was hit by a truck. He also sustained a fracture dislocation of the right ulnar and dislocation of the radius. The arm was treated with closed reduction, insertion of

Kirshner wires and long arm cast without any complications. He was comatose and had repeated episodes of decerebrate activity with hyperventilation, primarily in response to any external stiumlus.

His initial observations were (Table II): Blood pressure 100/60, pulse 140 per minute, CVP-O, respirations 28 per minute and timperature 98.6°F. His arterial blood gas was: pH - 7.31, PCO_2 23 mmHg, PO_2 - 86 mmHg on room air and base excess - (-10.6) MEQ. His hemoglobin was 11.6 GM% and hematocrit was 30 vol. %. His serum osmolality was 292 mosm. His cardiac output was 3.65 L/minute.

He was treated initially with nasal tracheal intubation, oxygen, Solumedral and Dilantin. Despite adequate oxygenation he continued to have repeated episodes of decerebrate activity with profound physiological and metabolic abnormalities over the first 24 hours. A tracheostomy was performed to improve tracheal toilet and he was then sedated with Valium. His decerebrate activity dramatically subsided and his physiological and metabolic abnormalities corrected spontaneously. He required intermittent sedation for the next 5 days to controll his decerebrate activity and become responsive to verbal stimuli 6 days post injury. By the time he was transferred to the neurological service for further care on 2/26/70 he was responding to simple command. He was discharged from the hospital 9 1/2 weeks post injury ambulating with minimal ataxic gait.

Methods:

On admission to the Shock Trauma Unit a central venous catheter and arterial catheter were inserted percutaneously via the median basilic veins and femoral artery respectively. The CVP catheter tip was positioned in the

upper portion of the right atrium, confirmed by chest x-ray. The catheters were connected to Statham transducers (Statham P 23 and P 37) and monitoring system for continous recording of pressures, EKG and rectal temperatures.[1] Arterial and mixed venous blood samples were drawn for measurements of blood gas using the Instruments Laboratories (Model 113).[2] Blood lactate was measured by the Boehringer Mannheim enzyme method. The osmolality was measured by using an Advanced Instruments Osmometer.[3] Cardiac outputs were measured by the method of Stewart and Hamilton using indocyanine green and a Beckman Cardio densitometer.[4] The minute and tidal volumes were measured on Case #2 by the Wright respirometer. The expired air was analyzed in the Fisher-Hamilton Gas partitioner.[5] The oxygen consumption was estimated with the cardiac output and A-V O_2 difference in the blood utilizing the Fick principle. In Case #2 the oxygen consumption was also determined by measuring the expired gases.

Results and Discussion

The physiological and metabolic observations noted on admission reflect the dynamic interactions of the patients response to injury. Although the initial blood pressures were normal in both cases, apparently taken during a quiet state, the pulse rates were markedly increased in both patients. The cardiac output in Case #1 was slightly increased and subsequent studies in Case #2 was also increased indicating that the heart was functioning at a higher capacity.

Despite the lack of evidence for perfusion failure severe metabolic acidosis was noted in both patients. In case #1 the serum lactate was

1. Statham, Oxnard, California
2. Instrumentation Laboratories, Waltham, Mass.
3. Advanced Instruments Osmometer, Newton Highlands, Mass.
4. Beckman Instruments, Mountainside, New Jersey
5. Fisher-Hamilton Gas, Pittsburgh, Pa.

abnormally increased. Whether this is related to wash out from the injured brain cannot be determined from our present studies. With the wide A-V differences and decrease in hemoglobin concentration in the blood along with the hyperpyrexia and increased oxygen consumption, a relative hypoxia might be considered since case #1 already demonstrated a mild hypoxemia. However his PaO_2 decreased further during the decerebrate state despite the hyperventilation. Since neither patients were in a steady state metabolic derangements probably reflected the combined resting and decerebrate states. Severe hyperventilation accompanied the decerebrate activity with concommitent severe hypocapnea. The episodic nature of the hyperventilation suggested a central etiology rather than a compensatory mechanism to the acidosis.

The marked hemodynamic swings are well documented in both patients and clearly demonstrate the excessive demands on the heart during the decerebrate states (fig. 1, 2, 3). Since these swings were episodic and transient,the continuous monitoring in these patients enabled us to document these changes and made us aware of the gravity of these abnormal swings on the hemodynamic balance of these patients. The effects of the hemodynamic swings, hyperpyrexia and hyperventilation are reflected by further increases in oxygen consumption. Case #1 increased his oxygen consumption more than 5 times the extimated normal resting value. He also widened his A-V O_2 difference to maximum levels of oxygen extraction. The metabolic swings were not as apparent but still demonstrated severe changes even at rest, indicating that serious metabolic effects were continually present even in the quiet states.

The changes observed in both patients after sedation were dramatic. The decerebrate activity subsided and the hemodynamic swings were controlled. The metabolic acidosis was corrected without the use of any alkalinizing agents. Serum lactate in case #1 returned to normal. The most striking change was the decrease in the A-VO_2 difference and decrease in O_2 consumption to a basal resting level. Not only was there restoration of the homeostatic balance but the clinical management and prevention of complications were also greatly facilitated since the hyperactive state was no longer a problem.

The syndrome of decerebrate rigidity, hyperpyrexia and hyperventilation in patients with severe craniocerebral trauma reflect a progressive deterioration of neural function due to direct injury or to hypoxia. The excessive oxygen requirements over prolonged periods with marginal compensation of oxygen delivery lead to profound alterations in the homeostatic balance of the patients. When aggravated by pulmonary dysfunction and hypoxemia the problem is further aggravated. With the presense of metabolic acidosis due to direct brain injury or to hypoxia conditions are now established to perpetuate a vicious cycle. This leads to progressive deterioration and death of the patients, a frustrating situation described clinically for many years[6]. The two patients presented similar clinical features as those previously described. Although the specific etiology of the decerebrate activity was not determined in our patients they were associated with episodes of severe hyperventilation. Thus, a specific mid brain lesion[7] or tentorial herniation secondary to cerebral edema were the probable etiologic cause. A surface hemorrhage was not evident in the patients.

The effects of severe hyperventilation on cerebral blood flow, cerebral oxygen consumption and hypoxia have been reported by a number of investigators[8-17]. Plum and Posner reported a marked decrease in sagittal sinus PO_2 following severe hyperventilation in dogs. This was indicative of a concommitent decrease in cerebral blood flow. The CSF lactate was markedly increased without an associated systemic acidosis which suggested that selective cerebral hypoxia was present [8]. Gotoh et al reported changes in EEG when the jugular venous PO_2 fell below 21 mmHg during hyperventilation. The relative hypoxic changes were due in part to the Bohr effect shifting the Hemoglobin Dissociation curve to the left as well as a decrease in cerebral flow. This has also been reported in dogs by Mallete[10]. Meyer also reported changes in EEG due to hyperventilation due to constriction of cerebral blood vessels[11]. Froman also reported changes in the state of consciousness due to hyperventilation as well as a decrease in CSF pH[12-14].

Kao reported increases in oxygen consumption with induced muscular work in animals[15]. Schmidt et al reported increase in oxygen consumption with seizure activity[16] and Kety demonstrated increases in cerebral VO_2 during active hyperventilation and not with passive hyperventilation[17]. Although controlled hyperventilation has been used effectively to control increased intracranial pressure in a number of investigators[13-20], this must be weighed against the hazards of uncontrolled hyperventilation or controlled hyperventilation which decreases cerebral blood flow beyond a critical level or which causes severe hypocapnea which shifts the hemoglobin dissociation curve to the left, preventing an adequate oxygen release.

The catastrophic consequences of respiratory complications and metabolic and acid-base abnormalities have been reported by a number of Investigators[21-24]. Prevention of these problems have been major factors in decreasing the mortality rate. Although monitoring of specific events in the brain was not accomplished in the 2 cases presented here, the profound changes observed might have had deliterious effects on the brain had they not been corrected.

The detailed monitoring of the physiological and metabolic changes which followed the severe craniocerebral trauma have given us insights into the interdependent relationships of the physiological and biochemical responses to brain injury and also demonstrated the importance of close monitoring of these changes in order to prevent a progressive neural deterioration.

A simple therapeutic regimen has been established which has proven to be quite effective. This includes early angiography to rule out expanding mass lesions, sedation to control the decerebrate activity and vigorous support of pulmonary functions and acid base balance. Controlled mechanical hyperventilation to decrease oxygen requirements and to prevent excessive swings in PCO_2 by breath to breath analysis of Alveolar gas utilizing a mass spectrometer is being reported in a separate paper[25].

Summary:

Two cases of craniocerebral trauma with profound physiological and metabolic derangements during decerebrate activity are presented. Marked hemodynamic and respiratory swings were noted with the decerebrate activity along with hyperpyrexia and marked metabolic acidosis. Excessive

oxygen consumption with increases in up to 5 times the basal requirement was noted which may have contributed to the relative hypoxic state of the brain. Control of the decerebrate activity and prevention of pulmonary and metabolic complications restored the homeostatic balance of the patients to normal. The value of close monitoring in these patients in order to document these profound disturbances and the therapeutic implications derived from them have allowed us to consider new therapeutic regimens for the treatment of severe craniocerebral trauma.

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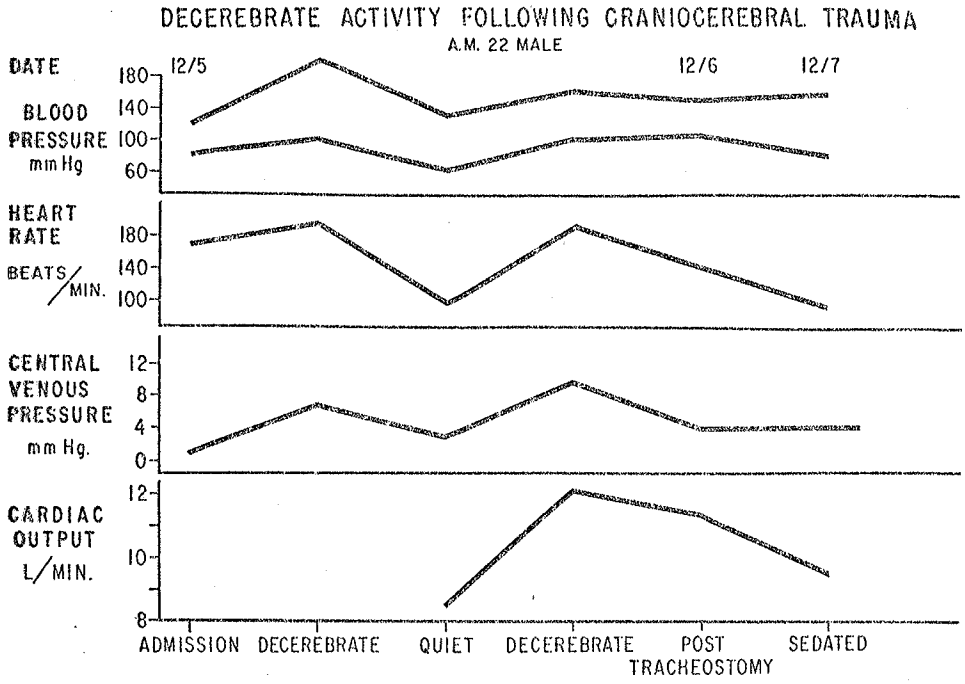
Fig. 1 - Case #1 Hemodynamic swings during recurrent decerebrate activity. Stabilization with sedation.

Fig. 2 - Case #2 Changes in cardiopulmonary function, A-VO_2 difference and oxygen consumption during recurrent decerebrate activity. Stabilization after sedation.

Fig. 3 - Case #2 Marked swings in blood pressure during recurrent decerebrate activity.


DECEREBRATE ACTIVITY FOLLOWING CRANIOCEREBRAL TRAUMA
A.M. 22 MALE
DATE
12/5
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BLOOD PRESSURE mm Hg
180
140
100
60
HEART RATE BEATS/MIN.
180
140
100
CENTRAL VENOUS PRESSURE mm Hg.
12
8
4
0
CARDIAC OUTPUT L/MIN.
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ADMISSION
DECEREBRATE
QUIET
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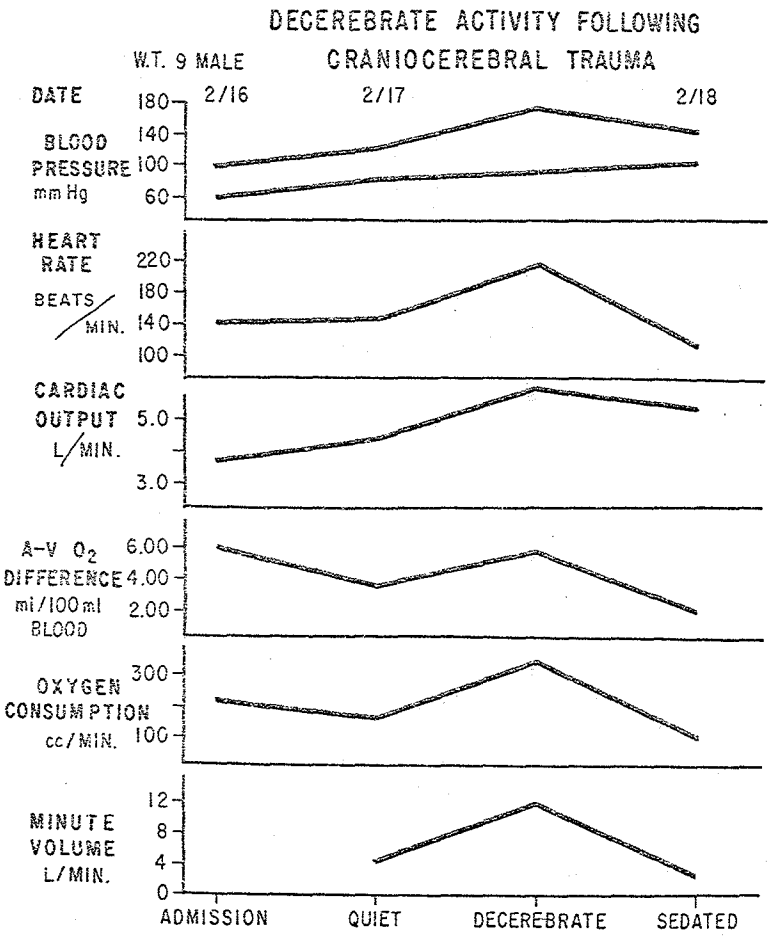
POST TRACHEOSTOMY
SEDATED


DECEREBRATE ACTIVITY FOLLOWING
CRANIOCEREBRAL TRAUMA
W.T. 9 MALE
DATE
2/16
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BLOOD PRESSURE mm Hg
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HEART RATE BEATS / MIN.
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140
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CARDIAC OUTPUT L/MIN.
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A-V O2 DIFFERENCE mi/100ml BLOOD
6.00
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OXYGEN CONSUMPTION cc/MIN.
300
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MINUTE VOLUME L/MIN.
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