EMERGENCY MANAGEMENT OF THE CRITICALLY ILL

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Preface

Over the past decade we have witnessed great advances in the management of critically ill patients. Units equipped to handle medical emergencies are now commonplace and are staffed by personnel trained in intensive care medicine. Most units have devices to assist ventilation, monitor hemodynamic and other physiological parameters, provide laboratory measurements and signal the onset of a serious event.

Management of critically ill patients involves the coordinated activity of many health workers. In addition to physician and nurses, well-run units require a team of technicians to set up monitoring devices, service equipment and assist in making measurements of physiological parameters. These advances aside, it is our understanding of the etiology and pathophysiology of disease processes which has probably been the single, most important factor in the provision of better care. Research has strengthened the theoretical and experimental basis for the principles which guide physician decision-making. It is the skillful application of this new knowledge to the clinical setting to which this volume addresses itself.

The clinical problems discussed are from the continuing education course on the treatment of the critically ill held at the Mount Sinai Hospital, Toronto, Ontario. Current concepts of illnesses and their management are outlined. Panel discussions which incorporate questions from practicing physicians are found throughout the book and often highlight controversies and speculate on future advances.

The problems selected are those most likely to be encountered in clinical practice. The focus is primarily on cardiovascular and respiratory diseases, fluid and electrolyte problems and acid-base disorders. In addition, there are papers on metabolic and renal abnormalities, gastrointestinal emergencies, drug overdose and management of the comatose patient. Thus, this volume should be a useful reference source for physicians involved in the treatment of critically ill patients.

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John Edmeads, M.D., F.R.C.P.(C), F.A.C.P.

Objectives

1. To outline the immediate treatment of any patient in coma.
2. To present a brief systematic examination that will provide an understanding of the physiologic process causing coma.
3. To indicate the procedures for establishing a definitive diagnosis and beginning specific treatment.

Introduction

Coma is one of the most alarming emergencies because the patient is obviously a hair's breadth from death. The management of this problem includes (1) immediate measures to preserve life and save the brain; (2) a rapid decision as to whether neurosurgical or medical investigation and treatment is indicated; and (3) specific therapy.

The dimensions of the coma problem are as follows. A few patients brought to an emergency department in coma have simple concussion from a known head injury or postictal coma following an observed seizure. These patients usually regain consciousness quickly and are neither a diagnostic nor a therapeutic problem. In most patients, however, coma persists, diagnosis is unclear and treatment is urgent. Of such patients, about two thirds have diffuse disturbances such as intoxications or hypoxia which produce coma by slowing the metabolic

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machinery of those neurons which subserve consciousness ("metabolic coma"). The remaining one third is split fairly evenly between those with a cerebral mass lesion displacing the brainstem downwards and damaging the neurons within it that mediate consciousness ("supratentorial mass lesion") and those with a lesion in or adjacent to those brainstem neurons ("subtentorial lesion").

Immediate Treatment

Regardless of the cause of the coma, the treatment immediately necessary to save brain and life is the same. This includes:

A. Airway
B. Breathing
C. Circulation
D. D.E.F.G.G. Draw blood to Examine For Glucose, then Give Glucose (50 cc of 50% solution IV). The glucose should be given immediately without waiting for the blood glucose result. There are few if any situations where 50 cc of 50% glucose will harm a patient, but it can be life-saving if the patient is hypoglycemic.

There are a few clinical saws which still cut keenly. These include:

1. More comatose patients die from airway obstruction or ventilatory insufficiency than from brain damage.
2. Most patients with severe head injuries are injured in other parts of the body as well and can bleed to death from a ruptured spleen while the physician is trying to decide whether the knee jerks are symmetrical.
3. Every head injury is a neck injury as well until proven otherwise; a brisk neck twist can evoke an oculocephalic response, proving that the brainstem is intact, while it renders the patient permanently quadriplegic by severing the spinal cord.

Appropriate Examination

After the above treatment has been given, the patient is examined in more detail. Many parts of the neurological examination such as sensory testing and coordination are

inaccessible in the comatose patient; fortunately, they are unnecessary. Examination of the following few neurologic parameters will permit diagnosis, provided that changes in these parameters are recognized by repeated examinations.

Level of Consciousness

Consciousness is mediated by the reticular activating system (RAS), a neural network joining the cerebral cortex to the brainstem. The cortex determines the content of consciousness and the brainstem determines the fact of consciousness. A moderate-sized lesion in the cerebrum may not appreciably affect consciousness; a small lesion in the brainstem may ablate it.

A variety of terms are used to denote disturbed consciousness. *Drowsiness* is a self-explanatory term. *Stupor* is the next lower stage of consciousness; left undisturbed the patient is unconscious of and unresponsive to his environment, but when stimulated by noise, movement or pain he rouses and is communicative with those about him; on cessation of stimulation, consciousness is lost again.

In the next lower stage, *coma*, the patient cannot be roused to communicate. Coma is therefore a very broad term, but can be focused into useful and meaningful subgroups by observing the patient's response to stimuli. In light coma the patient reacts to pain with a semipurposive withdrawal or "fending off" movement of his limb(s). In a somewhat deeper level of coma he may assume, either spontaneously or in response to stimuli, a *decorticate posture*. In this posture, which is the "motor output" of a brain functioning without its cerebral cortex, the upper limbs are flexed and the lower limbs are extended. In a still deeper level of coma *decerebrate posture* may occur; here the motor output is from the brainstem alone, with no contribution from the cortex and basal ganglia; all four limbs are tonically extended. In the most profound coma the limbs are flaccid.

Sufficient data must be recorded so that the patient's progress or deterioration can be appreciated. "Comatose" is an inadequate description. "Comatose-unresponsive to noise and shaking; responds to painful supraorbital pressure by assuming decorticate posture" is more useful.

Optic Fundi

While papilledema is the first thought that comes to mind when considering fundoscopic examination, other findings such as diabetic or hypertensive retinopathy or subhyaloid hemorrhages may be equally important in diagnosis. Note that while papilledema indicates increased intracranial pressure, the absence of papilledema in no way guarantees that the intracranial pressure is normal.

Pupils

Two opposing neural systems control the pupils. The dilator fibers (the sympathetic system) course from the hypothalamus downwards through the length of the brainstem and the cervical cord, then leave the central nervous system to ascend to the orbit by climbing up the carotid arterial system. The constrictor fibers (the parasympathetic system) originate in the upper midbrain and leave the brainstem at that level, in the third cranial nerve, passing forwards across the tentorial gap just under the overhanging hippocampal uncus and entering the orbit. Both the sympathetic and parasympathetic systems are influenced by impulses which descend upon them from the cerebrum, though this cerebral contribution is not well worked out.

A cerebral lesion does not produce striking pupillary changes, though the pupils may be slightly small because of hypothalamic involvement. As downward pressure from a cerebral mass lesion damages the brainstem in a rostrocaudal progression, the pupils dilate because of midbrain dysfunction and become unreactive to light. Simultaneous involvement of the sympathetic system does not, as might be expected, limit the size of the pupil through loss of "dilator tone," since the denervated pupil is hypersensitive to circulating adrenalin and may dilate widely on this basis. Unilateral pupillary dilatation usually means a third nerve lesion, due in the comatose patient most often to compression of the nerve by a herniated hippocampal uncus.

A lesion confined to the pons (e.g., hypertensive pontine hemorrhage) may destroy the sympathetic pupillodilator fibers, sparing the parasympathetic pupilloconstrictor fibers which have left the brainstem well above the lesion. The unopposed

constriction produces pinpoint pupils — tiny pupils which will react to a bright light. These are very suggestive of a primary subtentorial lesion; the only other thing that will produce them that is reasonably common is opiate overdose.

Eye Movements

Comatose patients cannot be commanded to move their eyes or to follow a moving object, but a brisk rotatory movement of the head to one side imparted by the examiner will elicit reflex conjugate movement of the eyes to the other side *if* the brainstem oculomotor mechanisms are undamaged. If this *oculocephalic maneuver* does not elicit eye movements, then a more potent stimulus should be applied to the brainstem oculomotor centers. This involves irrigating one ear canal with about 10 cc of ice water (after first making sure that the tympanic membrane is intact). The thermal change sets up a convection current in the semicircular canals of the inner ear, which sends impulses over the eighth nerve, thence through the median longitudinal fasciculus to the oculomotor centers, producing conjugate deviation of the eyes to the irrigated ear. Failure of the *oculovestibular maneuver* to elicit eye movements means severe brainstem damage.

Reflexes

A reflex asymmetry is useful in deciding the side of the lesion, though in deeper levels of coma (i.e., in those with prominent brainstem dysfunction) the reflexes are symmetrically hyperactive. In the most profound level of coma all reflexes — tendon, plantar, gag and corneal — are ablated, and this is an ominous sign.

Respiratory Pattern

Although the "respiratory center" is popularly conceived as occupying the medulla, all levels of the central nervous system from cortex to medulla influence respirations.

Removal of the cerebral modulation of respiration, as with a supratentorial lesion, produces periodic or Cheyne-Stokes respirations. Some pontine lesions are associated with tachypnea; in practice, however, this "central neurogenic hyperventilation" seems uncommon, and the finding of hyperventilation in an

obtunded patient is more likely to be due to metabolic acidosis. Medullary lesions may be associated with irregular, gasping and ultimately failing respirations.

Pulse and Blood Pressure

While all levels of the central nervous system influence pulse and blood pressure to some degree, faltering of these vital signs usually inplicates a medullary lesion. However, in increased intracranial pressure the pulse may slow and blood pressure rise as normal homeostatic mechanisms.

Other Features

Other parts of the physical examination must not be forgotten. These include a thorough search for evidence of cranial trauma; a search for nuchal rigidity which might indicate meningitis, subarachnoid hemorrhage, or herniation of the cerebellar tonsils through the foramen magnum; a sniff for the odors of alcohol, hepatic coma, etc.; and careful inspection for needle tracks and other evidence of drug administration.

History is essential, and relatives, friends and/or police should be questioned about the patient's health and habits and about the circumstances in which the patient was found.

Coma Syndromes

On the basis of the physical findings and changes in them, the patient can be recognized as falling into one of three distinct coma syndromes. This categorization is the key to further investigation and definitive treatment.

Supratentorial Mass Lesions

In order of frequency these include:

1. Large cerebral infarction with mass effect from edema.
2. Intracerebral hemorrhage (hypertensive, traumatic, aneurysm or AVM).
3. Subdural and extradural hematoma.
4. Cerebral neoplasm.
5. Others (brain abscess, hydrocephalus, inflammatory edema, etc.).

The clinical hallmark of this syndrome is the orderly, progressive and often accelerating decline in function that

occurs as the brain is telescoped downwards with successive damage and dysfunction of cortex, cerebrum, midbrain, pons and medulla (Table 1).

There is a variant of this pattern of progressive downward displacement that is extremely important — *transtentorial hippocampal herniation*. Here, the cerebral mass lesion pushes the hippocampal uncus down through the tentorial notch, and this extra mass within the notch "jams" the brainstem tightly, producing rapidly deepening coma and respiratory failure. As the hippocampal uncus passes through the notch, it compresses the subjacent third cranial nerve, producing an ipsilateral fixed dilated pupil. The finding of a *unilateral fixed dilated pupil* in an obtunded patient is one of the most crucial signs in clinical neurology. It tells the physician that there is a mass lesion, which side the lesion is on and that without treatment death is imminent.

Clearly, the physician must not wait until the last act of this progressive deterioration. As soon as the fact of this deterioration is appreciated, the following management should be begun:

1. Immediately send for a neurosurgeon and while awaiting his response lower the intracranial pressure by infusing intravenously 250 ml of 20% mannitol solution over a period of 15 to 20 minutes. The patient should have an indwelling urinary catheter to accommodate the ensuing diuresis. A patient with heart disease who might be catapulted into congestive failure by a sudden increase in intravascular volume should, of course, be given the mannitol at a slower rate.

2. While the mannitol is running perform a CT scan (if available) or a carotid angiogram in order to delineate the location, extent and nature of the cerebral mass lesion.

By the time this has been completed, the neurosurgeon has arrived and can proceed with definitive surgical therapy such as removal of a tumor, hematoma or abscess, or shunting of hydrocephalus. The edema that surrounds massive infarcts or inflammatory lesions is usually managed medically, though rarely subtemporal decompression may be necessary. Management of cerebral edema over a longer term than the first few hours involves the use of corticosteroids, since repeating mannitol infusions more than a few times may provoke disturbances of fluid and electrolyte balance. Steroids require

Table 1.

LEVEL OF DYSFUNCTION	LEVEL OF CONSCIOUSNESS	MOTOR RESPONSE	PUPILS	EYE MOVEMENTS	REFLEXES	RESPIRATIONS	PULSE + BP	PROGNOSIS
CORTEX AND CEREBRUM	DROWSINESS ↓ STUPOR ↓ LIGHT COMA	SEMIPURPOSIVE ↓ DECORTICATION	EQUAL, REACTIVE, SOMEWHAT SMALL	OCULOCEPHALIC AND OCULOVESTIBULAR INTACT	INTACT OR HYPERACTIVE	NORMAL ↓ CHEYNE-STOKES	STABLE	SALVAGABLE WITH PROMPT TREATMENT
UPPER BRAINSTEM	DEEPENING COMA	DECORTICATION ↓ DECEREBRATION	UNREACTIVE, ENLARGING	OCULOCEPHALIC AND OCULOVESTIBULAR FAILING	HYPERACTIVE	CHEYNE-STOKES ↓ HYPER-VENTILATION	STABLE	SALVAGE UNLIKELY
LOWER BRAINSTEM	PROFOUND COMA	DECEREBRATION ↓ FLACCID	UNREACTIVE, LARGE	ABSENT	ABSENT	FAILING	UNSTABLE ↓ FAILING	DEATH CERTAIN

12 to 18 hours to begin working, which is why they are not used for the emergency reduction of increased intracranial pressure. There is no evidence that one type of corticosteroid is better than another for reducing intracranial pressure, but *dexamethasone* is the one traditionally used. An appropriate dose of dexamethasone would be 10 to 12 mg IV stat, followed by 4 to 6 mg IM every six hours.

Note that lumbar puncture has no place in the management of this particular syndrome. It yields no useful information and, by accelerating the coning process, may worsen the situation.

Primary Subtentorial Lesions

In this syndrome a lesion within or adjacent to the brainstem damages the reticular activating system, producing *simultaneously* impaired consciousness, pinpoint pupils and brainstem signs such as decerebration and absent oculovestibular responses. The pinpoint pupils are produced by destruction in the pons of the descending pupillodilator fibers with the unaffected pupilloconstrictor fibers in the upper midbrain acting unopposed. Lesions that cause this easily recognizable syndrome include (1) brainstem infarct (basilar artery occlusion); (2) primary pontine hemorrhage (hypertension); (3) cerebellar infarction (with edema, compressing pons); and (4) cerebellar hemorrhage (compressing pons).

It is particularly important to recognize cerebellar hemorrhage because it is surgically treatable. Its presentation may differ little from the other subtentorial lesions, but sometimes there are features which suggest the diagnosis. The evolution may be slower, vertigo and ataxia may be prominent before consciousness is lost, and the eyes may be deviated to the side opposite the lesion.

In the subtentorial syndrome increased intracranial pressure is seldom a significant pathogenetic feature and measures to relieve it are thus not usually indicated. CT scan or angiography should be done immediately, primarily to identify the surgically treatable cerebellar hemorrhage. CT scan is far superior to angiography for this purpose, but if CT scan is not available, then angiography may give enough indirect evidence of a mass lesion in the cerebellum to prompt posterior fossa exploration. The prognosis for a patient comatose from a primary sub-