英文影印版

CRUZ

神经系统急症

Neurologic and Neurosurgical Emergencies

科学出版社
Harcourt Asia
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Julio Cruz, MD, PhD

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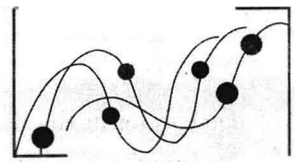

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This book is dedicated to my father
Julio Adamor Cruz *(in memoriam),*
for his daily demonstrations on the value of hard work and humanitarian help to human beings;

and to my Neurosurgical Instructor
Vittorio Rolando Boccaletti, M.D. *(in memoriam),*
for his superb teaching of Neurosurgery and Humanitarian Medicine
at Hospital São Paulo, Escola Paulista de Medicina,
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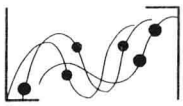

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Preface

The International Society for Neuroemergencies (ISONEUREM) was founded with the aim of converging nonsurgical (neurologic) and neurosurgical emergencies into a single body of scientific information, heavily based on clinical experience (without extrapolation from animal experimentation). The first ISONEUREM symposium (NEUROEMERGENCY '96), held in July 1996 in Freeport, Bahamas, was a very friendly, unbiased, and productive event, where all neuroemergency professionals could exchange clinically relevant information from the initial event through the emergency room and all phases of hospitalization, up to neurorehabilitation.

This book represents an original effort to concentrate in a single volume the roots of ISONEUREM, namely, to address unbiased, updated information regarding adult and pediatric neuroemergency issues in a most comprehensive fashion.

Up until now, the term *neuroemergency* has not yet been defined in medical dictionaries. According to the ISONEUREM standards, however, neuroemergency issues are expected to be covered in a combined and multidisciplinary fashion, rather than in isolated islands of pertinent medical information.

At the second ISONEUREM symposium (NEUROEMERGENCY '99), to be held in Italy, the above-described philosophy will be further enhanced, and all future ISONEUREM events will highlight the merit of addressing unbiased, strongly scientific and clinically relevant information on the diagnosis, pathophysiology, and management of acute insults to the nervous system.

It is therefore believed that this book will provide the reader with truly useful information on this rather complex topic, beyond previously published books or journal papers addressing questionable information regarding neurologic and neurosurgical emergencies, and neuro intensive care medicine.

JULIO CRUZ, M.D., PH.D.

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Graduate medical teaching is also strongly appreciated from my professors at Faculdade de Medicina do ABC, in Santo André, São Paulo, Brazil.

Following medical graduation, I strongly acknowledge the support of my neurosurgical professors at Escola Paulista de Medicina, Federal University of São Paulo, São Paulo, Brazil, and the University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania, USA.

As a faculty member, I heavily thank the physicians, nurses, scientists, and technicians from the University of Texas Medical School at Houston, Texas, USA, the University of Pennsylvania Medical Center, Philadelphia, Pennsylvania, USA, and the Allegheny University of the Health Sciences, Philadelphia, Pennsylvania, USA.

My 1980–81 experience as a Post-Graduate Fellowship Award recipient, sponsored by The Rotary Foundation of Rotary International, was rather gratifying and unforgettable.

JULIO CRUZ, M.D., PH.D.

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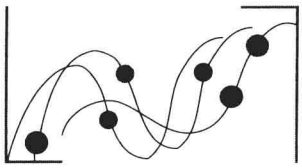

1

Initial Evaluation and Management of Neuroemergencies

Patti L. Peterson, M.D. • Brian J. O'Neil, M.D. •
Anthony L. Alcantara, M.D. • Daniel B. Michael, M.D., Ph.D.

Neurologic and neurosurgical emergencies frequently occur, particularly in busy, urban emergency departments. Severe permanent neurologic sequelae and death can result from these entities; therefore, they constitute true emergencies. The ability to rapidly assess the acute neurologically impaired patient, establish an accurate diagnosis, and effectively manage the underlying condition requires an experienced clinician.

This chapter summarizes the emergency department evaluation of the neurologically impaired patient, reviews pertinent features of the neurologic examination, and briefly summarizes the initial evaluation and management of common neurologic disorders. The majority of these neurologic disorders are covered at length in later chapters; therefore, only the emergency department management will be emphasized hereon.

During the past decade, considerable research efforts have been dedicated to a better understanding of the pathophysiology of neural injury.[1–5] These efforts have resulted in new theories and therapies pertaining to neuronal injury.[6–10] Injury to the nervous system is a progressive disorder with two distinct components termed "primary" and "secondary" injury. Primary injury is defined as the initial biomechanical damage that occurs immediately upon injury and consists of structural damage to neurons, their supporting tissues, and vasculature. Secondary injury is an evolving process that results in additional, progressive cellular damage and dysfunction and results both from degenerative biochemical cell processes, which are initiated by the primary injury, and from additional systemic insults such as hypoxia and hypotension.[11–13]

The recognition that secondary injury was an evolving process, which significantly contributed to morbidity and mortality, provided the impetus to delineate its exact biochemical nature. New pharmacologic treatments have been developed to ameliorate secondary injury.[10] Since these treatments are more effective when administered as soon as possible after injury, the necessity for rapid and accurate diagnosis of neurologic disorders has become imperative.

EMERGENCY DEPARTMENT EVALUATION AND MANAGEMENT

Prehospital Management

It is the responsibility of the Emergency Medical Services (EMS) to evaluate the airway, breathing, and circulation of the patient; immobilize and transport the patient to the nearest appropriate facility; and establish intravenous (IV) access and administer supplemental oxygen enroute to the hospital.

In the case of the neurologically compromised individual (particularly that with an altered mental status), the patient may be unable to communicate pertinent history and may be combative or agitated and therefore difficult to manage. In the comatose or traumatized subject, it is necessary to transport the patient with appropriate immobilization, cervical collar (c-collar), and long board until adequate assessment can be performed in the emergency department. If the patient has signs and symptoms of

opiate overdose or hypoglycemia and the appropriate equipment is available (e.g., Dextrostix, glucometer, naloxone [Narcan], glucose, and restraints), therapy may be initiated prior to hospital arrival.

Initial Emergency Department Evaluation and Management

Resuscitation

In all neurologic and neurosurgical emergencies, the initial evaluation and stabilization of the patient are of paramount importance to eventual neurologic outcome. It is well documented that hypotension, hypoxemia, seizures, hyperthermia, and other factors clearly increase secondary injury.[11–13]

The goals of resuscitation are to stabilize vital functions (airway, breathing, and circulation: the ABCs, vital signs and oxygenation) and perform a systematic evaluation of the patient in order to determine what emergent interventions are necessary. In all patients, the cervical spine (c-spine) should be assessed and stabilized and overt hemorrhage immediately controlled.

AIRWAY

The airway must be patent. A number of airway adjuncts exist and include the chin lift and establishment of nasopharyngeal or oropharyngeal airways; all improve airway patency. It must be stressed that these are only temporizing techniques and if oxygenation does not improve, a definitive airway technique (i.e., intubation of the trachea) is necessary.

BREATHING

The patient's respiration must be of adequate rate and tidal volume in order to avoid hypoxia and hypercapnia. Supplemental oxygen should be administered to patients who are severely traumatized or hypoxic as indicated by pulse oximetry. Oxygenation should be actively facilitated either with a bag-valve mask (if the patient is spontaneously breathing, it is best to assist ventilation during inspiration) or by use of 100% oxygen via a nonrebreather mask. The Sellick maneuver (application of cricoid pressure) reduces insufflation of the stomach during active oxygenation or during rapid-sequence induction. Rapid-sequence induction refers to the technique of rapidly preparing a patient for intubation while reducing the risk of pulmonary aspiration.[14]

Patients who are either comatose (Glasgow Coma Scale[15] score < 9 [Table 1–1]), hypotensive and refractory to volume replacement, unable to maintain adequate oxygen saturation despite the use of 100% oxygen, or unable to protect their airway should be intubated. Prior to intubation, preoxygenation can be achieved either actively with a bag-valve mask or by letting the patient breathe 100% oxygen via a non-rebreather mask, as previously described.

TABLE 1–1 **GLASGOW COMA SCALE**

Eye Opening			
	Opens	Spontaneously	4
		To verbal commands	3
		To pain	2
	No Response		1
Best Motor Response			
	To verbal command	Obeys	6
	To pain stimulus	Localizes pain	5
		Flexion withdrawal	4
		Flexion abnormal	3
		Extension abnormal	2
	No response		1
Best Verbal Response			
	Oriented and converses		5
	Disoriented and converses		4
	Verbalizes		3
	Vocalizes		2
	No response		1
Glasgow Coma Score (GCS) =		**TOTAL**	**3–15**

All patients should be presumed to have a full stomach at the time of presentation to the emergency department. The neurohormonal response to trauma predisposes these patients to aspiration. This is especially true in the case of shock and head injury. The coexistence of multiple trauma and head injury alters the approach to airway management. Cerebral blood flow (CBF), intracranial pressure (ICP), volume status, and cardiovascular function must be considered simultaneously so that no single factor is compromised.[16] Laryngoscopy and intubation may result in bradycardia, tachycardia, hypertension, and increased ICP.[17]

Intubation Techniques

Numerous intubation techniques and pharmacologic regimens for tracheal intubation currently exist, and the subject of airway management in the acutely ill patient is a controversial subject. For the sake of brevity, the most common intubation techniques and pharmacologic therapies will be reviewed and recommendations made.

Orotracheal intubation with rapid-sequence induction is becoming the procedure of choice for traumatic airway management. Blind nasotracheal intubation has long been advocated as the procedure of choice in patients with suspected c-spine injury. This procedure requires spontaneous breathing and

that the intubator can clearly recognize the patient's inspiration and expiration. In order to perform this procedure optimally, the nasal mucosa must be premedicated with an anesthetic and a vasoconstrictor (i.e., 4% lidocaine and 1% Neo-Synephrine). The effect of these medications is not immediate, and even after appropriate premedication, epistaxis can still occur. This may increase the patient's chance of aspiration and further complicate orotracheal intubation attempts.[18] Nasotracheal intubation causes intense cardiovascular stimulation that may cause a deleterious increase in ICP.[19] If nasotracheal intubation is performed, the patient should be adequately sedated, receive IV lidocaine (which may ameliorate some of the sympathetic circulatory effects), the c-spine should be immobilized, Sellick's maneuver should be performed, and the patient should be adequately oxygenated.

Despite the high success rate associated with orotracheal and nasotracheal intubation, it may be necessary to establish a surgical airway (cricothyroidotomy), if the nonsurgical procedures have failed or the patient has sustained massive facial and/or upper airway injuries.

Lidocaine (1.5–2 mg/kg/intravenous push [IVP] 2–3 minutes prior to intubation) attenuates much of the hemodynamic and cough reflex and rise in ICP during intubation.[20–22] The mechanism by which lidocaine accomplishes this is currently unclear. Etomidate (Amidate) is a nonbarbiturate, non-narcotic hypnotic agent that reliably facilitates induction in less than 1 minute, with a duration of action of 4–6 minutes.[23] Etomidate, when given in the above induction dose, has little effect on heart rate and cardiac output when compared with equipotent doses of thiopental.[24] Etomidate also has much less respiratory depressant effects than the barbiturates traditionally used in rapid-sequence induction, although transient apnea can occur if etomidate is given too rapidly.[25] Similar to the barbiturates, etomidate results in a reduction of ICP in a dose-dependent manner by decreasing CBF and cerebral metabolic rate.[26, 27] The aforementioned therapeutic attributes make etomidate an ideal induction agent in the setting of multiple trauma, head injury, and the potentially difficult airway.

Succinylcholine (1 mg/kg/IVP) provides rapid onset (<1 minute) and short duration of action (4–6 minutes) and makes it the paralytic of choice during rapid-sequence induction. The side effects of succinylcholine are fasciculations, hyperkalemia, and transient rise in intracranial, intragastric, and intraocular pressures. The fasciculations and increased intracranial, intragastric, and intraocular pressures all can be blocked by administration of a defasciculating dose of a nondepolarizing agent (e.g., 0.01 mg/kg/IVP of vecuronium [Norcuron]) 2–3 minutes prior to succinylcholine administration.[28] If a defasciculating agent is used, the dose of succinylcholine should be increased to 1.5–2 mg/kg/IVP in order to ensure rapid onset of action. A small number of individuals may develop vagally mediated bradycardia, histamine-induced hypotension, or malignant hyperthermia; unfortunately, there is no way to predict which patients will respond in this manner.

The recommended procedure for rapid-sequence induction is as follows:

1. Preoxygenation with 100% oxygen and use of the Sellick maneuver during active oxygenation.
2. Administration of lidocaine 1.5–2 mg/kg/IVP and vecuronium 0.01 mg/kg/IVP.
3. Slow administration of etomidate 0.3 mg/kg/IVP. The dosage should be reduced to 0.1–0.2 mg/kg/IVP if the patient becomes hypotensive.
4. Administration of succinylcholine 1.5–2 mg/kg/IVP.
5. Performance of the Sellick maneuver and orotracheal intubation of the trachea. The cuff should be inflated, breath sounds verified, tube placement confirmed, and the Sellick maneuver released.
6. Neuromuscular paralysis should be maintained, if indicated, with vecuronium 0.1 mg/kg/IVP.
7. End-tidal carbon dioxide detectors can be used to discriminate tracheal from esophageal intubation.

CIRCULATION

Circulation can be assessed according to the patient's vital signs and assessment of end-organ perfusion. All hypotensive patients (systolic blood pressure < 90 mm Hg) and those with signs of end-organ hypoperfusion (oliguria, poor capillary refill, confusion) should receive aggressive volume resuscitation with crystalloids, colloids, or packed red blood cells. Hypotension should never be attributed to head injury until an exhaustive search for other causes has been carried out. Hypotension secondary to intracranial injury occurs only as a terminal event resulting from failure of the medullary centers. Hypotension, however, can occur due to hemorrhage from scalp lacerations and also from high cervical cord injuries. It is also important to recall that vital signs alone are a poor indicator of shock states, as blood pressure drops only after loss of 30% of the blood volume. Intravenous access should be obtained and appropriate laboratory studies drawn.

Bedside glucometry and screening laboratory studies should be performed. Screening laboratory

tests should include complete blood count, electrolytes, blood glucose, blood urea nitrogen and creatinine, prothrombin time and partial thromboplastin time, blood type and screen (in the trauma patient), and arterial blood gases. If the patient has a nonfocal neurologic examination, urine and several tubes of blood should be saved in order to obtain serum and urine drug screens; liver enzymes; calcium, ammonia, carboxyhemoglobin and cyanide levels; serum osmolality; and anticonvulsant or other drug levels as indicated by the clinical presentation. The initial evaluation should also include an electrocardiogram (to rule out myocardial infarction or arrhythmia) and x-rays (to rule out lung, abdomen, and vascular abnormalities).

Rectal temperature, heart rate, blood pressure, respiratory rate, pulse oximetry, and cardiac rhythm should be closely monitored throughout the evaluation. In comatose patients, a urinary catheter and nasogastric tube should be carefully inserted if no contraindications exist (i.e., possible basilar skull fracture or urethral injury) while the appropriate invasive monitoring (e.g., central venous line, arterial line, ICP monitor) is placed.

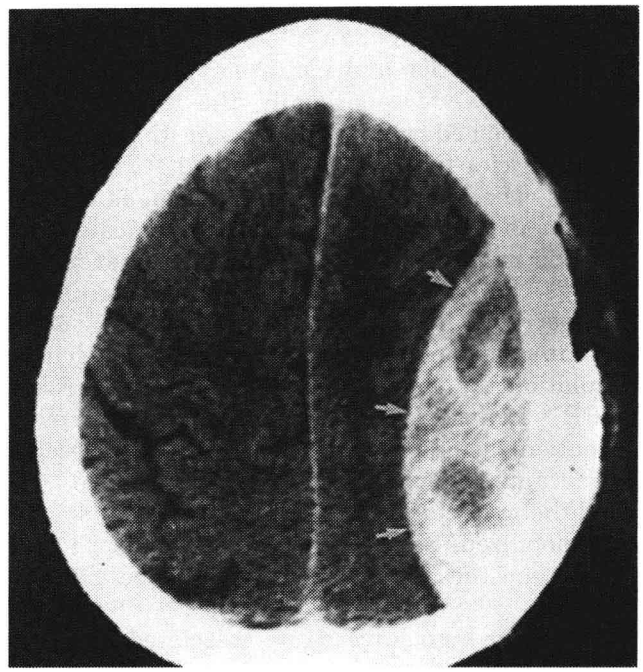

FIGURE 1-1 • A depressed skull fracture in the left parietal region is associated with a large epidural hematoma.

Initial Clinical Examination

The purpose of the initial clinical evaluation is to assess the patient's injuries/clinical disorder and prioritize further management. After the patient has been initially stabilized, a clinical evaluation should be performed in order to rapidly identify neurologic signs that indicate the need for immediate intervention (e.g., drainage of an epidural hematoma [Fig. 1–1]). The patient's level of consciousness should be assessed and examination of the pupils and oculomotor function performed.

A postresuscitation Glasgow Coma Scale (GCS) score and serial GCS evaluations should be performed in order to identify early signs of neurologic deterioration. The advantages of the GCS are its simplicity and high inter-rater reliability. The disadvantages are that the score is unreliable in patients who have sustained significant facial trauma (those whose eyes are swollen shut); are pharmacologically paralyzed, intubated, or dysphasic; in patients who are hypotensive; or in those who have suffered a spinal cord injury.

Initial Emergency Management

If bedside glucometry reveals a low glucose level, glucose should be administered (25–50 g/IVP). In suspected nutritionally deficient patients, e.g., alcoholics or chemotherapy patients, 100 mg of thiamine (1–2 mg/kg/IVP) should be administered prior to the administration of glucose. Thiamine is given to promote carbohydrate metabolism and to prevent the acute onset of Wernicke's disease (see later section). The newer formulations of thiamine do not cause anaphylactoid reactions when intravenously administered.

Naloxone (Narcan) should be administered (2 mg/IVP in adults) and repeated when indicated if narcotic intoxication is a consideration. If precipitation of opiate withdrawal is a concern, smaller doses (0.01 mg/kg/IVP) should be given and repeated if necessary. Naloxone is an effective antagonist of opiates and synthetic narcotics (e.g., propoxyphene), although with propoxyphene, larger doses may be needed to reverse the endorphin effects. Patients who are emerging from opiate overdose can become confused or combative and should be appropriately restrained prior to naloxone administration. If poisoning is a possibility, it may be necessary to lavage the stomach; emesis should be induced only in the alert adult patient (ipecac 30 mL orally). Evacuation of the stomach has been shown to be effective only after acute ingestion (<2 hours) unless drugs that decrease gastric motility (i.e., anticholinergics) have been ingested.

Elevations in systemic arterial pressure should not be treated, particularly in those patients who have sustained a cerebral infarction, intracerebral hemorrhage, or post-traumatic hemorrhage, unless (1) the systolic blood pressure exceeds 220 mm Hg or the diastolic blood pressure exceeds 120 mm Hg on three repeated measurements at 15-minute intervals; or (2) cardiac failure or aortic dissection is