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President's Notebook

This will be my last communication under this heading, as the new CAEP Executive takes office October 1, 1984. As such, by the time this reaches you, Dr. Terry Sosnowski, of Edmonton will be President of CAEP. I wish him the best of luck during the next year as our President, and offer whatever support and advice I can give.

A few items of interest have come up since the last issue of the Review. Many of you will have recently returned from Montreal where we held the first Emergency Medicine papers session in conjunction with the Royal College of Physicians and Surgeons of Canada. I think we can be encouraged with the number and quality of Emergency Medicine papers presented. In addition, I was very encouraged with the turnout to the session – not only from CAEP members, but also from non-EP's attending the RCPS meetings. They came specifically to hear our papers, and I think that bodes well for the future of academic Emergency Medicine in Canada.

As a result of the motion passed at our Annual Meeting in Vancouver, I wrote to various ministries and governments regarding seat belts in school buses. It took a while but I have now heard from most provinces, and have an enormous file. Most of the responses consisted of rationalizations for the current non-requirement of belts, but there are a few glimmers of hope. We will follow this issue with interest, and anyone wishing to pursue it should contact my office for the file.

A questionnaire was sent to all affiliate members, asking for input into current CAEP activities and future plans, particularly as they affect affiliate members. Most respondants favour CAEP moving to a similar membership profile to that of the Canadian Anaesthesia Society, who have almost as many affiliate GP-Anaesthetists as active FRCP-Anaesthetists. While our situation is somewhat more complicated because of a third category of CCFP – E.P.'s, it does seem the logical route to follow. CAEP should become the voice of Emergency Medicine in Canada, whoever is practicing it, and whatever their qualifications. The two Colleges have specific committees to pursue the interests of certificants in Emergency Medicine. To this end, we hope, over the next 12 months to make a direct appeal for input and membership from GP – Emergency Physicians across Canada.

Finally, we hope this year to improve both the quantity and quality of membership mailings. In particular, over the next few months, you can expect copies of the minutes from the April business meeting, copies of the newly revised constitution and by-laws of CAEP, and an Annual President's Report. By the same token, we would like to hear from our members more often. Both through letters to the Editor and through contacting individual executive members and committee chairmen, we specifically invite your input. If there is a particular issue that needs further policy development, you should feel free to get involved with a working group, or to start a new one.

In closing, I am thankful for the opportunity to have been your President for 1983–84. This has been a challenging year but, I feel, a positive one for the Association.

Peter L. Lane

Resident's Corner

Research – a state of mind

"Research has been called good business, a necessity, a gamble, a game. It is none of these – it is a state of mind."
MARTIN H. FISCHER (1879–1962)[1]

The establishment of emergency medicine in Canada has finally arrived with specialty recognition from both the Royal College of Physicians and Surgeons and the College of Family Practice. Yet, in order for emergency medicine to survive it must gain a sound research base and be accepted as a discipline.[2] It is generally recognized that the lack of original research in emergency medicine is a significant problem affecting the credibility of the profession. As residents, we are naturally concerned with acquiring the necessary clinical skills to successfully treat patients with multiple life threatening illnesses and injuries. However, this alone is not sufficient to ensure the future of emergency medicine as a specialty. The number of residency trained emergency physicians who devote themselves to research, as part of their academic responsibilities, is very small.[3] For this to change we should prepare ourselves for the future and seek further training in research methodology. As Krome[3] has stated: "The founders of emergency medicine owed to their progeny the formation of the specialty and its recognition; residents now owe their progeny solidification and definition of the academic discipline." This will come, with time, when emergency physicians become respected as teachers and researchers within academic teaching centers.

Training programs must recognize the need to prepare residents for practice both within the community and within the rigorously competitive "publish or perish" environment of large academic teaching centers. Unfortunately, the lack of research-competent and research trained emergency physicians[4] is striking, thus making it more difficult to obtain a reasonable exposure to high quality research as a resident. The rewards of resident research are tangible (Refer to Figure 1) and although they were espoused by Livingstone[5] last year in the CAEP Review, the large majority of emergency residents are not involved in research projects. Why not?

Often research is a requirement of residency programs apparently to expose residents to research methodology and encourage residents to participate in future research projects. It is my belief that it is virtually impossible to successfully impose research requirements on residents in the hope that they will learn research techniques, and hopefully produce meaningful data. In contrast one becomes "lost in the gloom of uninspired research".[6] How then can we foster the promotion of resident research? To borrow a phrase which has been used before, we must seek to develop the "climate for research". As stated by Seegal[7] "It is no forced extrapolation to state that every physician is perforce also a potential clinical investigator when he soundly fulfills his responsibilities for each patient". We should use a more analytical approach when treating patients. After all, is it not true that "the least questioned assumptions are often the most questionable".[8] When we ask whether a specific treatment protocol is best for a patient, as compared to other available therapies, is this not virtually a statement of a potential research hypothesis? Thus, fostering an inquisitive nature in patient management helps one become inspired to regard research in a new light. Research can provide answers to some of those often asked questions which confront us daily in the emergency department.

Formulating a hypothesis is only the first step in undertaking a research project. Time, that all too precious com-

modity, must be allocated to designing the study, collecting and analyzing the data, and finally writing and submitting a paper for publication. This can be a significant barrier to research in the demanding schedule of most emergency residents. Yet this, to a large part, can be overcome by fostering a "climate for research". On a more practical level this includes the availability of preceptors to help with the design of research projects, the use of computers and statisticians to facilitate data analysis, and the use of word processors in preparing publications.

A number of potential research designs are amenable to studies related to emergency medicine.[9,10] Cross sectional studies examine the relationship between diseases and other characteristics or variables as they exist in a defined population at one point in time.[9,10] These accounted for 27% of 48 clinical research studies published in Annals of Emergency Medicine in 1981.[11] Case control studies assess the relationship of an attribute to an existing disease by comparing the diseased to a nondiseased control group with regard to how frequently the attribute is present.[10] Such a study tends to be pursued retrospectively and accounted for 18% of the aforementioned research studies.[11] Cohort studies involve a study population free of disease in which the attributes of interest are measured initially. These patients are followed for a period of time and the relationship of the attribute to the disease is assessed by comparing the incidence of disease according to subgroups based on the presence or absence of the attribute in question.[10] Clinical trials are cohort studies in which treatment is initiated specifically for the evaluation of therapy.[9] Cohort studies accounted for 18%, and clinical trials 32%, of the research studies mentioned above.[11] The number of randomized clinical trials published in Annals of Emergency Medicine has increased significantly in the past ten years.[11]

Selecting which research design is best for a particular project is based both on the nature of the project and the time available. Cross sectional and case control studies may be more amenable to organizing research into monthly time blocks which fit into a residents schedule. Longitudinal randomized clinical trials are also amenable to resident research, but must be started early in training to allow sufficient time for the study period, data analysis and publication.

Funding has also been identified as a potential barrier to research.[4] However, it is of interest that of the research papers published in Annals of Emergency Medicine in 1981, 83.8% were not funded. This emphasizes the need for further funding of research in emergency medicine but also suggests that research can still be done with minimal or no outside funding. Funds for research are often hard to obtain with the current economic climate. Yet the CAEP research fund, Canadian Heart Foundation, Medical Research Council, The Laerdal Foundation for Acute Care Medicine, hospital research funds and pharmaceutical companies are all possible sources for funding. Emergency Physicians however, must first acquire the skills involved in writing a grant proposal[4] to avail themselves of these resources.

Publication of original research represents the final phase of a research project. This may take the form of a short abstract presented at a National meeting, which is then usually followed by submission of a full paper to a recognized journal. As Canadian emergency physicians, not only should we be involved in the promotion of research, we should help promote recognition of CAEP through publication in the CAEP Review. Further publications will help emphasize the contribution of emergency physicians to clinical research, foster respect from other specialities, and help improve funding for future research projects.

It is hoped that those residents currently involved in research find it a stimulating and rewarding experience. For those who are not, remember that the "research of a physician emanates from the problems he sees at the bedside".[13] Through fostering both an intellectual and practical "climate for research" the potential for research training exists for all residents. Let us not however, "lose sight of the pursuit of excellence in the scramble for funds, recognition and prestige."[14]

FIGURE 1

Benefits of Research

1 Allows development of insight into statistical analysis, experimental methodology and design.
2 Potential exists to make contribution to medical knowledge.
3 Allows personal creativity.
4 May become a criteria by which job applicants will be assessed.

Adapted from Livingstone[5]

References

1 Fischer MH. In: Strauss M.B. ed. Familiar Medical Quotations. Boston: Little, Brown and Company. 505, 1968.
2 Gerace R. "Presidents Notebook" *CAEP Review*. 4(2): 42, 1983.
3 Krome RL, Podgorny G. "Emergency Medicine: New Missing Blocks" *Ann Emerg Med*. 9(4): 229–230, 1980.
4 Rowat BMT. "Research So What" *CAEP Review* 5(2): 59, 1984.
5 Livingstone D. "Resident's Corner – Why do Research" *CAEP Review*. 4(2): 35 1983.
6 Wordsworth W. In: Strauss M.B. ed. Familiar Medical Quotations. Boston: Little, Brown and Company. 499, 1968.
7 Seegal D. In: Strauss M.B. ed. Familiar Medical Quotations. Boston: Little, Brown and Company. 251, 1968.
8 Broca P. In: Strauss M.B. ed. Familiar Medical Quotations. Boston: Little, Brown and Company. 323, 1968.
9 Fletcher R, Fletcher SW. "Clinical research in general medical journals" *N Engl J Med*. 301: 180–183, 1979.
10 Friedman GD. "Primer of Epidemiology" McGraw-Hill Book Company, Toronto. 48–57, 1980.
11 Gold I, Podolsky S, Kuhn M, Baraff, LJ. "A review of research methodology in an emergency medicine journal" *Ann Emerg Med*. 12(12): 769–773; 1983.
12 Podolsky S, Gold I, Kuhn M, Baraff LJ. "Research topics, author's specialty, and funding sources of articles published in an emergency medicine journal" *Ann Emerg Med*. 429–431, 1984.
13 Platt R. In: Strauss M.B. ed. Familiar Medical Quotations. Boston: Little, Brown and Company. 508, 1968.
14 Mason HL. In: Strauss M.B. ed. Familiar Medical Quotations. Boston: Little, Brown and Company. 508, 1968.

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Editorial

A Response to "Pre-Hospital Care in Quebec"

The historical background at the beginning of this paper should be a familiar one to most of us, regardless of where we live in Canada. Those who work in emergency care understand how the demands of the public affect access to facilities that are supposedly dedicated to emergencies. In many Canadian cities emergency department closures due to overcrowding have become an almost daily occurrence. In this respect Montreal is not very different from the rest of the country. The Urgence Santé is unique in Canada, but not when other countries are also considered.

Last year I attended an International Conference on E.M.S. in New York City at which delegates from ten foreign countries and ten large metropolitan centres in the U.S. discussed their systems. It soon became apparent that there were two quite different schools of thought about E.M.S. The French, Russians, Germans, British and the city of Montreal each used physicians rather than paramedics to respond to acute medical emergencies outside of hospital. This contrasted sharply with the American cities, Australia, and the rest of Canada (as represented by Vancouver).

The Americans were engrossed in the technological side of their operations (as demonstrated by their preoccupation with telemetry). They prided themselves on the consistency of their systems, the quality and depth of the training they provided, and on the mechanisms they had developed for ensuring medical control. Elaborate proofs of every detail of their operations were clearly being given a high degree of priority and visibility.

In the U.S. success rates for cardiac arrest are widely accepted as an index of effectiveness in an urban E.M.S. The Europeans, however were not impressed by these statistics, and, although they acknowledged the American expertise in cardiac resuscitation, they seemed to consider cardiac arrest management a waste of time as far as an E.M.S. was concerned. They showed more interest in on-scene triage followed by rapid transport for definitive care in a hospital than in trying to "revive the dead" in the field.

In fact, the Europeans could not understand why the Americans were bothering with paramedics at all. As far as they were concerned, since physicians were more highly trained, it was obvious that any system using paramedics had to be inferior, if not downright dangerous. Instead they wanted to know why the Americans could not find enough doctors to man their ambulances. If they could, they reasoned, then they would not have to bother with all of the training and the quality control aspects of their E.M.S. systems.

To the Europeans, these assumptions were so basic that there was no need to examine the subject in any more depth than that. The Americans on the other hand, defended the use of paramedics as preferable to physicians, at least in this capacity. They argued that having an M.D. after one's name did not necessarily qualify one for this kind of work. Also, they felt that paramedics could be more easily supervised and therefore performed more consistently than doctors (who generally like to have more independence).

After several hours of this kind of discussion in which neither side prevailed, it became clear that the differences in the two philosophies were culturally based, and therefore not open to rational argument. This might explain why Montreal, which has stronger cultural ties across the Atlantic than across the Ottawa River, chose the method of E.M.S. that they did.

In the development of any E.M.S. one must decide whether the system will be trying merely to provide quality emergency care, or whether it will be trying to solve the more global problem of distribution of health care resources. Most urban E.M.S. systems do only the former, but the Urgence Santé seems to be trying to do both. Normally, each of these is in itself a complex task, so there is some cause for concern that Urgence Santé might be biting off more than it can chew.

Perhaps in the case of the Urgence Santé it is too early for any verdicts. In any case there are clearly some major advances over what existed in the city of Montreal in the past. In the meantime, it must seem a liability to the city not to be able to take advantage of what has been learned about E.M.S. in neighboring communities.

However, since the differences between the two approaches are trans-cultural in nature, they are not likely to ever be resolved by logical arguments or by elaborate statistical proofs. But this is hardly important. In the end, the only thing that really matters is the ultimate effect on patient care. The onus should be on *both* schools of thought to first state clearly what their intended objectives are, and then to demonstrate how they propose to measure the effects they have produced. In this sense the European systems all have some catching up to do.

It is true that there is more than one way to skin a cat, but whichever method one chooses there must be a pelt at the end to show for the effort.

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A Canadian guide to the management of head injuries in the polytraumatized patient

MICHAEL L. SCHWARTZ MD

Introduction

Head injuries are common.[1] Approximately 70 percent of patients admitted to the Sunnybrook Medical Centre Regional Trauma Unit with multiple injuries have as a component of their injuries, a significant head injury.[2] Physicians who treat patients with multiple injuries should be knowledgeable about the pathophysiology of craniocerebral trauma and be prepared to deal with injuries to the nervous system. The Advanced Trauma Life Support (ATLS) Course[3] devised by the Committee on Trauma of the American College of Surgeons is recommended, as the course teaches the provider of care to patients with multiple injuries to view each injured system in its correct context and to treat the patient according to what are generally recognized priorities. Nevertheless, some of the advice offered in the manual to non-neurosurgical practitioners regarding brain injuries is unsuitable for physicians in Canada, where neurosurgeons are less numerous than in the United States, where longer distances from small communities to neurosurgical centres are the rule and bad weather often slows or prevents transfer of patients from one place to another. Furthermore, in Canada, CT scanners are virtually limited to major hospitals in large communities, with the result that decisions regarding transfer or treatment in the primary hospital are made on clinical grounds alone. Considering the specific requirements of Canadian practice, these guidelines for the management of patients with head injuries are offered. They should be considered in the general context of multiple injuries as described in the Advanced Trauma Life Support Course.

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Introduction

Les traumatismes crâniens sont courants.[1] Environ 70 pour cent des sujets admis au Centre de Trauma Régional du Sunnybrook Medical Centre avec des blessures multiples souffrent d'un traumatisme crânien.[2] Les médecins qui reçoivent ces polytraumatisés doivent bien connaître la physiopathologie du traumatisme crânien et cérébral et être en mesure de traiter les blessures au niveau du système nerveux. Il est conseillé de suivre le cours de réanimation avancée des polytraumatisés (Advanced Trauma Life Support – ATLS) mis sur pied par le Committee on Trauma du American College of Surgeons. Ce cours enseigne aux personnes appelées à soigner des polytraumatisés à considérer chaque système atteint dans son contexte et à traiter le malade en accord avec les priorités généralement reconnues. Cependant, certains conseils offerts aux médecins non spécialisés en neurochirurgie concernant les traumatismes cérébraux sont inadéquats pour les médecins canadiens. En effet, les neurochirurgiens sont moins nombreux au Canada qu'aux États-Unis, les distances entre les petites communautés et les centres de neurochirurgie sont plus grandes, et le mauvais temps ralentit souvent ou empêche le transfert d'un malade d'un endroit à un autre. De plus, au Canada, les tomodensitomètres sont presque tous concentrés dans les hôpitaux importants des grandes villes. Par conséquent, dans un hôpital périphérique, les décisions concernant le transfert ou le traitement d'un malade sont basées uniquement sur les observations cliniques. À partir de ces particularités de la pratique médicale au Canada, voici la ligne de conduite suggérée pour le traitement des traumatismes crâniens. Celle-ci doit être considérée dans le contexte général des polytraumatismes, tel que décrit dans le cours de réanimation avancée des polytraumatisés (ATLS).

Anatomy and physiology

SKULL

The shape of the skull and the varying thickness of the bones composing it predispose to characteristic injuries. In younger patients blunt impact may separate the interdigitations of suture lines. In patients of all ages blunt trauma to the face and frontal regions may produce fractures through the anterior cranial fossa so that there may be communication between the paranasal air sinuses (ethmoid, sphenoid and frontal) and the subarachnoid space. Such an occurrence may be detected by an air fluid level in a sinus on a brow-up lateral skull x-ray, or by pneumocephalus. Linear fractures in the relatively thin squamous temporal bone may be caused by direct trauma or by outbending of the bone from a blunt impact to the occiput. The sphenoid wings which create a buttress separating the anterior from the middle cranial fossa tend to focus shear

stresses within the substance of the brain when there is violent angular acceleration of the head.[4,5,6] As a result, a blunt impact to the occiput may result in disruption of the frontal or temporal lobe opposite to the site of impact; a "contre-coup injury".

MENINGES

The dura mater or tough covering of the brain is adherent to the inner table of the skull. The major draining veins of the brain, i.e., the superior, sagittal, transverse, and sigmoid sinuses, are located between its two layers and may be injured by depressed fractures. The middle meningeal artery, which emerges from the foramen spinosum and traverses the temporal bone in a groove, may be torn when the bone is fractured, with blood released that strips the dura from the inner table of the skull. The arterial grooves are visible on a lateral skull x-ray. A fracture line on the lateral skull x-ray that crosses an arterial groove may signal an epidural hematoma.

Inside the dura are the leptomeninges; the arachnoid layer which contains the cerebrospinal fluid, and the pia on the surface of the brain. Draining veins bridge from the surface of the brain to the venous sinuses and may be torn when angular acceleration of the cranial cavity and its contents causes movement of the brain relative to the skull. Characteristically, blood from torn bridging veins is released into the potential space between the dura and the arachnoid with the production of a subdural hematoma.

BRAIN

The cerebrum is divided into two hemispheres with one side receiving sensation from and governing muscle movement in the contralateral side of the body. Different regions of the brain subserve different functions. In most patients, the "dominant" hemisphere that subserves language and other analytical functions, is the left cerebral hemisphere. Vegetative functions, such as the control of body temperature, blood pressure, heart rate and respiration, are controlled by central portions of the brain including the hypothalamus and brain stem. The "brain stem" comprises the mid-brain, pons and medulla. The reticular activating system, which subserves consciousness, is located in the midbrain. The cranial nerve nuclei and cranial nerves controlling eye movement are located in the mid-brain and pons. Their assessment may aid in the diagnosis of stupor and coma.

The cerebrum is located in the anterior and middle cranial fossae or supratentorial compartment. The cerebellum and brain stem are in the posterior fossa or infratentorial compartment. Herniation of the medial temporal lobe through the hiatus or hole in the tentorium may impinge upon the oculomotor (III) nerve or the midbrain and cause a characteristic constellation of signs.

CEREBROSPINAL FLUID (CSF)

Cerebrospinal fluid is produced by the choroid plexus within the ventricular system. The normal circulation of CSF is from the lateral ventricles, via the foramen of Monro to the 3rd ventricle, thence via the aqueduct of Sylvius to the 4th ventricle at the level of the pons. From there, the CSF emerges, principally by the foramen of Magendie and circulates in the subarachnoid space upward through the tentorial hiatus to be reabsorbed via the arachnoid villae into the superior sagittal sinus. The flow of cerebrospinal fluid may be interrupted by distortions of the brain, or impeded by blood released into the cerebrospinal fluid, when cerebral tissue is disrupted. The obstruction of cerebrospinal fluid circulation may further compound an already serious brain injury. Fractures resulting in communication of the subarachnoid space, which contains the cerebrospinal fluid, with paranasal air sinuses or the exterior may be diagnosed by the observation of CSF rhinorrhea (a leak via the nose) or otorrhea (a leak of CSF via the ear).

CRANIAL NERVES

Inferences regarding the site and severity of craniocerebral trauma may be made from the examination of the cranial nerves. In the management of acutely injured patients the examination of the cranial nerves should be abridged to include only those observations that can be made rapidly and accurately and which serve as a guide to therapy.

Optic (II) – Funduscopic examination may show subhyaloid (preretinal) hemorrhages that result from a rapid, severe rise of intracranial pressure transmitted to the optic nerve sheath. Papilledema takes hours to develop and is not seen early after an injury. Most patients with multiple injuries will not cooperate for a detailed examination of visual acuity, so that presence or absence of vision in an eye may be the only assessment possible. Severely reduced visual acuity in an eye reduces or abolishes the direct pupillary (constriction) response to light. The consensual response (constriction of the pupil when the light is shone in the contralateral eye) is preserved.

Oculomotor (III) – The 3rd nerve, the nucleus of which is in the midbrain, and which passes along the medial edge of the temporal lobe, subserves constriction of the pupil of the ipsilateral eye. Distortion of the nerve by displacement of the temporal lobe (for example, transtentorial herniation) produces a dilated pupil that does not react to light (fixed) on the same side (94 percent of the time) as the displaced temporal lobe.[7] In addition, there is ptosis or drooping of the eyelid and inability to adduct the eye. A unilateral 3rd nerve palsy may be an important lateralizing sign in planning an emergency operation for an intracranial clot. An ocular injury may also cause fixed pupillary dilatation (traumatic mydriasis). When there is orbital bruising, traumatic mydriasis may be suspected but should not allay concern regarding transtentorial herniation.

Trigeminal (V) – The trigeminal nerve is the sensory nerve of the face. An injury to the nerve or its nucleus may render part of the face anesthetic, with the result that there may be abolition of corneal sensation and

absence of the corneal reflex, that is, closing of the eye when the cornea is touched.

Abducens (VI) – The 6th nerve abducts the eye. A long slender nerve which is easily injured, it is not useful in localizing a brain injury.

Facial (VII) – The facial nerve may be injured in (basal) skull fractures of the temporal bone. Asymmetrical grimace to painful stimuli or inability to close an eye may indicate a facial nerve injury on the side of the palsy.

TENTORIUM

The tentorium separates the anterior and middle cranial fossae from the posterior fossa of the skull. The tentorial hiatus transmits the midbrain. The 3rd cranial (oculomotor) nerve passes near the edge of the tentorial hiatus in relation to the medial edge of the temporal lobe. It may be distorted by transtentorial herniation of the uncus, a hook-like medial gyrus of the temporal lobe. Pressure on the cerebral peduncle containing the corticospinal tracts characteristically produces a contralateral hemiparesis. As the entire brain stem may be displaced to impinge on the sharp contralateral edge of the tentorium with the production of a hemiparesis ipsilateral to the uncal herniation,[8] the hemiparesis is a less reliable guide to the site of a probable intracranial clot than is the dilated pupil.[7]

UNCONSCIOUSNESS

In blunt trauma, unconsciousness is usually the product of diffuse injury throughout the brain produced by shear strains that tend to separate one layer of cerebral tissue from another.[9] Relatively minor stresses producing only physiological abnormalities without structural damage cause short-lived concussion, whereas ruptured axons, cerebral petechiae and intracerebral hematomas produce permanent injury and possibly prolonged or permanent coma. Unconsciousness may also result from direct injury to the reticular activating system in the midbrain.

INTRACRANIAL PRESSURE

Diffuse injury to the brain, with damage to small blood vessels or to axons, may result in diffuse cerebral edema or hematomas within the substance of the brain or on the surface when blood vessels are torn by energy imparted to the brain by acceleration. Accommodation to an expanding intracranial mass is made by displacement of cerebrospinal fluid through the foramen magnum and, to a lesser extent, by compression of cerebral veins. If ventilation is inadequate and the partial pressure of carbon dioxide in the blood rises, there will be venous dilatation and a further rise in intracranial pressure. As the cerebral blood flow to injured regions of the brain varies directly with cerebral perfusion pressure (the difference between systemic arterial pressure and intracranial pressure), failure to adequately treat shock and maintain a good systemic arterial pressure may lead to worse cerebral damage or even death. For these reasons, adherence to the resuscitation priorities is an essential first step in the management of raised intracranial pressure.

Assessment of head injuries

HISTORY

When management of unconscious patients must be based on clinical grounds alone, one must distinguish, if possible, between mechanical damage caused by angular acceleration and expanding intracranial hematomas. Nothing can be done about the former, but successful treatment of the latter makes the difference between survival and death. The characteristic story of a patient unconscious because of direct mechanical injury is: immediate unconsciousness and/or focal neurological deficit followed either by no change or by improvement. This history may be obtained from bystanders who accompany the patient to hospital or from ambulance attendants and paramedics.

In contrast, failing level of consciousness and/or increasing focal neurological deficit are the characteristic indications of an expanding intracranial hematoma. Once the patient comes under continuous observation in the emergency department, repeated, simple, stereotyped observations are sufficient to decide whether the patient is improving, staying the same or getting worse. It is not always possible to make the distinction between mechanical damage and an expanding intracranial hematoma on clinical grounds alone, as the two may co-exist, or the intracranial pressure may be rising and causing worsening coma as the effects of the initial concussion recede.

VITAL SIGNS ASSESSMENT

The blood pressure, pulse rate and the pattern and frequency of respiration are in part governed by the state of the brain stem. Cervical spine injury, always a possibility in patients with head injuries, may produce hypotension because the sympathetic outflow via the cervical spinal cord is interrupted, or inadequate respiration because of paralysis of the intercostal muscles and/or the diaphragm. Hypotension should not be attributed to brain injury. For the purpose of resuscitation, all that really matters is the adequacy of ventilation and systemic arterial pressure. Intubation and mechanical ventilation and intravenous volume infusions must be used as required. Hyperthermia is rare but may result from an injury to the anterior hypothalamus. Hypothermia from exposure is common, especially in patients who also have spinal cord injuries which abolish shivering and render them poikilothermic.

MINI-NEUROLOGICAL EXAM[3]

The Glasgow Coma Scale, originally described in 1974[10] and then modified in 1978,[11] characterizes the patient's level of consciousness by observation of eye-opening, best motor response and best verbal response. Widely used in Canada and easily available from many sources, the description will not be repeated here. Pupillary size and reaction are assessed and reassessed. The strength of the limbs is assessed for lateralized weakness.

The initial mini-neurological exam carried out in the first few minutes of assessment and resuscitation, serves as a baseline reference against which repeated neurological examinations are compared. Deterioration or improvement will be observed and treatment modified accordingly.

SPECIAL ASSESSMENT

Cervical spine

As a blow to the head sufficient to render a person unconscious is also sufficient to produce a spinal cord injury, the two conditions not infrequently co-exist. Unconscious trauma patients must be treated as though they had unstable spinal injuries until radiographic proof of stability is obtained.

Flaccid areflexia, decreased or absent anal sphincter tone or diaphragmatic breathing may signal a cervical spinal cord injury. Response to painful stimuli in the upper portion of the body but not in the lower, or a sweat level with the lower portion of the body dry and the upper wet, may localize the level of a spinal cord injury in a semi-conscious or unconscious patient. Hypotension may be the sign of cervical spinal cord injury and requires treatment by volume expansion; but, a source of blood loss as the cause should be sought first. Spinal cord injury and internal blood loss, for example, hemothorax or ruptured speen, may co-exist.

Special brain stem responses

The function of cranial nerves III, IV and VI may be assessed by turning the head; oculocephalic response or "doll's eyes" and/or the oculovestibular response (ice-water irrigation of the ear canals). These tests contribute little to the emergency management of the unconscious patient and should be reserved for the neurosurgical unit, as the former may aggravate a spinal cord injury and the latter may introduce bacteria via a basal skull fracture.

Skull x-rays

With the exception of penetrating injuries where metallic foreign bodies may be identified, skull x-rays contribute relatively little to decision making. Skull x-rays must never be obtained at the expense of continuing efforts at resuscitation.

The presence of a linear skull fracture increases the probability of an intracranial hematoma, especially when it crosses vascular skull markings, for example, the middle meningeal artery as it runs in the squamous temporal bone. Fractures of the petrous temporal bone may not show on skull radiographs but can be diagnosed by examination of the external ear canal. Otorrhagia (blood running from the external ear canal) and hemotympanum (blood behind the tympanic membrane) are reliable signs of a basal skull fracture and may also signal the presence of an epidural hematoma.

Closed (intact scalp) depressed skull fractures do not constitute an emergency but may be significant if the outer table of the depressed fragment is depressed as far as the inner table of the adjacent skull. Compound (open) depressed skull fractures require early operative intervention with elevation or removal of the fragments. Elevation of the fragments should only be undertaken in an operating room setting by a physician prepared to effect hemostasis, as the fragments sometimes tamponade torn blood vessels. If a decision is made to transport a patient with a compound depressed skull fracture to a neurosurgical centre, any superficial bleeders should be secured and a voluminous sterile dressing applied.

Basal skull fractures that produce a communication between the subarachnoid space and paranasal air sinuses or the exterior may result in air/fluid levels within those sinuses on brow-up lateral radiographs, or in pneumocephalus, air inside the head. Dripping of fluid from the nose (CSF rhinorrhea) or from the ear (otorrhea) may signal the basal skull fracture and alert the physician to the danger of subsequent meningitis. As the spinal fluid of patients with significant head injuries is often mixed with blood, the characteristic "ring" or "target" sign may be observed. If a drop of spinal fluid is allowed to fall on filter paper or the patient's sheet, the liquid phase of the mixture migrates farther than the solid components that form a circular central "bull's-eye" to the target.

Fractures of orbital bones release blood that is constrained by periosteal attachments to a crisp periocular distribution; the "raccoon-eye" sign. Contusion of orbital contents may produce ecchymoses with more diffuse boundaries.

Frontal basal skull fractures may permit the inadvertent intracranial passage of a nasogastric tube.

Reassessment and management

As soon as a complete assessment of the patient has been made, and resuscitation is underway, the patient should be reassessed. A decline in the level of consciousness or an increase in neurological deficit may be the result of inadequate ventilation or systemic arterial pressure. These causes of neurological decline should be considered first. If the neurological condition is worsening despite adequate ventilation and perfusion pressure, then an expanding intracranial clot as the cause is likely.

CONCUSSION

Angular acceleration, if not too severe, may produce a short and apparently completely reversible loss of consciousness.[10] If mild enough, angular acceleration may not alter the patient's apparent level of consciousness but will temporarily interrupt the continuous recording of memory. Conscious patients should be questioned regarding the details of their accident to establish whether or not there is retrograde or post-traumatic amnesia. If the neurological examination is completely normal and if the patient's home is not too remote from the hospital, he may be released in the care of a responsible person with instructions to return if drowsiness or neurological deficit develops. Recent evidence is that despite an apparent return to a normal

mental status, there may be a measurable injury caused by concussion that accounts for the "post-concussive syndrome" of headache, irritability and impaired concentration.[12,13]

CONTUSIONS

A prolonged episode of unconsciousness or persistent drowsiness in the wake of a concussion may signal petechiae or contusions within the cerebral substance. A focal neurological deficit may be present. These patients should be observed in hospital until they are alert, usually a period of 48 hours or more. These patients may harbour surprisingly large intracranial hematomas that may only be detected in the first instance by CT scanning.

INTRACRANIAL HEMATOMAS

Hematomas requiring surgical evacuation may be classified as extracerebral or intracerebral.

Extracerebral

- Epidural hemorrhage – Epidural hematomas are less common than subdural hematomas. The commonest cause of epidural bleeding is interruption of the middle meningeal artery with release of blood between the dura and the inner table of the skull. Venous sinuses or diploic veins (venous channels between the inner and outer table of the skull) may also be the source of epidural bleeding. The so-called "lucid interval" between a period of unconsciousness from concussion and the onset of coma from raised intracranial pressure is the exception rather than the rule.[7] Acute epidural hematoma are indistinguishable on clinical grounds from acute subdural hematomas. An epidural hematoma will most likely be found in relation to a fracture diagnosed by an overlying scalp hematoma, a skull radiograph or by hemotympanum and/or otorrhagia. The pupil on the side of the hematoma may be dilated.

An intravenous infusion of 20 percent mannitol (one gram per kilogram body weight, or 350 cc in a 70 kg person) should be administered and the hematoma evacuated promptly. The results of early evacuation are usually excellent because there is no direct injury to the underlying brain.

- Acute subdural hematoma – In contrast to acute epidural hematomas, the outcome after evacuation of subdural hematomas is not as likely to be good.[14] Blood is released into the subdural space by torn cortical or bridging veins or from cortical lacerations. As for rapidly failing level of consciousness and increasing neurological deficit due to epidural hematoma, mannitol should be given and the hematoma evacuated promptly.[15]
- Subarachnoid hemorrhage – Blood may also be released into the cerebrospinal fluid by traumatic injuries with the production of headache, photophobia and nuchal rigidity.

Traumatic intracerebral hematomas

Severe angular acceleration and strong shearing forces may tear blood vessels within the substance of the brain. Focal neurological deficit and coma may result. Intracerebral hematomas are best diagnosed by CT scanning as they will be missed by exploratory burr holes or craniotomies performed in the expectation of finding a surface hematoma.

Patients who present with a foreign body protruding from the skull should be transferred for definitive neurosurgical treatment with the object in place, as it may be tamponading torn blood vessels. Skull x-rays are necessary to demonstrate the extent of penetration.

Missile injuries are far less common in Canada than in the United States and, in general, the calibre of bullets tends to be smaller. Skull x-rays are necessary to demonstrate the distribution of metallic fragments so that an effective surgical procedure may be planned.

CEREBRAL EDEMA, VASOSPASM AND/OR LOSS OF CEREBRAL AUTOREGULATION

Cerebral edema may develop as a consequence of diffuse shearing injury to the brain which disrupts axons and small blood vessels. Severe, diffuse cerebral edema is more likely to occur in younger patients with severe head injury.[16] The control of diffuse cerebral edema may be difficult and therapeutic manipulations, such as relative fluid restriction and mannitol, may be the opposite of those recommended for non-neurological injuries.

Vasospasm caused by direct injury to cerebral blood vessels or by the release of vaso-active substances into the subarachnoid cerebrospinal fluid may be sufficiently severe to cause cerebral infarction.

Cerebral autoregulation refers to the phenomenon by which a constant blood flow per volume of cerebral tissue is maintained despite fluctuation in systemic arterial blood pressure. With a brain injury, cerebral autoregulation is lost and the regional cerebral blood flow to the traumatized portion of the brain varies directly with cerebral perfusion pressure, that is, the difference between systemic arterial and intracranial pressure. Inadequate systemic arterial pressure or uncontrolled intracranial pressure, both of which reduce the cerebral perfusion pressure, will lead to further cerebral infarction. With the loss of vascular integrity from such an episode and the re-establishment of high cerebral perfusion pressure, cerebral edema and intracranial hypertension are made worse. The continuous maintenance of adequate cerebral perfusion pressure (>60 torr) is essential.

Fluid restriction

In patients with isolated head injuries, neurosurgeons have generally limited the intake of fluids. The syndrome of inappropriate antidiuretic hormone secretion (SIADH) with hyponatremia is very common and may contribute to impaired consciousness.[17] SIADH is prevented and treated by fluid restriction and at times hypertonic saline solutions. Large volumes of fluid tend to exacerbate cerebral edema.

It is the rule to treat severe systemic injuries with

large volumes of balanced salt solutions. The physician caring for a patient with multiple injuries must use judgement in the administration of intravenous fluids. Overhydration, which makes cerebral edema worse, should be avoided.

Steroids
Glucocorticoids in high or low doses are of no benefit in the treatment of patients with severe brain injuries and are not recommended.[18]

Diuretics
Mannitol as a 20 percent solution, given as a rapid intravenous infusion in an initial dose of 1 gm/kg (350 cc for a 70 kg person) creates an osmotic gradient between intact brain and the blood stream.[19,20] This results in the movement of brain water into the blood stream with reduction in brain bulk and relief of intracranial hypertension. When the amount of intact brain is relatively small, the drug is less effective. With an expanding intracranial clot, the duration of action of mannitol may be relatively short. Subsequent doses are progressively less effective. The drug is most useful in lowering intracranial pressure while preparations for evacuation of an intracranial hematoma are being completed. A urinary catheter should be in place when mannitol is given, as there is usually a brisk diuresis. The diuresis is sometimes of sufficient volume to nullify the effects of fluid administered for vascular support in systemic injuries. Judgement in its administration must therefore be used.

In general, the drug should be given if a decision is made to evacuate an intracranial hematoma prior to transfer to a neurosurgical centre or in consultation with a neurosurgeon who has agreed to accept the patient in transfer. The drug should not be given unless one has reason to believe that there is raised ICP.

Hypocapnia
Carbon dioxide is an extremely potent vasodilator. When mechanical ventilation is indicated for other reasons, intracranial pressure seems best controlled with the partial pressure of carbon dioxide adjusted to between 25 and 30 torr. With respect to the head injury alone, intubation and mechanical ventilation are indicated if the patient is unconscious and therefore unable to maintain an adequate airway or if arterial blood gases indicate hypercarbia.

Other signs and symptoms of head injury

CONVULSIONS
Grand mal seizures may occur shortly after significant blunt craniocerebral trauma. Epileptic activity increases brain metabolism and hence the need for oxygen and glucose. Furthermore, respiration is impaired by convulsions. Initial treatment consists of Diazepam, 10 mg I.V. infused at 2 mg/minute. Respirations, blood pressure and heart rate should be monitored. Repeat in 15 minutes if seizures recur. Maximum dose – 20 mg. Phenytoin (Dilantin) may be administered to a total dose of 1.2 g for a 70 kg person at a rate not to exceed 50 mg/minute. The electrocardiogram, in addition to vital signs, should be monitored. If these measures fail, phenobarbital may be infused at a rate not to exceed 100 mg/minute until the seizures stop or to a maximum of 30 mg/kg (1.4 g for a 70 kg person). Intubation and mechanical ventilation will be required if this dose of phenobarbital is given.[21]

RESTLESSNESS
Patients with multiple injuries, with or without significant brain injury are often restless. A diligent search for a cause of restlessness, such as hypoxia, hypotension, distended urinary bladder, undiscovered fractures, excessively tight plasters and bandages, etc. should be made. Morphine, which significantly reduces the level of consciousness, or chlorpromazine, which may cause hypotension and drowsiness, are not indicated.

SCALP WOUNDS
The scalp has a luxurious blood supply. Unless tissue loss makes apposition of the edges of scalp wounds difficult to approximate, they are usually easy to repair and heal well.

Blood loss
Although blood loss from scalp wounds can be particularly severe, hypotension should not be attributed to bleeding from the scalp until other causes are ruled out. As with any wound, large vessels should be clamped and ligated. The smaller ones may be cauterized. As the blood vessels of the scalp run between the skin and the galea (fibrous aponeurotic inner layer of the scalp), temporary tamponade of the vessels may be obtained by placing hemostats on the galea and everting the wound.

Inspection of the scalp wound
If the wound gapes, then the galea is torn. The wound should be carefully inspected and palpated using sterile technique. Bone fragments and foreign bodies may be identified in this way. CSF in the wound indicates a compound depressed skull fracture with laceration of the dura and arachnoid.

Repairing the scalp wound
The region of the wound should be shaved, prepared and draped in a sterile field. Hair and foreign bodies should be removed and the wound should be irrigated with a large volume of saline. Loose bone fragments may be removed, but an operating room setting is recommended as impacted fragments may sometimes tamponade torn blood vessels which will bleed as the pressure is released.

If a good cleansing of the wound has been possible, it is best closed in layers. The galea is reapproximated first and then the skin. If there has been gross contamination of the wound or if a definitive repair is not possible and a prolonged transfer time is anticipated, the wound may be closed with vertical mattress sutures.

A bulky dressing that applies mild pressure to the wound is preferred.

Additional supportive diagnostic measures

CERVICAL SPINE X-RAY

Unconscious patients may have cervical fractures; a blow to the head sufficient to cause unconsciousness, may also cause a fracture or fracture dislocation of the neck. A cross table lateral view showing all the cervical vertebrae and the rostral edge of T-1 is required. It may be necessary to pull the arms downwards so as to expose the lower vertebrae. If this cannot be done, a semi-rigid collar, for example, a Philadelphia collar, must be placed. A swimmer's view or tomograms of the cervical spine should be obtained as soon as the opportunity arises. Unconscious patients must be managed as though they had unstable cervical fractures until it is proven by radiography that they do not.

COMPUTED TOMOGRAPHY (CT SCAN)

A CT scan is recommended at the first opportunity permitted by ongoing resuscitation, especially when pharmacological paralysis and ventilation are required for the treatment of other injuries (lung contusion, flail chest, etc.), or when the patient will be anesthetized for a long surgical procedure such as open reduction and internal fixation of fractures. If a laparotomy is required for management of the patient in a centre that has no CT scanner, and there are clinical grounds to suspect an intracranial hematoma, exploratory burr holes should be placed in lieu of a CT scan.

OTHER TESTS

Electroencephalography and isotope scanning have no role in the acute management of patients with head injuries. Lumbar puncture, which may exaggerate pressure gradients across the tentorium and the foramen magnum, predisposing to herniations of the brain, is not only useless but potentially lethal as well.

Surgical management

In the early stages, as an intracranial clot expands, displacement of cerebrospinal fluid and, to a lesser extent, compression of cerebral veins, mitigate rising intracranial pressure and slow the decline of the level of consciousness. As these mechanisms of compensation are exhausted, the deepening of coma accelerates. If there is any decline in the patient's neurological status as the mini-neurological examination is repeated, then transfer may be inadvisable. The advice and consent of the neurosurgeon who is to receive the patient should be obtained. In deciding whether exploratory burr holes and possibly a craniotomy should be done in the primary hospital, the patient's other injuries, the neurological status of the patient, his rate of decline, the expected transport time and the skill and experience of the sending physician must all be considered.

The development of a unilateral fixed, dilated pupil during resuscitation of a patient who initially localized a painful stimulus but who begins to exhibit spastic flexion or extension, signals the need for an immediate evacuation of his intracranial hematoma. The dilation of a second pupil when one has been dilated is equally ominous. Mannitol should be given. The entire scalp should be shaved, prepared and draped in a sterile field, but the first burr hole should be placed on the side of the first dilated pupil. With the pressure once relieved, a craniotomy may be completed with less urgency or the burr hole may be enlarged to a craniectomy to permit better access to the cranial cavity. Craniotomy is the preferred method of evacuation of acute intracranial hematomas.

If a hematoma on the surface of the brain is not discovered but intracranial hypertension is identified, the prognosis is poor as the cause may be an intracerebral hematoma or diffuse brain swelling. The patient should then be transferred to a neurosurgical centre with a CT scanner for definitive diagnosis and treatment. The advice of the receiving neurosurgeon should always be obtained.

Patients with spastic extensor posturing and bilateral fixed dilated pupils from the outset may harbour intracranial hematomas but the likelihood of a good outcome is very small. Neurosurgical advice should be obtained regarding management.

Summary

- Ensure good ventilation and maintain good systemic arterial blood pressure.
- Establish a baseline neurological examination.
- Discover and treat associated injuries.
- Avoid overhydration.
- Re-examine for deterioration in neurological status.
- Obtain a neurosurgical consultation regarding transfer.
- Treat life-threatening neurosurgical emergencies that preclude transfer.

References

1 Nelson WR, McMurtry RY, Flafferty, DA, Fulton LA. The epidemiology of head injuries admitted to Ontario's Regional Trauma Unit. *CAEP Review* 5(1): 11–14, 1984.

2 Schwartz ML. Head injury in multiple trauma. *Can J Surg* 26(1): 23–26, 1983.

3 American College of Surgeons, Advanced Trauma Life Support Course. *Instructor Manual*. 1981.

4 Holbourn AHS. Mechanics of head injuries. *Lancet* ii 438–441, 1943.

5 Peerless SJ, Rewcastle NB. Shear injuries of the brain. *Can Med Assoc J* 98: 577–582, 1967.

6 Strich SJ. Shearing of nerve fibres as a cause of brain damage due to head injury. *Lancet* ii 443–448, 1961.

7 Teasdale G, Galbraith S. Acute traumatic intracranial hematomas. *Prog Neurol Surg* 10: 252–290, 1981.

8 Kernohan JW, Woltman HW. Incisura of the crus due to contralateral brain tumor. *Arch Neurol Psychiatry* 21: 274–287, 1929.

9 Ommaya AK, Gennarelli TA. A physiopathiologic basis for non-invasive diagnosis and prognosis in severe

head injury. In McLaurin RL (Ed.) *Head Injuries*. Grune and Stratton Inc. 49–75, 1976.

10 Teasdale G, Jennett B. Assessment of coma and impaired consciousness: A practical scale. *Lancet* ii, 81–84, 1974.

11 Teasdale, G, Knill-Jones R, Van der Sande J. Observer variability in assessing impaired consciousness and coma. *J Neurol Neurosurg Psychiatry* 41: 603–610, 1978.

12 Gronwall D, Wrightson P. Memory and information processing after closed head injury. *J Neurol Neurosurg Psychiatry* 44: 889–895, 1981.

13 Rimel RW, Giordani MA, Barth JT et al. Disability caused by minor head injury *Neurosurgery* 9: 221–228, 1981.

14 Gennarelli TA, Spielman GM, Langfitt TW, et al. Influence of the type of intracranial lesion on outcome from severe head injury: A multicenter study using a new classification system. *J Neurosurg* 56: 26–32, 1982.

15 Seelig JM, Becker DP, Miller JD, Greenberg RP, et al. Traumatic acute subdural hematoma: Major mortality reduction in comatose patients treated within four hours. *New Eng J Med* 304: 1511–1518, 1981.

16 Schwartz ML, Tator CH, Rowed DW, Reid SR, et al. The University of Toronto Head Injury Treatment Study: A prospective, randomized comparison of pentobarbital and mannitol. (In press) 1984.

17 Bartter FC, Schwartz WB. The syndrome of inappropriate secretion of antidiuretic hormone. *Am J Med* 42: 790–806, 1967.

18 Braakman R, Schouten HJA, Blaauw-Van Dishoeck M, Minderhoud JM. Megadose steroids in severe head injury. *J Neurosurg* 58: 326–330, 1983.

19 Shenkin HA, Goluboff B, Haft H. The use of mannitol for the reduction of intracranial pressure in intracranial surgery. *J Neurosurg* 19: 897–901, 1962.

20 Wise BL, Chater N. The value of hypertonic mannitol solution in decreasing brain mass and lowering cerebrospinal fluid pressure. *J Neurosur* 19: 1038–1043, 1962.

21 Hwang P. Emergency management of seizure disorders. In Earnest MP (Ed.) *Neurologic Emergencies*, New York, Churchill-Livingston, 1983 p. 103–125.

An unusual case of bilateral leg edema

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Abstract

The infrequent presentation of acute cord compression as lower limb edema is described. The importance of emergency physicians anticipating this problem in cancer patients is emphasized, as aggressive early therapy with steroids and radiotherapy can preserve cord function and reduce the catastrophic morbidity associated with this lesion.

KEY WORDS: acute cord compression, lower limb edema, metastatic cancer

Résumé

La compression médullaire aigüe se présentant sous la forme peu fréquente d'oedème des membres inférieurs est décrite. On insiste sur l'importance pour le médecin d'urgence de prévoir cet état chez le cancéreux afin d'instituer rapidement un traitement vigoureux à l'aide de stéroides et de radiothérapie pouvant préserver la fonction médullaire et réduire le taux de morbidité catastrophique associé à cette lésion.

MOTS-CLÉS—compression médullaire aigüe, oedème des membres inférieurs, cancer métastatique.

The differential diagnosis of bilateral lower limb edema includes many disease entities (Table 1). Rarely emphasized, however is the possibility that paralysis or paraplegia can present in this way. The major textbooks of internal medicine and physical examination do not even cite this as a possible cause of lower limb edema.[1,3,4] The following case is therefore presented to sensitize emergency doctors to this unusual mode of presentation.

TABLE 1: Differential diagnosis of bilateral lower limb edema[1,2]

1 Cardiovascular (CHF, constrictive pericarditis, portal vein obstruction, inferior vena cava obstruction, pelvic vein obstruction)
2 Loss of venous tone (convalescence, paralysis)
3 Hypoalbuminemia (nephrosis, anemia, cachexia, beri-beri)
4 Ascites
5 Compression (garter belts etc.)

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The patient, a 69 year old male, presented to the Emergency Department complaining of increasing leg swelling over the preceding three weeks, with three days of orthopnea and perhaps PND. He was a 50 pack-year smoker with a past medical history of oat cell carcinoma of the lung (diagnosed by lung biopsy 6 months previously) which was being treated with chemotherapy (methotrexate, lomustine, cyclophosphamide) every three weeks. There were bone-scan documented metastases at the level of his ninth thoracic vertebral body. He also suffered from angina, congestive heart failure and mild aortic stenosis, and had survived a myocardial infarction eleven years previously. His current medications included: Digoxin 0.25mg po daily; Lasix (furosemide; Hoechet) 80mg po each morning; and Aldactazide (spironolactone 25mg, hydrochlorothiazide 25mg; Searle) one tablet thrice daily.

On physical examination, the blood pressure was 100/50; the pulse 88/min. and irregular; the respiratory rate 16/min. The patient was afebrile. There were scattered rales in an otherwise clear chest; no JVD or HJR; and normal heart sounds without an S_3 or S_4. A 2/6 systolic, ejection-type murmur radiated to the carotids from the left sternal border. The abdomen was distended but soft and non-tender with a firm liver edge palpable two finger-breadths below the costal margin and normal bowel sounds. There were no stigmata of liver failure. The rectal sphincter tone was within normal limits. There was 2+ pitting edema of both legs to the mid-calf and tenderness over T_9–T_{12} in the back.

It was at this point that it was noted that the patient was hardly able to move his legs. Further questioning revealed that he had noted progressive weakness of his legs (worse in the left than the right) over the last three weeks, and that he had been seen for this in the clinic where a walker was given to him and little else done. In the last two days he had been totally unable to walk and had, radiating from his back down both legs, almost continuous shooting pains which were minimally relieved when he shifted his sitting position. He had been constipated for the last few days and was having increasing trouble voiding.

Neurological examination revealed an alert male, oriented to person, place, and time, with normal mentation and cranial nerve function. Sensation to light touch and pinprick was slightly diminished below the T_9–T_{10} level but position sense was intact. Motor bulk was normal but the power was, at best, 2/5 in his quadriceps, decreasing distally (the left side was slightly stronger than the right). The strength of his upper limbs was unaffected. His deep tendon reflexes were normal in his upper limbs, slightly diminished at his quadriceps, markedly diminished in his left ankle and gone in his right. Both toes were upgoing. The CBC and electrolytes were unremarkable. As the patient was re-examined by subsequent physicians over the next two hours, position sensation in his lower limbs began to fail and his rectal tone weakened.

Acute cord compression secondary to metastatic lung

cancer was the entertained diagnosis and the patient, after an L-S spine x-ray which showed an old compression fracture at T_9, went to myelography where a complete epidural block was seen at T_9. The patient was admitted for urgent radio-therapy which started that evening and continued over the next 10 days with the recovery of some strength in his legs, normalization of his rectal sphincter tone, and improvement in his ability to urinate. His general condition however continued to decline and he died soon after developing an aspiration pneumonia.

Discussion

This patient presented with a major complaint of increasing leg swelling over three weeks time. On examination it soon became apparent however, that the swelling was not secondary to liver failure or congestive heart failure (both possibilities in this patient) but to his inability to move his legs; an unusual way for acute cord compression to present.

Distant metastases are frequent with oat-cell carcinoma of the lung. In about one-quarter of patients, bone marrow involvement will be found when the patient is first seen. Symptomatic central nervous system disease is present in 10% of patients initially and will eventually develop in at least 30% of those who remain untreated.[5]

The skeleton, and more specifically the vertebral bodies of the thoracic spine, are the third most common sites for cancer to spread to in the body. Bronchogenic carcinoma is the second most common primary (after breast cancer and ahead of lymphoma).[6,7] The route of spread is most likely hematogenous via the paravertebral extradural venous plexes. Symptoms result from one of four pathological processes:[6,7,8]

1 most commonly, pressure from an enlarging extradural deposit. Often the tumor extends directly from the area of involved bone to impinge upon the spinal cord. Since the vertebral body is the most commonly involved area, this explains why most extradural metastases lie anterior to the cord. Extension however, can also be from retroperitoneal or mediastinal lymph nodes through the intravertebral foramina;
2 pressure from a pathological fracture with attendant dislocation of the vertebral body;
3 severe spinal cord angulation after vertebral body collapse, and rarely;
4 pressure from a metastasis within the dura.

The clinical presentation of developing spinal cord compression is actually quite predictable. Pain localized to the involved area is the most common finding, occurring in 96% of patients with epidural metastasis.[7,9] It usually predates other symptoms and signs. The pain is often worse at night and increased with spinal movement, coughing or straining. The area is tender to palpation. Radicular pain is found in 50% of patients with thoracic level involvement.[6] After a very variable period of time, pain is followed by muscle weakness and eventually paraplegia and paralysis. Numbness accompanies the decline in strength. Of note, the sensory level is often several segments below the actual level of the cord compromise. Bowel and bladder dysfunction are late problems. Where paraplegia develops rapidly (within 24 hours) the chance of recovering function, even with aggressive therapy, is slight.[6,10] This is especially common with metastases from renal or pulmonary primaries.

Diagnostic studies

Roentgenographic studies of the vertebral column will not demonstrate metastasis until 30–50% of the bone is destroyed.[6] At that point the findings include osteolytic or osteoblastic involvement of any part of the vertebra with or without a paraspinal mass. Vertebral body collapse, when it occurs, is typically flat as opposed to wedge shaped.[6]

Bone scan is the most sensitive method of detecting metastatic disease but, with improving resolution, CT scanning may have a more important role to play. Radionuclide scan still has a 3–5% false negative rate which jumps to 50% in patients with multiple myeloma.[7]

Myelography remains the single most useful test in terms of localizing and typifying the obstruction. The upper level of the block can be identified by introducing contrast at the $C_1 - C_2$ level or via the cisterna magna. This should not be omitted as multiple levels of involvement are the rule rather than the exception. Of course a normal mylogram does not exclude diffuse microscopic subarachnoid spread of the disease or other causes of weakness in cancer patients such as carcinomatous neuropathy, transverse myelitis or radiation myopathy. The neurologic-myopathic syndromes are actually quite rare, occurring in only about 1% of lung cancer patients. Polymyositis, cortical degeneration, subacute cerebellar degeneration, peripheral motor and/or sensory neuropathies, and the myasthenic Eaton-Lambert Syndrome should all be considered as potentially confusing complications of oat-cell carcinoma.[4]

Treatment

The key is that it be early and aggressive to prevent complete cord compression. High dose steroid therapy (Dexamethasone 10mg IV loading dose, followed by 4mg IV Q6h during treatment and then rapidly tapered) by itself, is not enough to halt the progression of the disease process; but, it does decrease the amount of edema resulting from radiotherapy. Oral antacids or cimetidine should also be started to decrease the risk of significant GI hemorrhage. Radiation therapy is the next step, especially for those patients who are too sick, or whose disease is too far advanced for surgery. It is almost invariably able to decrease the pain associated with the lesions. Surgery is reserved for those patients deteriorating during radiotherapy, or those with instability, severe pain, or blockage not resolving with therapy. Skeletal traction and/or external braces are used before surgery to maintain alignment in unstable situations.

Prognosis

The results of therapy depend on the type of tumor and the rapidity of symptom onset. Patients who have endured a longer duration of pain, a slower onset of deficit, incomplete paraplegia and intact sphincter tone at the start of therapy all tend to do better.[11] The mortality from surgical decompression varies from 7–24%.[11] Sixty per cent of patients who are ambulatory before the commencement of therapy remain so afterwards. Thirty-five per cent of those initially paraparetic (with only mild to moderate weakness) become ambulatory. Less than 25% of those who present with paraplegia ever walk again.[7]

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Bibliography

1 Degowin EL, Degowin RL. *Bedside Diagnostic Examination*, MacMillan Publishing Co., Inc., New York, 1976.
2 Friedman HH, editor. *Problem-Oriented Medical Diagnosis*, Little, Brown and Company, Boston, 1975.
3 Beeson PB, et al. *Cecil, Textbook of Medicine*, (15th Edition), W.B. Saunders Company, Philadelphia, 1979.
4 Wintrobe MW, et al. *Harrison's Principles of Internal Medicine*, McGraw-Hill Book Company, New York, 1980.
5 Greco FA, Oldham RK. "Current Concepts in Cancer – Small-Cell Lung Cancer", *New Eng J Med* 301(7): 355, 1979.
6 Boland PJ, et al. "Metastatic Disease of the Spine", in *Clinical Orthopaedics and Related Research* 169: 95, 1982.
7 Davis JM, et al. "Metastases to the CNS", in *Radiologic Clinics of North America* 20(3): 417, 1982.
8 Hughes JT. *Pathology of the Spinal Cord*, Year Book Medical Publishing Inc., Chicago, 1966.
9 Vinken PJ, Bruyn GW. *Handbook of Clinical Neurology*, (Vol. 19 and 20), North-Holland Publishing Co., Amsterdam, 1975.
10 Seydel HG, et al. *Cancer of the Lung*, J. Wiley and Sons, New York 1975.
11 Natier A, Bose K. "The Results of Decompression of Cord or Cauda Equinia Compression from Metastatic Extradural Tumors", in *Clinical Orthopaedics and Related Research*, 169: 103, 1982.

Sudden death following fluorocarbon inhalation

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Abstract

The death of a young, previously healthy adult male following the intentional inhalation of an aerosol spray containing fluorocarbon is presented. Clinical observations during resuscitation attempts and subsequent post-mortem finding suggest that death may have been as a result of a major pulmonary insult. Experimental evidence is provided demonstrating that Freon, the most common aerosol hydrocarbon, has significant adverse effects on the cardiorespiratory system. With high concentrations of inspired Freon gas, depressed myocardial contractility and arrhythmias are seen frequently. At concentrations below the threshold for cardiac depression, Freon gas exerts its deleterious effects on the alveolar surfactant layer. As a consequence of the widespread alveolar collapse, profound hypoxemia undoubtedly is a factor leading to cardiorespiratory arrest.

Résumé

Le cas d'un jeune adulte mâle en bonne santé, décédé à la suite de l'inhalation volontaire d'un aérosol contenant du fluorocarbone est présenté. Les observations cliniques tout au long de la tentative de réanimation et les constatations lors de l'autopsie laissent croire que la cause du décès serait une atteinte pulmonaire majeure. Des données expérimentales démontrent que le gaz Fréon, l'hydrocarbure en aérosol le plus courant, a des effets nocifs importants sur le système cardio-respiratoire. En présence d'une inhalation de fortes concentrations de gaz Fréon, la dépression de la contractilité myocardique et les arythmies sont fréquentes. A des concentrations en deçà du seuil de la dépression myocardique, le gaz Fréon exerce ses effets nuisibles au niveau du surfactant. Suite au collapsus alvéolaire étendu, une hypoxémie profonde est sans aucun doute le facteur déclenchant de l'arrêt cardio-respiratoire.

Case report

A twenty-four year old, previously healthy male was brought to our Emergency Department by ambulance in cardiorespiratory arrest. Approximately ten minutes previously, just prior to collapse, his landlady had seen

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him cyanosed and gasping for air. There was no additional history regarding events immediately preceding his collapse.

On arrival, cardiopulmonary resuscitation was in progress. The patient was being oxygenated with a bag-valve-mask apparatus. No signs of recent injury were seen. there was no respiratory effort and the pupils were nonreactive. The cardiac monitor revealed asystole.

Intubation was performed and sodium bicarbonate and epinephrine were given intravenously. Within five minutes, ventricular fibrillation was noted. Defibrillation resulted in a junctional bradycardia. Initial laboratory values were: normal hemogram, Na 141 mmol/L, K 4.1 mmol/L, Cl 99 mmol/L, Glucose 7.8 mmol/L, arterial blood: pH 7.23, PO_2 22.7, PCO_2 54.2. An infusion of isoproterenol followed by dopamine achieved a blood pressure of 120/80 mg Hg. and a sinus rhythm of 100/minute. Fifteen minutes after arrival, the patient showed light reactivity; ventilatory efforts and withdrawal to painful stimuli were noted.

The patient's rhythm progressed to a rate of 150 beats per minute and then to ventricular fibrillation. Dopamine and isoproterenol were discontinued. Arterial blood gases taken at forty minutes revealed pH: 7.34, PO_2 128, PCO_2 26.7. Ventricular fibrillation persisted in spite of the institution of a lidocaine infusion, followed by intravenous procainamide, bretylium and numerous attempts at defibrillation. Attendants were simultaneously noting a progressive increase in resistance to manual ventilation. After ninety minutes of resuscitative attempts, a persistent asystole was noted on the monitor. A transthoracic pacemaker failed to capture any rhythm. The patient was pronounced dead one hundred and ten minutes after his arrival at hospital.

Information obtained shortly after the patient's death revealed that an aerosol container of Pam cooking spray and a plastic bag containing a similar fatty substance were found in the deceased's room.

Post-mortem findings included evidence of bilateral pulmonary congestion with extensive alveolar collapse and areas of patchy acute hemorrhage. Microscopic examination revealed remarkable alveolar consolidation. In addition, there were scattered foamy macrophages which stained positive for oil-red-O, indicating the presence of a lipid foreign material in the alveoli. This finding supported the suspicion of deep inhalation of a fatty substance.

Forensic examination detected trichlorofluoromethane (Freon), propane and isobutane in blood samples. These substances are the volatile ingredients of Pam cooking spray. There were no traces of alcohol or common drugs detected in blood or urine samples. It was concluded that the patient died following cardiorespiratory arrest secondary to the inhalation of the volatile substances in Pam cooking spray.

Discussion

In North America, the inhalation of various forms of commercially available solvents for the purpose of intoxication has been a recognized problem since the early 1960's. Glue sniffing was first reported in the medical literature in 1962[1] and, interestingly enough, early reports concluded that it was "probably physically harmless".[2] While early isolated reports of sudden unexpected deaths in teenage solvent abusers attributed plastic bag suffocation as the cause, increasing awareness of the problem by the early 1970's began to implicate other factors. Two reviews of the problem of "sudden sniffing death" presented a total of 144 deaths occurring between 1968 and 1977.[3,4] The reports implicated volatile hydrocarbons, pressurized in an aerosol container for the purpose of delivering the contents, as the toxic agent. In fact, Freon gas was involved in 104 of the 144 deaths reported. During the same period of time, a notable increase in the sudden deaths of asthmatic patients seemed to correlate with the increased and often excessive use of pressurized aerosol bronchodilators.[5] Information compiled from witnessed sudden sniffing deaths pointed to the likelihood of a fatal cardiac arrhythmia as the probable mechanism of death. One proposal suggested that sniffing was capable of the induction of a light plane anaesthesia with the resultant increase in cardiac irritability exacerbated by stress or increased physical activity.[3] These contentions were supported by laboratory evidence demonstrating that fluorocarbon propellants were capable of inducing serious cardiac arrhythmias when inhaled at high concentrations.[6,7,8] Additional experimental work using isolated rat papillary muscle revealed that Freon gas alone was capable of inducing significant negative inotropic effects on the myocardium. Under hypoxic conditions there was an apparent synergistic action of Freon and hypoxia to abolish nearly all cardiac contractile activity.[9]

There was also an appreciation of the fact that Freon inhalation was capable of inducing quite separate and significant physiological alterations in respiratory function. One report investigating an observation of transient reduction of arterial PO_2 following inhalation of Freon through an aerosol propellant, provided evidence to show that Freon caused bronchoconstriction and alteration in tidal volume.[10]

Our particular case report and others in the medical literature[11] suggest a form of evolving adult respiratory distress syndrome by virtue of progressive ventilatory difficulties during resuscitation. This appeared to correlate with pathological evidence found at post-mortem examination.

Findings of widespread pulmonary edema, alveolar collapse and hemorrhage in the lungs of a population of young, previously healthy subjects could be explained on the basis of hypoperfusion experienced during prolonged external cardiac massage and manual ventilation. Serial blood gases done on patients suffering cardiac arrest have shown that arterial oxyhemoglobin saturation is significantly decreased in those patients who had major cardiopulmonary abnormalities prior to arrest and whose lungs were markedly edematous and heavy at post-mortem examination.[12] However, investi-

gators from Toronto's Hospital for Sick Children, using a carefully designed experimental model have shown that the fluorocarbons in Pam cooking spray, at inspired concentrations below that necessary to induce cardiac arrhythmias, will precipitate an acute disintegration of normal alveolar surfactant.[13] One can only surmise that the progressive difficulty in manual ventilation during our resuscitation was the end result of overwhelming alveolar collapse. With an appreciation that surfactant destruction is a direct effect of Freon exposure, it is quite likely that the post-mortem findings were a reflection of the fatal consequences of a toxic inhalation.

One can now begin to appreciate that the inhalation of Freon gas by individuals for the sole purpose of intoxication is an exercise with fatal consequences deserving more attention in the form of public education. There is no explanation presently to account for the fact that this seemingly popular and accessible form of substance abuse will lead to fatal complications only in isolated individuals. However, the knowledge that Freon gas can evoke major physiological changes in the form of both cardiac arrhythmia and surfactant destruction leading to pulmonary collapse, should mandate some concern with respect to ready availability.

Conclusion

This paper focuses on the fatal consequences of the inhalation of a commercially available fluorocarbon aerosol. Clinical observations attest to the apparent irreversibility of the major alterations in cardiorespiratory function during some incidences of the inhalation of Pam cooking spray for the purpose of intoxication. In fact, there are no reported cases of successful resuscitation of those patients presenting in cardiac arrest following such an inhalation. Experimental and pathological evidence indicates that Freon is capable of inducing fatal cardiac arrhythmias and destroying pulmonary surfactant with widespread resultant alveolar collapse. While this type of substance abuse continues to present to hospital Emergency Departments with some regularity, as yet there is nothing to offer to the patient other than standard methods of life support, all of which seem to be to no avail.

Bibliography

1 Glaser HH, Massengale ON. Glue-sniffing in Children. Deliberate Inhalation of Vaporized Plastic Cement. *JAMA* 181: 300–303, 1962.

2 Massengale ON, et al. Physical and Psychological Factors in Glue-sniffing. *New Eng J Med* 269: 1340–1344, 1963.

3 Bass M. Sudden Sniffing Death. *JAMA* 212: 2075–2080, 1970.

4 Garriot J, Petty CS. Death from Inhalant Abuse: Toxicological and Pathological Evaluation of 34 Cases. *Clin Toxicology* 16: 305–315, 1980.

5 Speizer FE, et al. Investigation into the use of Drugs Preceding Death from Asthma. *Brit Med J* 1: 339–342, 1968.

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PRE-HOSPITAL CARE FORUM

Prehospital care in Japan and China

RV JOHNSTON MD

Introduction

In May of 1984 a delegation of Canadian Emergency Physicians led by Dr. Greg Powell visited both Japan (Tokyo and Kyoto) and the Peoples Republic of China (Beijing, Changsha, Quangzhou). Expertise in prehospital care from across the country was well represented in the delegation in the persons of Dr. Jane Findlater (Fredericton), Dr. Sandy Henry (Ottawa), Dr. Don MacLachlan (Calgary) and Dr. Jim Christenson (Vancouver).

The purpose of the delegation was to exchange technical information with both the Chinese and Japanese hosts on a variety of subjects relating to Emergency Medicine. Our experience in various aspects of prehospital care seemed to be one of the more sought after topics by our hosts.

The group however had the opportunity to learn about quite different methods of care in the prehospital phase in both countries visited.

Japan

The delegation's first exposure to the problem and extent of prehospital care came while on route to the Nippon Medical School for the first morning's briefing in Tokyo. Police, ambulance and sirens rushed past our bus only to stop a short distance ahead. By the time the traffic cleared, and the bus proceeded, the ambulance was pulling away and a somewhat twisted bicycle lay beside a covered body.

At Nippon Medical Center our North American concept of an Emergency Department seeing large numbers of undifferentiated patients was challenged. The critical care center (unit) was located on the fourth floor of a typical hospital and received only patients with acute life threatening medical/surgical problems. There was an ambulance bay but no specific Emergency Department. Patients with less critical illness, but still requiring ambulance transport, would be taken to a primary or secondary level care facility.

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The ambulance service in urban Japan is fire department based; a tour of the Tokyo fire department was arranged for the following day. Ambulance personnel are all fire fighters first and prehospital care workers subsequently. In a city of eleven million, the ambulance division responds to approximately three hundred thousand calls per year. These consist of a somewhat different mix than would be expected in North America in that ischemic heart disease is much less prevalent. As a result the ambulance personnel are at a level consistent with an EMA-1 in Canada. A 228 hour course covers many of the modules that would be used in North American courses. There were no ambulance members at an advanced life support level, although the company chief required an additional 114 hours of training in the administrative and supervisory areas. The reason for no advanced life support in personnel was explained to be related to the low incidence of out of hospital cardiac arrest from ischemic heart disease, and the rapid response time, both to the scene and to the hospital; three minutes to the scene and fifteen minutes to the hospital. The survival figures which were quoted were very impressive; approximately 20% survival from out of hospital cardiac arrest. In terms of equipment, the vehicles themselves were modified Toyota or Nissan vans. They seemed very compact, particularly compared to the large North American vans or modulars with which we are familiar. The cost seemed to be low, approximately fifteen thousand dollars Canadian. The department operated 153 units, and equipment included that usually found in North American vehicles, with the exception of vacuum type splints for extrication and immobilization.

Communications are obviously critical in such a large urban area. A central entry number is provided (119). Call-takers and dispatchers have at their finger tips a constantly updated (by computer of course) list of bed availability throughout the city. Furthermore, the presence of a specialist at one or another hospital can be determined in order to direct the ambulance crew. Communication with a physician appeared to be a relatively infrequent occurrence and was not formally arranged.

Air evacuation did not play a major role in the prehospital scenario in Japan. The Tokyo Fire Department did own five helicopters which, on occasion, would transport critically ill patients from the neighbouring islands; however, its main duty appeared to be fire suppression/rescue.

The cost of the system was unable to be determined since ambulance and fire budgets were not specifically separated.

The visit to Kyoto (also Senre) confirmed much of the information gathered at Tokyo. A lecture from Dr. Ischida detailed the overall scope of prehospital care in Japan. He noted that the minimum standards for training and equipment had been established nationwide in 1961. In 1983, nation-wide, there were 38,000 EMT's and a total of 4,000 vehicles, the Tokyo figures of response times were essentially confirmed; 10–20 minutes 8.9%, 5–10 minutes 38.6%, 3–5 minutes 31%, less then 3 minutes 19%. In his study of treatment given by prehospital care system he noted several interesting points. Temperature regulation was performed in 35% of cases, O_2 administered in 13.5%, dressing applied in 12%, hemostasis attempted in 11%, airway maintenance in 7.2%, immobilization of fractures in 5% and cardiac massage in 1.5%. Temperature regulation appeared to be the equivalent of keeping the patient warm. Unfortunately there was insufficient time for any of us to engage in a ride-a-long to see the system first hand.

The Peoples Republic of China

Our visit to China began in Beijing. From a medical standpoint the delegation visited a number of different hospitals – namely, the Beijing Heart, Lung and Blood Vessel Institute, the Beijing Friendship Hospital and the Peoples Liberation Army Hospital. The staff of these institutes, as well as our hosts (the China Association of Science and Technology) were very proud of their hospitals, currently being renovated and equipped with the most modern equipment. From a prehospital care point, however it became apparent that the system was currently being revised, and as such, unfortunately was not open to visitors. Apparently individual hospitals operate their own ambulances which may be called in the case of an emergency. Alternatively a central ambulance station exists, comprising 70 ambulances and staffed with both physicians and ambulance personnel. Finally (and more likely) it appeared that the patient would arrive via any other conveyance; in particular, a truck, a horse cart or even a bicycle.

The level of training of physicians in the Peoples Republic of China is variable, and the level of training of ambulance physicians was similarily variable. The delegation met with Dr. Li, head of this particular service, to discuss in more detail the Canadian prehospital care system. Unfortunately the requests to view the central ambulance station could not be accomodated.

From a prehospital care point of view, Changsha in Hunan province was similar to that in the country's capital Beijing. The delegation members' interest in prehospital care noted several factors in those two cities which would severely impede an efficient and effective service as we know it in North America. First, access would be difficult given the lack of telephones as usual household items; secondly, the traffic situation, in Beijing in particular, seemed quite overwhelming. The latter point is, no doubt, a transient phenomena as Beijing is currently under major construction; however, it would appear that for several years rapid ambulance response would be unobtainable. This may be one reason the country, at least in the cities we visited, has opted for a physician staffed ambulance.

The delegation visit to the southern city of Quongzhou (Canton), in particular the Red Cross Hospital, was instructive in that the hospital had a well developed ambulance service. The city itself was covered by a series of call boxes to summon emergency aid. The

ambulance crew consisted of a physician, a nurse, a driver and a stretcher bearer. The ambulance nurse had a special training in the use of equipment, rescue, and the treatment of common ailments. The physicians sent on ambulance calls were all specialists; ie. if the call involved trauma, a surgeon would be sent, if the call was medical, an internist, etc. There was no particular ambulance training for these individuals, but difficult cases would be reviewed retrospectively and post mortem discussion would occur. Members of the delegation questioned the response time of assembling such a group. We were told that the crew was available 24 hours a day; that time leaving the hospital was 5 minutes during the day and 10 minutes at night. A second car would be readied upon departure of the first. Our inspection of the vehicles (Japanese vans) explained part of the delay, in that equipment (inflated rubber bags of oxygen and airway supplies) would have to be collected prior to departure.

Summary

In summary, the prehospital care in Japan is predicated upon a rapid response, emergency medical technician staffed, primarily fire based ambulance system. This is supported by an elaborate communication system, as well as a fully categorized hospital system.

The system in the Peoples Republic of China varied depending upon the locale, but was generally a physician staffed hospital based system with somewhat longer response times. Data was unavailable to substantiate the benefit of this system.

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6 Taylor GJ, Harris WS. Cardiac Toxicity of Aerosol Propellants. *JAMA* 214: 81–85, 1970.

7 Azar A, et al. Cardiac Toxicity of Aerosol Propellants. *JAMA* 215: 1501, 1971.

8 Reinhardt CF, et al. Cardiac Arrhythmias and Aerosol Sniffing. *Arch Environ Health* 22: 265, 1971.

9 Kilen SM, Harris WS. Effects of Hypoxia and Freon 12 on Mechanics of Cardiac Contraction. *Am J Physiology* 230: 1701–1707, 1976.

10 Brooks SM, et al. Changes Occurring after Freon Inhalation. *Am Rev Resp Dis* 105: 640–643, 1972.

11 Fagan DG, et al. Acute Pulmonary Toxicity of a Commercial Fluorocarbonlipid Aerosol. *Histopathology* 1: 209–223, 1977.

12 Fillmore SJ, et al. Serial Blood Gas Studies during Cardiopulmonary Resuscitation. *Annals of Int Medicine* 72: 465–469, 1970.

13 Swann HE, et al. Inhalation of Aerosols by Multi-species in a Head Exposure Chamber with Simultaneous Cardiopulmonary Measurements. *Toxicology and Applied Pharmacology* 22: 307, 1972.

TOXICOLOGY FORUM

Acute digoxin poisoning: using scavenger antibodies

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Digoxin toxicity due to acute overdosage is fortunately a relatively uncommon occurrence. However, its management is worth reviewing for several reasons. Its mortality rate is significantly higher than for other drugs that are more commonly involved in poisoning, it differs from chronic digitalis toxicity and a new specific therapy has recently been developed.

As with most acute poisonings, the minimal toxic amount of digoxin is unknown. However, 5 mg or less can cause toxicity. If the patient is on chronic digoxin or diuretic therapy, or if he has pre-existing heart disease, then he would be more likely to develop toxicity from small overdoses.

Clinically the patient presents with vomiting, cardiac dysrhythmias, drowsiness and lethargy. Hyperkalemia is common in contrast to the hypokalemia of chronic digitalis toxicity. Onset of toxicity may occur within the first hour after overdose and peaks a few hours later. Duration of toxicity is dependent upon amount of the drug ingested but may persist for 24 hours or longer. If digitoxin rather than digoxin is involved, then onset and peak of toxicity occur later and the duration is longer.

The hyperkalemia of acute digitalis poisoning may be profound and life-threatening. The degree of potassium elevation has been shown to be a very good prognostic sign. It has a better correlation with survival than serum digoxin level or ECG findings. The pathophysiology of this hyperkalemia is interesting and can be viewed as a "first step" in the development of the hypokalemia of chronic digitalis toxicity. Digitalis compounds inhibit membrane adenosine triphosphatase (ATPase). This results in reduced potassium influx into myocardial and other muscle cells producing intracellular hypokalemia and extracellular hyperkalemia. In chronic digitalis toxicity, hypokalemia develops because the renal response to the increased potassium load is to excrete it. Simply put, the potassium is leached from the body over time. However, in both acute and chronic digitalis toxicity, it is the myocardial intracellular hypokalemia that contributes to the cardiac dysrhythmias.

Management of acute digitalis poisoning can be

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divided into nonspecific and specific therapy. For the purpose of brevity, we shall assume that assessment of level of consciousness and measurement of vital signs reveal the patient not to be in imminent danger, or that initial resuscitative measures have stabilized the patient. Cardiac monitor leads should be immediately attached to the patient and the ECG should be continuously monitored. Blood should be sent for digoxin and potassium levels. Important historical data include the presence of pre-existing heart disease and current digoxin or diuretic therapy. The estimated amount of the overdose is important because it is used to calculate the dose of the antidote.

As in any other acute poisoning, measures directed towards decreasing the absorption and increasing the excretion of the ingested toxin should be undertaken. Gastric emptying by ipecac-induced emesis or gastric lavage should be considered. However, the amount of spontaneous vomiting that has already occurred may make these procedures unnecessary. Activated charcoal with an accompanying cathartic should be administered to decrease absorption. Because of the large volume of distribution of digoxin, nonspecific measures of increasing excretion are not effective and therefore not indicated. These include forced diuresis, dialysis and hemoperfusion. However, for digitoxin poisoning, multiple dose oral cholestyramine therapy has been shown to increase its excretion into the stools.

Cardiac dysrhythmias are managed with appropriate drugs and the insertion of a transvenous pacing catheter. Choice of anti-arrhythmic agent is dependent upon the nature of the dysrhythmia. Consider atropine for significant bradycardia or heart block and lidocaine and/or phenytoin for ventricular dysrhythmias. Lower doses of phenytoin may improve atrioventricular conduction and might be useful in heart block. In all situations of ECG evidence of acute toxicity or hyperkalemia (6.0 mmol/L or greater), the insertion of a transvenous pacing catheter should be carried out because the severely digitalis-poisoned myocardium usually responds poorly to anti-arrhythmic agents.

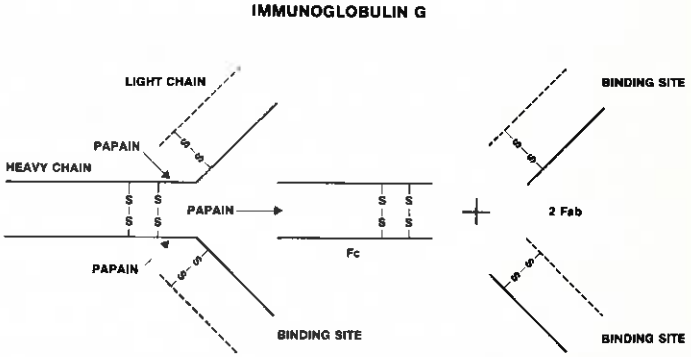
If the degree of hyperkalemia is life-threatening, then emergency measures directed towards the lowering of the serum potassium would be indicated. These include any or all of the following therapies: glucose and insulin, sodium bicarbonate and Kayexalate enemas. The first two treatment modalities make the most sense from the physiologic point of view as they would tend to drive the potassium back into the myocardial cells as opposed to Kayexalate therapy which removes it from the body. However, when faced with extreme hyperkalemia, all three approaches may be required.

If dysrhythmias or hyperkalemia persist despite the above therapeutic interventions and if they are of life-threatening magnitude, then specific antidotal therapy is indicated. This consists of the administration of anti-digoxin antibodies.

Anti-Digoxin Antibodies

Anti-digoxin antibodies were first produced for the development of the radioimmunoassay that measures serum digoxin levels. However, this antibody's large size (150,000 daltons), makes it unsuitable for the treatment of acute digoxin poisoning. It cannot be excreted by the kidneys, therefore the digoxin would remain within the body with the potential to cause toxicity at a later date when the antibody is catabolized. Of greater risk is the fact that this foreign protein is itself antigenic, thus capable of producing various forms of allergic reactions.

These problems were overcome by 15–20 years of elegant research and development by Smith and his colleagues. They injected protein-bound digoxin into sheep and harvested the IgG anti-digoxin antibodies. These were subjected to digestion by the enzyme papain, producing three subunits, two identical Fab fragments and one Fc fragment (see Figure). The Fab fragment is small (50,000 daltons) and contains the binding site for digoxin. Because of its size, it is excreted in the urine even when bound to digoxin and is non-antigenic. A subsequent multicentred field trial in 26 seriously digitalis poisoned patients demonstrated efficacy and safety. In fact, in many of these cases, the reversal of the toxic manifestations was so rapid and complete that it bordered on the miraculous.



Indications for Fab therapy in acute digitalis poisoning are life-threatening cardiac dysrhythmias or hyperkalemia unresponsive to conventional therapeutic interventions. It is administered in amounts equimolar to the estimated amount of digitalis that was ingested; hence the importance of a historical estimation of this amount. Unfortunately, at the time of the writing of this commentary, there is as yet no commercial supply of antidigoxin Fab fragments. Hopefully this will be rectified in the near future.

The use of "scavenger antibodies" represents a new frontier for medical toxicology. Its virtue is that it can hasten the excretion of toxins with large volumes of distribution that are unaccessible to other more invasive therapeutic interventions such as hemodialysis and hemoperfusion. The Fab fragments remove tissue-bound digoxin as well as serum digoxin. Perhaps this approach could be used for other serious poisonings such as those due to tricyclic antidepressants.

Additional Reading

Smith, T.W., Butter, V.P., Haber, E. et al: Treatment of life-threatening digitalis intoxication with digoxin-specific Fab antibody fragments. *New Engl. J. Med.* 307:1357–1362, 1982.

Noticeboard

Emergency Physicians
Applications are invited for full-time and major part-time casualty officers for the Emergency Department of this 510 bed teaching hospital. Formal training in Emergency Medicine is required. Preference will be given to those with experience in Family Medicine. Opportunities for part-time Family Practice are available.

Submit curricula vitae to:
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Director of Emergency Services
Mount Sinai Hospital
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Enquiries should be directed to:
Bruce M.T. Rowat, M.D., F.R.C.P.(C)
Director, Emergency Medicine Residency Programme
c/o Toronto General Hospital
101 College Street,
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Contact:
Dr. R. V. Gerace
Dept. Emergency Medicine
Victoria Hospital
275 South Street
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519-432-2352

Meetings to note

The Institute for Emergency Medical Education in co-operation with Washington Chapter of The American College of Emergency Physicians presents **KAUAI 1984 "CURRENT CONCEPTS IN EMERGENCY CARE"**, 5th Annual Meeting, Kauai Surf Resort, Kauai, Hawaii, December 2nd – 7th, 1984.

For information contact: Group Travel Department/Georgine Fleck, Kailani World Travel, 4192 Meridian Avenue, Bellingham, Washington 98227-9951. Phone: USA 800-426-2561, Washington State 800-562-2597 or 206-671-1800.

This program has been reviewed and is accredited for 25 hours of Category I Credit for the AMA and ACEP. It has been reviewed and accredited for 25 hours of prescribed credit for the AAFP.

Appropriate nursing credits will be applied for.

CME Calendar

	9th Annual Course on Emergency Management
DATES:	Thursday May 9th, 1985 Friday May 10th, 1985 Saturday May 12th, 1985
SITE:	Holiday Inn (Downtown) 89 Chestnut Street (behind City Hall) Toronto, Ontario, Canada
COURSE CONTENT:	This course has been designed by and for both part-time and full-time emergency physicians. It will focus upon common practical emergency problems with active participation in workshops.
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CREDITS	Hour for hour credits (21) applied for as in past years to the College of Family Physicians of Canada and the Canadian Association of Emergency Physicians (CAEP)