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President's Notebook

For a whole host of reasons it is not hyperbole to state that our Fifth Annual Scientific Assembly in Vancouver was our best yet. The superb organization, gratifying turnout and attractive venue all contributed, as did the scientific and social programs. In terms of the business of the Association though, we were I think most productive. All of our Standing and Ad Hoc Committees found time to meet, some several times. And our working groups put the finishing touches on some position papers which were eventually adopted by the Association. This process has been I feel very positive and some of those positions are reproduced in this issue of the "Review" for your information.

One issue that I would like to ask you specifically to think about is that of economics. Many members have identified this as a concern, and I think there is a role for the Association here. CAEP can serve to co-ordinate and facilitate realistic tariff and fee schedule bargaining in the different provinces. We need to identify people in each province involved in negotiations, and begin to exchange information on their current status, and future strategies and plans. Please give this matter some thought and let the Executive know how you think CAEP should proceed in this area.

Another area, also arising out of discussions at the Annual Meeting, that deserves further thought is the role of affiliate members. We have, over the past two years made, I think, a very successful attempt to broaden the base of input into the Association. But we are still perceived in some quarters as representing the interests of "Royal College" Emergency Physicians. I feel that is wrong – CAEP should become the representative voice for *all* of Emergency Medicine. There are forums within the Royal College to pursue the specific interests of fellows. Other specialty associations function effectively with a large number of G.P.'s as affiliate members, and CAEP should be no different. G.P. – Emergency Physicians collectively account for a large volume of Emergency Medicine in Canada, and they currently have no focus for this interest.

A final item I would like to explore is that age-old one of "turf" – but from a different perspective. For the early years of the development of our specialty, we found ourselves fighting and elbowing our way onto other's turf, (putting on casts, inserting chest tubes, etc.), as well as occupying previously unoccupied territory (toxicology, cardio-pulmonary resuscitation etc.). One area that has *always* been undisputed Emergency Medicine "turf" has been pre-hospital care. But the tide has turned – I think we should now become healthily paranoid about territorial incursions. I recently was invited to another University Centre to discuss the implementation of an advanced life support program. Once there, it became quite clear to me that there was no intent whatsoever to involve Emergency Physicians in the program, in either training or base hospital physician roles. It was all to be done by the intensivists. Similarly, a large part of the active lobbying for both Designated Trauma Units and Advanced Life Support Pre-hospital Care Programs across this country is coming from surgeons, not Emergency Physicians. Pre-hospital care and E.M.S. are well within the domains of Emergency Medicine, but we stand the real risk of losing them by default.

Peter L. Lane MD

Dosage in Adult Patients with Impaired Renal Function

MEFOXIN* may be used in patients with reduced renal function but a reduced dosage should be employed and it is advisable to monitor serum levels in patients with severe impairment.

In adults with renal insufficiency, an initial loading dose of 1 g to 2 g should be given. After a loading dose, the following recommendations for maintenance dosage may be used as a guide:

| RENAL FUNCTION | CREATININE CLEARANCE mL/min | DOSE | FREQUENCY |
|-------------------------|-----------------------------|---------|---------------|
| Mild impairment | 50-30 | 1-2 g | every 8-12 h |
| Moderate impairment | 29-10 | 1-2 g | every 12-24 h |
| Severe impairment | 9-5 | 0.5-1 g | every 12-24 h |
| Essentially no function | <5 | 0.5-1 g | every 24-48 h |

In the patient undergoing hemodialysis, the loading dose of 1-2 g should be given after each hemodialysis, and the maintenance dose should be given as indicated in the Table above.

Infants and Children

The recommended dosage in children three months of age and older is 80 to 160 mg/kg of body weight per day divided into four to six equal doses. The higher dosages should be used for more severe or serious infections. The total daily dosage should not exceed 12 g.

At this time no recommendation is made for children from birth to three months of age (see PRECAUTIONS).

At present there is insufficient data to recommend a specific dosage for children with impaired renal function. However, if the administration of MEFOXIN* is deemed to be essential the dosage should be modified consistent with the recommendations for adults (see Table above).

PROPHYLACTIC USE

For prophylactic use, a three-dose regimen of MEFOXIN* is recommended as follows:

Vaginal or abdominal hysterectomy and abdominal surgery

2 g administered intramuscularly or intravenously just prior to surgery (approximately one-half to one hour before initial incision).

The second and third 2 g doses should be administered at 2-6 hour intervals after the initial dose.

Cesarean Section

The first dose of 2 g should be administered intravenously as soon as the umbilical cord has been clamped. The second and third 2 g doses should be given intravenously or intramuscularly four hours and eight hours after the first dose.

AVAILABILITY

Sterile MEFOXIN* is a dry white to off-white powder supplied in vials containing cefoxitin sodium as follows:

No. 3356 1 g cefoxitin equivalent in boxes of 10 vials

No. 3357 2 g cefoxitin equivalent in boxes of 10 vials

PRODUCT MONOGRAPH AVAILABLE ON REQUEST

1. Quintiliani, R.: Overview of cephalosporin antibiotics in the 1980's, in "Considerations in the selection of antimicrobial chemotherapy, a round-table discussion on treatment concepts", Merck & Co. Inc., 1982, pp 6-25.
2. Trunkey, D.D.: Cephalosporin in the management of trauma, *Ibid.* pp 44-52.

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Resident's Corner

The resident attendance at the annual CAEP assembly held in Vancouver in April was superb. It's exciting to see that the resident involvement in CAEP is becoming stronger with each passing year. Representatives from most of the residency programs attended the Resident Committee meeting, and the general enthusiasm led to some interesting discussion. I will briefly touch on some of the highlights at this time.

Our annual elections were held, and I am pleased to present the following as our 1984-85 Resident Committee executive:

Chairman: Dr Pauline Head - Calgary

Vice-Chairman: Dr Dave Williams - Ottawa

Program Representatives:

Dr. Robert Noseworthy - Vancouver

Dr Stuart Turner - Calgary

Dr Ross Claybo - Toronto

Dr Dave Williams - Ottawa

Dr Jillene McEwen - Kingston

Dr Jim Welch - Montreal

Dr Dave Austin - London

It is a well-known fact that every residency program in Canada has electives which are deficient due either to lack of patient volume or lack of local expertise. For this reason, the elective bank was established to give all residents opportunity to apply elsewhere for electives in order to obtain the best experience possible. Unfortunately, there were no new submissions to the bank last year. A current list of the electives in the bank has been circulated to all representatives. Please submit any electives which you feel are outstanding, as well as those to be avoided. Electives not well represented in the bank at this time are emergency pediatrics, trauma, and disaster planning.

Perhaps the hottest issue at the meeting was that of the discrepancies which exist among the various residency training programs. A very well-attended meeting was held between residency program representatives and program directors, during which many interesting topics were discussed. One of the foremost concerns was the amount of *totally* unsupervised time that many residents are spending during their Emergency rotations. The general feeling after much discussion was that there should be a staff member available IN THE DEPARTMENT at all times for discussion of cases and for informal teaching during the quiet periods.

Both residents and program directors agreed that research should be included in the residency programs. However, the general feeling was that, if research is to be mandatory, then time, which is reserved for research work, must be allotted during the year. Some staff members should also be involved in research in order to serve as an example as well as a resource for the residents involved. A case report per resident per year, to be presented at the CAEP annual assembly was felt to be a reasonable expectation as well.

Core content of formal lectures, as well as the time spent per week at formal lectures, varies considerably across the country. Many programs are not giving adequate coverage of important topics such as disaster planning, prehospital care, Emergency administration and medicolegal issues. Many programs do not have regular mock oral examinations or practice written examinations throughout their curriculum.

These are just a few of the exciting issues discussed at the meeting. No firm guidelines or actual decisions came out of the discussions. However, the program directors were made aware of the problems and concerns that the residents have about the training programs and we can only hope that they left the meeting with a keen interest to improve upon any deficiencies which were pointed out at the meeting.

I look forward to meeting with the nine resident representatives at our semiannual committee meeting to see what progress has been made towards improvement of the programs.

In closing, I would like to wish all those residents who finish their residencies this year all the best on their exams and with their new jobs. And for those of us who are still in the thick of our training - have a good year!

Pauline W. Head, Resident Committee Chairperson

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CAEP Position Papers

CAEP Position Paper on Drinking and Driving *Adopted April 1984*

Whereas nearly 50% of M.V.A.'s are associated with drivers who have been drinking alcohol.

Whereas the majority of fatal motor vehicle accidents involve those under the age of 40.

Whereas many of those not fatally injured are disabled for the rest of their lives.

Whereas Emergency Physicians are the members of the medical profession to give initial care to the victims of motor vehicle accidents.

a) Be it resolved that the Canadian Association of Emergency Physicians take a lead, advocacy role in identifying the causes, the preventative measures and optimal methods of treatment to minimize the impact of alcohol related motor vehicle accidents in Canadian society.

b) Be it resolved that CAEP should play a major role in highlighting the social unacceptability of drinking alcohol and driving.

As such, it is the position of CAEP:

- i) That driving a motor vehicle be considered a privilege rather than a right.
- ii) That crimes involving drunk drivers who cause personal injury or death should be considered as crimes of violence, and should be tried as such, under the appropriate Section of the Criminal Code (e.g. Assault causing bodily harm, criminal negligence causing death, manslaughter, etc.)
- iii) That penalties for impaired driving convictions be reviewed with a view to improving their value, both as a deterrent, as well as their relationship to the communities' view of the severity of this crime.
- iv) That blood alcohol levels be drawn by a trained technician, on police direction by statute. Such levels should be reserved for patients who, for medical or other reasons cannot provide a suitable breathalyzer estimate of blood alcohol level.
- v) That refusal to provide a breathalyzer sample or blood sample carry the same penalty as being impaired.
- vi) That there should be increased general public and school education of the effects of alcohol and driving, and that taxes on alcoholic beverages should be increased to underwrite the costs of these educational activities.
- vii) That random roadside checks, as an active deterrent, be increased in as cost effective manner as possible.
- viii) That licences should include the picture of the licensee to further discourage driving with a suspended licence.

CAEP Position Paper on Citizen CPR Training *Adopted April 1984*

Preamble:

Citizen CPR training refers to the process of educating the general population in the cognitive and psychomotor skills of artificial ventilation and external cardiac massage. These skills include the knowledge of coronary disease risk factors, recognition and response to cardiac emergencies, and physical training in cardiopulmonary resuscitation introduction. It has been demonstrated that the morbidity and mortality of cardiac arrest victims can be significantly reduced in communities where a large portion of the population has been educated in the techniques of CPR. The methodology used in planning and training programs for those large numbers of citizens must be coordinated through a variety of different training organizations, and emergency physicians must be involved in supporting the development of appropriate training in their respective communities and in Canada as a whole. Emphasis must be placed on making CPR training programs easily accessible both geographically and financially to as many Canadians as possible. Also, considerable emphasis must be placed on periodic retesting and retraining in the cognitive and psychomotor skills of CPR so that those initially trained in the skills may retain them at optimum levels.

Resolution:

Whereas it has been well documented that the prompt administration of effective CPR to victims of cardiac arrest has a positive effect on the outcome for such individuals, and whereas the chance for prompt administration of effective CPR increases with the number of bystanders trained in this procedure, therefore be it resolved that:

- i) CAEP endorses the fundamental principle that basic cardiac life support training be provided to the maximum number of citizens in every Canadian community.
- ii) CAEP recognizes the standards of basic cardiac life support training as established by the Canadian Heart Foundation.
- iii) CAEP members actively involve themselves in leadership roles on the appropriate cardiac care committees, nationally, provincially, and within their own communities.
- iv) CAEP endorses the inclusion of CPR training and certification in the curricula of all Canadian medical and allied health care training programs.
- v) CAEP endorses the principle of periodic retraining and reassessment in the cognitive and psychomotor skills of basic cardiac life support.
- vi) CAEP encourages and promotes the development of scientific research in the area of cardiopulmonary resuscitation.

Position Paper on the use of seat belts
Adopted April 1984

Whereas, it has been conclusively shown that the safest way to travel in a motor vehicle is in a properly adjusted seat belt harness, and whereas, at present no Canadian Province has a law requiring the use of seat belts in school buses, *be it resolved that* CAEP urges appropriate Government Officials to make the installation of seat belts mandatory in school buses, and to make their use mandatory.

CAEP Position Paper on Medical Control of Prehospital Emergency Services
Adopted April 1984

The Canadian Association of Emergency Physicians has previously passed a resolution noting the planning, design and operation of the total EMS system falls within the domain of Emergency Medicine and that prehospital care is an integral part of the Emergency Medical Services system.¹ Since those practising Emergency Medicine are recipients of patients from the prehospital phase, it may be that the receiving physician has assumed liability for the prehospital medical care given by the agents in the field.^{2,3} Because the system's purpose is to provide emergency medical care, the physician who renders direct or indirect patient care must be in control of the medical aspects of the system.⁴

The provision of control in the prehospital phase is dependent upon numerous factors including:

- (1) the level of prehospital care provided, i.e. ALS vs. BLS;
- (2) the manner in which the prehospital care is organized, i.e. provincial, civic or private;
- (3) the presence of provincial legislation under which prehospital workers are certified;
- (4) the presence of categorization of facilities;
- (5) the extent and sophistication of communication systems;
- (6) the presence of advanced training programs for prehospital care providers.

Whereas one of CAEP's stated positions is that EMS falls within the domain of Emergency Medicine and whereas there is a marked discrepancy in the level of medical control practised across Canada, *be it resolved that*:

- (1) physicians whose continuing interest lies in the provision of prehospital care be responsible for the medical direction in all aspects of this phase;
- (2) provincial governments designate an administrative (off-line) medical director with appropriate EMS experience who is responsible for the overall planning and development of a medically sound EMS system for regional prehospital, inter-hospital, and in-hospital programs;⁵
- (3) where advanced life support procedures are performed in the field, these be done under the direct

medical control of a qualified physician (on-line) either via voice-to-voice communications, or standing orders (protocol) approved by the EMS medical director;

- (4) certification for personnel using advanced life support procedures require the ongoing approval of the EMS medical director;
- (5) physicians designated as medical directors of programs have control over the establishment of standards and capabilities of all medical and communication systems used;
- (6) prehospital care services providing basic life support procedures be directed medically by a qualified physician (off-line);
- (7) qualified physicians play an integral part in all aspects of both advanced and basic life support provider training including planning, implementation, assessment, both initially and subsequently for continuing education.
- (8) qualified physician be defined as a physician with certification in Emergency Medicine or significant EMS experience;
- (9) provincial governments be urged to recognize prehospital care providers at basic, intermediate and advanced support levels through health occupation acts, ambulance regulations and enabling legislation;
- (10) resource hospital be designated in order to facilitate direct (on-line) medical control;
- (11) physicians responsible for medical control be in a position to direct prehospital care providers to the institution most appropriate for the particular patient's needs;
- (12) qualified physicians be in a position to evaluate prehospital care, both immediately and in a retrospective fashion so that medical control may be based upon sound data.

References

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Venous air embolism

D. MONTOYA MD

Abstract

Venous air embolism is a well recognized complication of diagnostic, therapeutic and surgical procedures; yet, its true incidence is unknown. Upon entry of air into the venous system a pathophysiologic cascade is initiated affecting predominantly the cardiopulmonary system. Many methods of detecting and treating venous air embolism have been advocated, but effective management is based on a clear understanding of the clinical presentation and pathophysiology.

Résumé

L'embolie gazeuse constitue une complication bien connue des actes diagnostiques, thérapeutiques et chirurgicaux; cependant, son incidence réelle est inconnue. Au moment où l'air pénètre dans le système veineux, une série de réactions physiopathologiques est déclenchée, affectant principalement le système cardiopulmonaire. De nombreuses méthodes de dépistage et de traitement de l'embolie gazeuse ont été mises de l'avant, mais un traitement efficace se fonde sur une bonne compréhension de la présentation clinique et de la physiopathologie.

Introduction

The pathophysiology, clinical consequences and treatment of venous air embolism have intrigued famous men of science for many years. In 1822 the use of a cannula to remove intravascular air was described by Magendie. He also reported a case three years earlier of the first apparent fatality due to air embolism.¹ In his monograph "Recherches sur l'Introduction Accidentale de L'Air dans les Veines" published in 1839, Amussat described the principle sites of entry of venous air during procedures involving the neck and upper chest. He referred to these areas as "regions dangereuses".² In 1844 Erichsen described the classical changes in heart sounds as venous air enters the right heart.³ Still others like Dupuytren, Velpeaux and Virchon added to the growing body of knowledge in this area such that by the mid 1800's there were dozens of documented cases of venous air embolism in the world literature.²

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Dr D. Montoya
Resident, Emergency Medicine
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More recently, Harkins, Harman and others throughout the 1930's experimented with the physiological consequences of injecting air into the venous system. Varying volumes and rates of injection were manipulated to determine those factors resulting in a fatal outcome. Durant in 1947 introduced the left lateral decubitus position with Trendelenberg (Durant's manoeuvre) as a non-invasive therapeutic manoeuvre designed to keep air at the apex of the right ventricle.⁴ In 1959 Shivpuri refined Erichsen's classical work of 1844 by describing the drum sign which preceded the millwheel murmur of venous air embolism.⁵ Erichsen was the first to advocate closed cardiac massage in the treatment of venous air embolism in 1964⁶ and in 1966 Michenfelder introduced the use of a right atrial catheter for the aspiration of intracardiac air.⁷

Much work continues to be done in the area of venous air embolism and it is the focus of this review to address the scope of this entity, its clinical presentation and effective management based on an understanding of its pathophysiology.

Etiology

KW Kizer compiled a list of conditions and circumstances leading to documented or suspected air embolism⁸ [Table I]. The possible etiologies are myriad; nonetheless, the fundamental basis for generating an air embolism remains constant: a vein which is open to air and has a pressure gradient that favours entry of air into that vein.

TABLE I: [modified from Kizer et al Radiology 1982]

Trauma:

- penetrating injuries esp. head/neck/chest/abdomen/pelvis
- blast injuries
- high pressure air injection
- autotransfusion

Surgical:

- head, neck, spine, chest, abdomen, pelvis
- pericardiocentesis
- thoracocentesis
- percutaneous lung biopsy

OB/GYN:

- childbirth
- abortions
- douching/vaginal insufflation
- cunnilingus

Intravascular:

- transfusions
- TPN (total parenteral nutrition)
- IV cannulation
- central venous catheterization

Miscellaneous:

- venography
- decompression sickness "chokes"
- pneumography
- PEEP (positive end expiratory pressure)

Pathophysiology

The spontaneous entry of air into the venous system depends on the magnitude of the pressure gradient between the exposed vein (portal of entry) and the right side of the heart. The magnitude of the gradient drawing air into the vein can be increased by decreasing the mean intrathoracic pressure or by increasing the vertical distance between the portal of entry and the right heart. Factors such as hypovolemia (blood loss, dehydration, anemia) which lead to a low central venous pressure (CVP) will also enhance the gradient.

A surprisingly large amount of air can pass into the venous system very quickly. Flanagan et al⁹ calculated that 100 cc/sec would flow through a 14 gauge needle with a 5 cm pressure gradient. Others have shown similar figures.¹⁰ The minimal amount of air that will cause death in man is unknown. Some have quoted as little as 50 cc as a bolus¹¹ and there are reports of deaths with 100 cc¹² and 300 cc¹³ of air. There seems to be little doubt that both the total amount of air, as well as the rate of entry, are important factors in the outcome. In canine experiments injection of 5–8 cc/kg/min has proven fatal whereas 1400 cc were tolerated before death when injected over 460 min.¹⁴

Upon entry of air into the venous system, Durant et al^{4,15} showed that air bubbles 30–40 μ in diameter behave like plastic solids and lodge in small pulmonary vessels. They also showed that injection of air led to the formation of a bloody foam that obstructed the right ventricular outflow tract and pulmonary vasculature. The resultant turbulent flow around the air bubbles enhances fibrin formation with secondary aggregation of platelets, RBC's and fat globules. Smaller pulmonary arterial vessels are obstructed with these aggregates.¹⁶ This obstructive conglomerate of air and blood products sets the stage for a host of complex pathophysiologic consequences (Table II).

TABLE II: Pathophysiologic consequences of venous air embolism

| |
|-------------------------------------|
| Increased airway resistance |
| Pulmonary edema |
| Decreased lung compliance |
| Obstruction of pulmonary blood flow |
| Paradoxical embolization |
| Hypoxemia |
| Adult respiratory distress syndrome |

INCREASED RAW (INCREASED AIRWAYS RESISTANCE)

Khan et al¹⁷ noted an increased RAW with oxygen and nitrogen gas emboli while CO₂ and inert gas emboli (helium, argon, xenon, neon) had no effect. Carbon dioxide's great solubility in blood could explain its lack of effect on RAW but why the inert gases had no effect is unknown.

Bronchospasm (increased RAW) seen with O₂/N₂ could be prevented by: a) pretreating the animals with heparin, b) inducing thrombocytopenia or c) by ad-

FIGURE I: [Adapted from Khan et al J. Appl. Physiol. 1972]

AIR EMBOLISM

Heparin

ACTIVATION OF THROMBIN

Thrombocytopenia

PLATELET AGGREGATION

RELEASE OF SEROTONIN/HISTAMINE/OTHER SUBSTANCES

Methysergide (antiserotonin)

AIRWAYS CONSTRICTION

ministration of methysergide (an antiserotonin agent). Figure I outlines their proposed pathophysiological schema.

PULMONARY EDEMA

Two mechanisms have been proposed to account for the development of pulmonary edema. The first is increased capillary permeability. Evidence for this includes increased lymph and lymph/plasma protein ratio after air embolism¹⁸ and is further supported by ultrastructure studies showing cell injury and herniation through the basal lamina.¹⁹ The second theory, that of increased hydrostatic pressure in the pulmonary vasculature, attributes the increases in right sided and pulmonary vascular pressures to mechanical obstruction as well as reactive vasoconstriction.^{20,21}

DECREASED CL (DECREASED LUNG COMPLIANCE)

This seems to be directly proportional to the total amount of injected air and results from increased RAW and pulmonary edema^{20,17,22}

OBSTRUCTION OF PULMONARY BLOOD FLOW

This is the major fatal consequence of venous air embolism. Mechanical obstruction²³ (by air bubbles or air lock) and reactive vasoconstriction are the predominant mechanisms responsible for obstruction. The relative importance of each, however, is debated.

There is little doubt that venous air accumulates in the right heart, but whether the "air lock" phenomenon (an air bubble sealing out venous return) actually takes place or has any clinical significance is contested.^{22,24} Intracardiac air has been reported and occasionally demonstrated radiographically.^{9,13,25,26} This is usually a poor prognostic sign⁸ but whether the intracardiac air caused an air lock – no flow phenomenon^{8,11} or was merely evidence of a pulmonary arterial vasculature already filled with air and now backing up into the right heart²⁷ is still debated.

Berglund et al²⁰ postulated that if the right heart and pulmonary artery pressures rose strictly on the basis of mechanical obstruction then balloon occlusion of the right pulmonary artery and unilateral air embolization

should produce similar changes in cardiovascular parameters. His results, summarized in Table III, suggest that vasoconstriction plays a very significant role in pulmonary vessel obstruction after air embolism.

Pulmonary angiography after experimental unilateral air embolization showed corkscrewing, rapid tapering and delayed emptying of contrast from the pulmonary arterial vessels of the unembolized lung. These are radiographic findings of vasoconstriction and could not be prevented by pretreatment with beta blockers, vagotomy, atropine, antihistamines or anti-serotonins.¹⁶ Propranolol partially blocked vasoconstriction in some animals. To date, there is no published data available on the effects of antiprostaglandins or calcium channel blockers on vasoconstriction due to air embolization.

PARADOXICAL EMBOLIZATION

Venous air embolizing the arterial system through a patent foramen ovale has been described in the literature.^{16,28,29} It is estimated that at least 20% of the population has a potentially patent foramen ovale^{30,31} which has proven to be the portal of entry for venous air into the arterial system during cardiopulmonary bypass surgery when mean PA pressures have exceeded 29 mm Hg.³² However, paradoxical arterial air embolism has been documented in the absence of a patent foramen ovale when air (more than 30 cc) was injected as a bolus in animals pretreated with aminophylline. Pulmonary arteriovenous anastomoses are presumed to be responsible for arterial access.¹⁶

HYPOXEMIA

V/Q (ventilation/perfusion) mismatching is responsible for hypoxemia as physiologic dead space is increased by the ventilation of nonperfused areas.^{33,20}

ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)

ARDS has been reported infrequently following air embolism.^{11,34} The mechanism by which this occurs is unclear but capillary damage by fibrin microemboli, regional pulmonary ischemia and high regional pressures in unobstructed pulmonary vessels have all been implicated.³⁴

Clinical presentation

[The clinical manifestations are summarized in Table IV.]

As soon as the pulmonary vasculature is assaulted by air, a sudden and dramatic constellation of signs and symptoms develops. There is air hunger, laboured respirations, cough and chest pain.¹¹ There may also be tachypnea, tachycardia, hypotension, cyanosis and the characteristic millwheel heart murmur. Shivpuri et al³⁵ showed experimentally the characteristic heart sounds preceded the millwheel murmur. The heart sounds first became metallic and resonant in character, taking on a drum-like quality (drum sign) considered pathognomonic of air embolism. The heart sounds then became

TABLE III: Pulmonary vasoconstriction with venous air embolism

| | OCC RPA* | VAE† | |
|-------------------------------|----------|-------|-------|
| | | UNIAT | BILAT |
| Pulmonary artery pressure | 8% | 116% | 110% |
| Pulmonary artery flow | 17% | 27% | 23% |
| Pulmonary vascular resistance | 19% | 215% | 233% |
| Aortic pressure | — | 14% | 20% |

Legend: *occluded right pulmonary artery
†venous air embolism

When compared to mechanical obstruction of the right pulmonary artery (occ. RPA) both unilateral and bilateral venous air embolization produced significantly more pulmonary vasoconstriction leading to a marked decrease in pulmonary flow and aortic pressure.

louder, more rhythmic and finally a systolic churning millwheel murmur was heard.

In a study of 14 patients with venous air embolism Coppa et al³⁶ found that all (100%) patients developed respiratory difficulty. Thirty-five percent became apneic initially and of these 60% died. Tachypnea and cyanosis were present in 35% and 64% showed early neurological deficits as manifested by coma, hemiplegia, or dysphasia.

Electrocardiographic patterns in air embolism tend to be nonspecific and thus nondiagnostic. Sinus tachycardia, myocardial ischemia and acute right ventricular strain have been documented. Asystole has been reported infrequently; VPB's and bradycardia occur more commonly.³⁷

Arterial blood gases may show hypoxemia and an elevated pCO₂ but are otherwise nondiagnostic.¹⁶

Chest x-ray may reveal air in the main pulmonary artery (diagnostic) though this is often pre-terminal. Other less specific radiological findings include a normal CXR, pulmonary edema, focal oligemia, atelectasis and enlarged central pulmonary arteries.⁸

Diagnosis

RW Buckland et al³⁸ and others⁵ have compared various detection modalities in neurosurgical patients. A group of 17 patients was extensively monitored by Doppler, end tidal CO₂ analysis, central venous pressure catheter (plus or minus aspiration of air), ECG, BP, and esophageal stethoscope. In summary they found that:

- 1 Blood pressure – was unreliable in that hypotension was a late sign indicative of cardiovascular compromise and substantial air entry,
- 2 ECG – was also unreliable showing abnormalities in only 40% of patients with intravascular air. In addition, abnormalities were nonspecific (i.e. VPB's),
- 3 Heart sounds – monitored for the classic millwheel murmur were disappointing. Characteristic heart sounds were heard only transiently in 10% of cases and required injection of large amounts of air in canine studies before they could be appreciated.
- 4 A rise in CVP – was useful in detecting 40% of cases and having a catheter in situ allowed aspiration of air

in an additional 20% of cases. Again, however, rises in CVP were considered a late sign.

- 5 A decrease in end tidal CO_2 – indicating an increase in dead space with V/Q mismatch was positive in only 50% of cases. Again, only when the volume of air was sufficient to significantly compromise pulmonary perfusion did the CO_2 rise. The measurement of end tidal CO_2 was quite an expensive and elaborate set-up with practical application limited to the invasively monitored neurosurgical patient.
- 6 Doppler ultrasonic flow detector – was by far the most sensitive method of detecting air and was used as the “gold standard” in these papers. It, however, lacks ability to quantitate and thus has little clinical correlation.

MS Albin et al³⁹ using technitium macroaggregated albumin tracer, showed that when air enters the venous system rapidly in moderate amounts (bolus) there is a tendency to develop segmental lesions that mimic pulmonary thromboemboli. In contrast, air entering over a longer period of time (even in large amounts) produced a pathognomonic decortication pattern manifested as a scalloping of the cortical lung on the scan.

Prevention

Prevention merits discussion before treatment since attention to the former may obviate the need for the latter.

The key to prevention is an awareness of the situations and circumstances that will predispose the patient to air embolism. Being aware of this potential complication when inserting a central venous catheter and taking the appropriate preventative measures (Trendelenberg, asking the patient to hold breath or valsalva, occluding the exposed hub of the needle, firmly connecting IV tubing plus or minus taping) will greatly reduce the potential for any air embolism. Recognizing that volume contracted, tachypneic patients are at increased risk and taking the proper precautions will ensure a minimal incidence.

Treatment

Factors influencing the outcome of the patient include the amount of air, rate of entry, paradoxical embolization and the patient's age and pre-existing medical condition. It is generally accepted that the initial steps in managing the victim of a venous air embolism should include halting any continued entry of air into the venous system and the administration of oxygen.^{8,11,16,40} The application of oxygen helps in the denitrogenation of the air embolism thus reducing its size and facilitating its resorption. It also ensures maximal oxygenation of ischemic tissues.

The next most widely advocated manoeuvre is to place the patient in the left lateral decubitus position (left side down) in Trendelenberg.^{8,11,16,40} Durant et al⁴ showed increased survival of dogs following air embolism when managed in the left lateral decubitus position and this has been substantiated by subsequent experimental and clinical trials.⁸ This position is postu-

lated to clear the right ventricular outflow tract of air and avoid any “air lock” phenomenon by allowing the air to displace itself into the more superior right ventricle thus clearing the outflow tract which has assumed a more inferior position. The advantage of Trendelenberg seems somewhat more tenuous. Reportedly, it facilitates displacement of air from the outflow tract.⁴¹ However, one must exercise discretion in its use if the suspected portal of entry is below the diaphragm where Trendelenberg might encourage further entry of air.

The patient should have blood pressure and electrocardiographic monitoring. If a central venous line is in place, aspiration should be attempted. This has proven to be an unreliable method of retrieving air or improving the clinical condition of the patient.^{23,30,42} Animal experiments using dogs have shown little effect on survival when adding central venous aspiration to other treatment modalities, though resuscitative time was decreased.⁴³ Debate exists over the type of catheter most efficacious in retrieving air. It remains inconclusive whether central venous, Swan Ganz or multiple lumen catheters provide significantly different results.^{27,44}

A deterioration in vital signs such that vital organ oxygenation or perfusion are compromised should be treated by prompt cardiopulmonary resuscitation.⁸ Though there is no evidence that CPR is of any specific therapeutic benefit in venous air embolism there is little recourse in managing the patient in cardiopulmonary arrest. Any patient who exhibits evidence of central nervous system insult or dramatic myocardial ischemia should alert the physician to the possibility of paradoxical embolism. Consideration should be given to hyperbaric therapy.^{8,11,45,46} Treatment with heparin, oxygen and steroids may be beneficial in arterial embolism but hyperbaria is the only specific therapy.⁴⁶ Hyperbaric therapy may be life saving even after a delay of several hours.^{47,48}

Conclusion

Where does all this leave the emergency physician? Detection of the inevitable minute amount of air introduced in many emergency procedures is of little concern. From a practical standpoint, the emergency physician must first have a high index of suspicion. Secondly, he must monitor those parameters which signify and corroborate clinically significant air embolism. In addition to the clinical scenario, a drop in blood pressure, rise in CVP and ECG abnormalities continue to be the most pragmatic indicators of significant air embolism.

Today's emergency physician is likely to be more aggressive and invasive in the early and definitive management of the ill patient in his department. As a result, the potential for venous air embolism increases. It is paramount therefore that he be acutely aware of the potential complications of his procedures, their prevention, and if need be, their management.

The classical clinical presentation of venous air embolism in a patient who has been found with a

TABLE IV: Clinical manifestations of venous air embolism

| | |
|---------------------------------------|------------------|
| PULMONARY: Shortness of breath | Air hunger |
| Tachypnea | Cough |
| Cyanosis | Wheeze |
| Apnea | |
| CARDIOVASCULAR: Tachycardia | Millwheel murmur |
| Hypotension | |
| Chest pain | |
| Cardiopulmonary arrest | |
| NEUROLOGIC: Focal effects – dysphasia | |
| – hemiplegia | |
| Global effects – coma | |
| – seizure | |
| – confusion | |

TABLE V: Management of venous air embolism (VAE)

Diagnosis of VAE

- 1 Halt further entry of air
- 2 100% oxygen
- 3 Left lateral decubitus position plus/minus Trendelenberg
- 4 BP & ECG monitoring
- 5 Aspiration through central line if in place
- 6 CPR as indicated by vital signs
- 7 Consider hyperbaric therapy if:
 - a) persistent neurological deficits
 - b) marked myocardial ischemia

disconnected CVP line or who develops the constellation of signs and symptoms while having a central line inserted should not pose a great diagnostic dilemma. The astute emergency physician must maintain a high index of suspicion in order to diagnose air embolism in less conventional scenarios.

The pathophysiology of venous air embolism has been discussed in detail, in the hope of arriving at a set of rational management principles. Though various pharmaceuticals have been used experimentally in managing specific pathophysiological manifestations of air embolism; their use cannot be advocated at this time. A more conventional management protocol is outlined in Table V.

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PRECAUTIONS: *Head injury and increased intracranial pressure:* The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing elevated intracranial pressure. Furthermore, narcotics may produce adverse reactions which can obscure the clinical course of patients with head injuries.

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Special risk patients: PERCOCET should be given with caution to certain

patients such as the elderly or debilitated, because of the danger of cardiac or respiratory depression, as well as to those patients with hemorrhage, severe impairment of hepatic, respiratory or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.

Headache: Because headache often involves a significant psychological component, a narcotic analgesic should only be employed for the treatment of headache when no other treatment is effective, in order to minimize the risk of psychological and physical dependence.

Drug interactions: The CNS depressant effects of PERCOCET may be additive with those of other CNS depressants. See WARNINGS.

Other: Patients should be instructed to store PERCOCET, as for any medication, safely out of the reach of children.

ADVERSE REACTIONS: The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in non-ambulatory patients, and some of these adverse reactions may be alleviated if the patient lies down.

Other adverse reactions include euphoria, dysphoria, constipation and pruritus.

SYMPTOMS AND TREATMENT OF OVERDOSAGE: *Signs and symptoms:* Serious overdose with PERCOCET is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdose, apnea, circulatory collapse, cardiac arrest and death may occur. The ingestion of very large amounts of PERCOCET may, in addition, result in acute acetaminophen intoxication, characterized by anorexia, nausea, vomiting and sweating within two or three hours of ingestion, and possibly cyanosis with methemoglobinemia. Within 48 hours, liver function tests rise abnormally, and the liver becomes enlarged and tender. Within three to five days jaundice, coagulation defects, myocardiopathy, encephalopathy, and renal failure occur, followed by death due to hepatic necrosis. The ingestion of 10 g of acetaminophen is considered to result in intoxication, with the possibility of a fatal outcome if the amount exceeds 15 g. Hepatotoxicity occurs when plasma levels of 300 µg/ml are observed within four hours of ingestion.

Treatment: Primary attention should be given to the re-establishment of adequate respiratory exchange through provision of a patent airway and the institution of assisted or controlled ventilation. The narcotic antagonist naloxone is a specific antidote against the respiratory depression which may result from overdose or unusual sensitivity to narcotics, including

oxycodone. Therefore, an appropriate dose of this antagonist should be administered, preferably by the intravenous route, simultaneously with efforts at respiratory resuscitation. Since the duration of action of oxycodone may exceed that of the antagonist, the patient should be kept under continued surveillance and repeated doses of the antagonist should be administered as needed to maintain adequate respiration. The instructions contained in the package insert provided by the manufacturer should be carefully observed.

An antagonist should not be administered in the absence of clinically significant respiratory or cardiovascular depression.

Oxygen, intravenous fluids, vasopressors and other supportive measures should be employed as indicated.

Gastric emptying by emesis or lavage may be useful in removing unabsorbed drug, and should be carried out at an early stage of treatment. Plasma levels of acetaminophen should be determined. If hemodialysis is carried out within ten hours of ingestion, it may be of some value.

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Pre-hospital care in Quebec

M-M BOUCHER MD

Historical considerations

Before 1971, the date of the implementation of the health insurance system in Quebec, out of hospital emergencies were treated in a most non-uniform approach. For emergencies in the home, the family doctor would be called. Transport to the hospital could be done by private ambulance at the request of the physician. The patient would be billed by both the doctor and the ambulance, so poor (and sometimes not so poor) people would tend to ask the doctor only for extreme situations and preferably be treated in their home without the use of ambulances or hospitals. For public place emergencies, the police would usually be called and they would transport to a hospital, free of charge, the ill or injured person in a police ambulance or patrol car. In Montreal the police have had ambulances in growing numbers (up to 39) since the late 1950's; their services were fast (within 4-5 minutes) and appreciated by the public, but no systematic training was ever given to the policemen who manned these ill-equipped vehicles. "Load and go" was the rule. A very small number of hospitals had their own ambulance fleet as a marginal operation. In 1971, the government of Quebec instituted *total gratuity* for medical services in this province.

There was an immediate and major increase in the demand for medical care and doctors, who suddenly could have waiting lists of 3 to 5 weeks in their offices became almost totally unavailable for house calls. People started using the hospital emergency departments for acute illness treatment. In the late '70s, two changes in government policy influenced pre-hospital care in a major way. First, the fee a doctor could receive for an emergency out of hospital call was more than doubled; second, emergency ambulance transport for people over 65, welfare recipients, workers involved in accidents and road accident victims was made free. These government decisions, meant to improve access to emergency hospital and out of hospital care, had the effect of seeing the number of ambulance transports by private companies largely increased. Some doctors formed private organizations to answer home emergencies. These private companies, owned sometimes in part by doctors, had a 24 hour answering service, and a dispatch module. Doctors in cars, or ambulances, or both could be sent according to the need expressed by the caller. All the elements were present to lead people into abuse and it happened. These organizations grew and flourished to the benefit of doctors, ambulance companies and people who could not, or did not have the services of a family doctor, from 1977 to 1981. The cost for the public treasury was getting indecently high, five times more in 1980 than in 1976. Meanwhile, because of numerous factors involving an aging population, and a change in family values and structure, hospitals were getting half full of long term patients, and emergency wards full to the point of having patients stay for weeks in observation sections, on stretchers. Some hospitals *simply closed* their emergency wards for a few days every week. Something had to be done and the solution adopted in December 1981, after two years of prepara-

tion, was to implement a public EMS system for greater Montreal (population 2,000,000 approx.).

There were three objectives for that new organization: first, coordination of the admission of patients to hospitals to prevent unjustified and eventually to eliminate, emergency room closures. Second, coordination of the ambulance transport system to make sure the personnel were qualified, the vehicles well equipped and the coverage for emergencies was adequate at all times. (The private companies tended to put ill-trained and ill-paid attendants on as few ill-equipped vehicles as possible, hoping to have them make as many possible calls per shift. Response time could easily reach 20 minutes.) The third goal was to rationalize the out of hospital "emergency" visits by the doctors as three out of four were non-emergency cases. In fact, by that third step alone, enough would be saved by the province to more than cover the cost of the two first objectives. So, in December 1981, "Urgences santé", the health emergencies coordination center, was born.

Urgences santé

The EMS system in Montreal (operating under the authority of the Montreal regional council of health and social services) consists of:

1) COORDINATION OF HOSPITAL EMERGENCY ADMISSIONS

24 hours a day, seven days a week, Urgences santé receives information on the situation of the 29 hospitals who operate an emergency room in Montreal. The number of available emergency observation beds, intensive care beds, and medical or surgical department beds is constantly monitored and decisions can be made to redirect the flow of ambulance traffic from one or more hospitals to one or more others according to the changes in the situation. A coordinator (often a doctor) is named at the hospital level and is responsible for the application of strict admission and discharge policies, collection of data and deciding with "Urgences santé" what solution to try and for how long. (Example: emergency "closed" to ambulances, except for trauma or "open" to ambulances except for cardiac "monitoring cases", etc.)

2) AMBULANCE TRANSPORT

About 90 vehicles owned manned and equipped by five private companies constitute the fleet of ambulances. Up to about 50 can be on the road at the same time, never less than about 15. The personnel assigned to these vehicles have to answer to the standards of training that "Urgences santé" determines. Calls for ambulances come from public places, private homes or hospitals. They are all received at the Urgences santé answering module, evaluated for priority triage and appropriateness of demand (people can be refused an ambulance and referred to their own doctor or clinic), and, after the call has been evaluated, the dispatch module sends the ambulance it feels is the more likely to give an adequate response time. No ambulance can be on the road and/or go to a call unless dispatched by

Urgences santé. Incidentally, the police ambulance system has become unnecessary and obsolete and should be phased out by mid 1984. About 500 transports a day are made, half of those from hospital to hospital, the other half for out of hospital to hospital emergencies.

3) RATIONALIZATION OF MEDICAL OUT OF HOSPITAL EMERGENCY CALLS

At Urgences santé 160 doctors share a 39 x 8 hour shifts per day schedule. They work in a passenger car, with a driver-technician who has the same training as the ambulance attendants. Up to 18 doctor-cars can be on the road at the same time, the cars, the driver and the equipment being rented by Urgences santé from private companies. Calls for doctors can come from private homes or public places to a central Urgences santé phone line where they are triaged for appropriateness and priority. The nurses who answer can thus decide according to the need expressed to send a doctor, an ambulance or both. They can also decide to refer a person to his own doctor, clinic or hospital by his own means if the situation doesn't appear to be urgent. Private doctor companies have been outlawed and of 1000 requests a day, around 200 receive urgent medical intervention on site, compared to 1000 a day before Urgences santé existed. The doctors are paid by the hour, with no relationship to the number of calls they get (average 5 or 6 per 8 hour shift). Telephone triage by the nurses is constantly supervised by one or two doctors 24 hours a day, and the "decision trees" the nurses use are revised by a "medical committee" of seven emergency physicians.

4) QUALIFICATION OF PERSONNEL

a) *Ambulance attendants and driver-technicians* have a minimum 160 hours training including basic CPR. Of course, with that of EMT-1 level, they are not authorized to install IVs, give medication or intubate patients. This defines the need for doctors, in the present context, to achieve proper pre-hospital treatment and stabilization of major emergencies.

b) Nurses

answer all the calls for triage. All are licensed nurses with emergency room or intensive care experience and three weeks telephone triage training.

c) Doctors

are all ACLS providers with recent hospital emergency room post graduate experience (minimum 500 hours). Most are GPs with emergency room experience. Some are residents in internal medicine, surgery, family practice or emergency medicine. They are nominated according to their credentials by a medical committee. This medical committee composed of seven emergency physicians is the supreme medical authority in the system. It determines what the hiring criteria for doctors will be, the scheduling, the discipline, standards of care, and what equipment will be put on the vehicles. The Regional Council is responsible for the administrative aspects of the system.

5) RECENT EVOLUTION AND PLANNED CHANGES

a) *Coordination of the admissions through the emergency rooms*

In the first two years of Urgences santé existence, this has been the most frustrating aspect. Even though, the constant redirectioning of the ambulance traffic helps, there is still a great need for chronic care beds in the Montreal area in order to be able to "free" the short stay beds so occupied by long term patients. Until some major changes are made at a higher level, the emergency wards will continue to operate with a high input and a trickle of an output.

b) *Coordination of the ambulance transport*

During the first two years a great deal has been accomplished in this field. Every attendant on the road now has the minimal training. Vehicles are well equipped. Response times are good with an average of 4 minutes for emergencies. No communication is allowed except through the Urgences santé dispatch. The vehicles are now paid for "stand-by" time and a bonus on each call. The near future will see raised training requirements, more sophisticated equipment on the ambulances, better radiocommunication equipment (dispatch unit) and better scheduling of the hospital to hospital transports.

c) *Medical coordination*

The medical team has properly trained all its members in ACLS and about 25% in ATLS also. A continued medical education committee has been instituted and is planning regular pre-hospital care emergency seminars. A permanent committee on the revision of decision trees, equipment, and medical audit are also in operation. The next few months should see the ATLS certification of the remaining group, a scientific testing of the triage decision trees and a more coherent articulation between the medical teams working on the road and in the emergency rooms. It should be mentioned that since the beginning of Urgences-santé, compliance to decision trees by the triage nurses has been a problem. Under these circumstances, risk evaluation of the calls is poorly standardized. Finally, disaster planning is part of the projects that will be receiving full attention.

The rest of Quebec

Outside of Urgences santé in Montreal, there is no public EMS system to coordinate pre-hospital care. In most small cities, there are a couple of ambulances privately owned and paid by the transport, annually inspected by the government. In a few larger cities, there are private doctor-companies operating legally (they have been outlawed in Montreal only) and billing the government by the visit. In my opinion the more remote the area, the better trained the EMT's should be. As for the future of pre-hospital care in Quebec, a lot will depend on the success or failure of the new Montreal public EMS system. Meanwhile, emergency physicians are getting more involved in their communities and therefore the pre-hospital part of emergency

care. Until higher levels of government really push for major provincial changes, local teams of firemen, policemen, doctors and ambulance attendants are and will be important factors in increasing the quality of pre-hospital care in Quebec.

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Activated charcoal: black magic Q4H

M TENENBEIN MD, FRCP(C), ABMT

Activated charcoal has been called the single most effective agent in the therapy of acute poisoning. This article will review its use for acute overdosage and will describe a new approach – multiple dose charcoal.

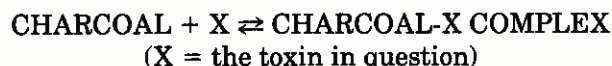
Charcoal was used as a medicinal agent in ancient times. Its use as an antidote for poisoning dates back to nineteenth century Europe where most of the work demonstrating its efficacy was done. As a matter of fact, its use in North America has only been advocated over the past twenty years.

Charcoal is manufactured by the pyrolysis of carbonaceous substances at temperatures of 600–900°C. It is activated by exposure to oxidizing gases (usually steam) at similar temperatures. This process erodes the surface of the charcoal particles creating an internal network of pores and a very large adsorptive surface area. The final step in the manufacture of medicinal activated charcoal usually involves washing with acid to remove some of the inorganic constituents.

The actual adsorptive process occurs within seconds after the charcoal comes in contact with the substance in question. Various factors influence the extent of adsorption. These include the temperature of the milieu, the solvent for the reaction (gastrointestinal fluids), the surface area of the charcoal and its spore structure. However, in the clinical situation these factors are relatively constant. The latter two are controlled by using finely powdered, medicinal activated charcoals.

Two factors that do vary from patient to patient are the chemical nature of the toxin and the presence of competing solutes. Most organic substances are well adsorbed with the degree of adsorption being inversely related to their polarities. Adsorption of inorganic compounds is dependent upon the degree of ionization. Neutral compounds are usually well adsorbed whereas ionized forms are not. The presence of other solutes influences the extent of adsorption by competing for the charcoal binding sites. In clinical situations these substances include many of the constituents of gastrointestinal fluids, food and the various products of digestion. This interference is overcome by administering a large dose of activated charcoal.

Although the process of adsorption to charcoal is potentially reversible and is described by the equation



the equilibrium state for most substances strongly favours adsorption. In clinical practice, the large dose of charcoal administered to the patient also shifts this reaction to the right. In most situations, if desorption occurs within the gut, it does so to a degree that will not influence the outcome of the patient.

Most reviews of this topic include a long list of substances adsorbed by activated charcoal. It is easier to list (and therefore remember) those that are not. These are the inorganic compounds that dissociate in vivo into ionized components. Examples include strong acids or bases, iron salts and cyanide. Therefore, charcoal therapy is not indicated in these situations.

Clinically, charcoal should be administered as soon as possible after the overdose. If syrup of ipecac has been used, one must wait until vomiting has ceased before giving the charcoal. As much as possible should be given to overcome the effect of competition for charcoal binding sites by other solutes and to inhibit desorption of the toxin. The common recommendation of administering charcoal at a dose of ten times the amount of the ingested toxin is not practical because the latter information is rarely known. Reasonable guidelines would be 25–50 grams for children less than five years old and 50–100 grams for older children and adults. It should be administered as an aqueous slurry (approximately one part charcoal to four parts water). An ionic cathartic such as magnesium sulfate should also be given. Charcoal tends to constipate and without the aid of a cathartic it may take on the consistency of briquets.

Multiple dose charcoal

Recent research has shown that charcoal, in addition to preventing the absorption of toxic substances into the body, can also increase the rate of excretion of some of these substances out of the body. In order to accomplish this, it must be given in several doses a few hours apart throughout the course of the intoxication.

Multiple dose charcoal has been shown to decrease the half-lives of phenobarbital, theophylline, nortriptyline and dapsone. Various mechanisms have been proposed to account for these observations including the interruption of enterohepatic and enterogastric circulations and "enteral dialysis". For any individual toxin, more than one of these mechanisms may be operative.

Some drugs are excreted into the bile in their active form or as glucuronides which can be hydrolyzed releasing free drug. The free drug can then be reabsorbed. Also, drugs that are weak bases (such as tricyclic antidepressants) may diffuse from gastric mucosal capillaries into the acidic stomach contents. This is a form of ion trapping analogous to alkalinizing the urine to enhance salicylate excretion. However, upon reaching the neutral to mildly alkaline environment of the small intestine, such drugs are no longer "trapped" and are available for reabsorption. For both of these "circulations" drug reabsorption would be interrupted by the administration of repeat doses of activated charcoal.

M Tenenbein MD, FRCP(C), ABMT

From the Manitoba Poison Centre and Children's Hospital Emergency Department, Winnipeg, Manitoba

Probably the most important mechanism contributing to the efficacy of multiple dose charcoal is "enteral dialysis". Most drugs can diffuse in either direction across gut mucosa. Direction of flow is determined by the concentration gradient. Continually administering charcoal lowers the gut concentrations of free drug so that it continues to diffuse from the circulation into the gastrointestinal tract. In effect, the gut epithelium becomes a semipermeable dialysis membrane.

Multiple dose charcoal should be used for significant ingestions of phenobarbital, theophylline and tricyclic antidepressants. It can be instituted immediately even in remote areas as it requires no secondary or tertiary care medical facility support. It may obviate the need for invasive forms of therapy (dialysis or hemoperfusion) or it may be used in addition to these techniques.

Guidelines for dosage are as follows:

- 1) Administer the usual dose of activated charcoal.
 - 2) Subsequently administer one-half of the above dose every four hours.
 - 3) Administer an ionic cathartic such as magnesium sulfate with the initial charcoal dose and every twelve hours thereafter if a stool does not occur.
- Multiple dose charcoal should be stopped when blood levels of the drug (if available and applicable) reach non-toxic levels or when symptoms disappear. Further clinical investigation and experience will probably refine this approach and may expand the list of poisons amenable to this form of therapy.

Chief of Emergency

The Sir Mortimer B. Davis – Jewish General Hospital is presently searching for a full-time Chief of Emergency. The hospital is a 600-bed McGill University teaching hospital. It has a very busy fully departmentalized Emergency Department which sees approximately 52,000 patients a year.

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 Director of Professional Services
 Sir Mortimer B. Davis –
 Jewish General Hospital
 3755 Cote Ste. Catherine Road, Room A-142
 Montreal, Quebec
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Interviews will be held in Calgary, after the Emergency Physicians Board examinations in September.

Please apply quoting competition #AX84-0125, by August 24, 1984, to:

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 T2P 2M5



CITY OF CALGARY

Emergency Medical Training Programs

RCPS

Toronto, Ontario

University of Toronto

Hospitals: Toronto General Hospital, Sunnybrook Medical Centre, Hospital for Sick Children

Length of Program: four (4) years after graduation, three (3) years after rotating internship.

Number of Positions: 3 Residents per year

Accreditation: RCPS (C)

Deadline for Applications: September 30 of each preceding year. However, in order to schedule interviews, applicants are encouraged to submit their applications by September 1 of the preceding year.

Program Director: Bruce MT Rowat, MD, CM, FRCP(C), Director of Emergency Department, Toronto General Hospital, 101 College Street, Toronto, Ontario M5G 1L7

Vancouver, British Columbia

University of British Columbia

Hospitals: Vancouver General, Royal Columbian, B.C. Children's, and Grace Hospitals.

Program Director: V Wood MD, FRCP(C) Director, Residency Training, Emergency Medicine, Department of Surgery, 910 West 10th Ave., 3rd Floor, Vancouver, BC V5Z 4E3

Length of Program: 3 years after rotating internship.

Size: Up to six residents per year (depends upon funding).

Accreditation: Royal College of Physicians and Surgeons (site survey recommended new approval status).

Deadline for Applications: October 1 of each preceding year.

Kingston, Ontario

University: Queen's

Hospitals: Kingston General Hospital, Hotel Dieu Hospital

Program Director: Dr LE Dagnone, Emergency Dept., Hotel Dieu Hospital, Kingston, Ontario, K7L 3H6

Length of Program: 4 yr post-MD or 3 yr post internship

Size: Maximum of four (4) residents per year.

Accreditation: RCPS (pending)

London, Ontario

University of Western Ontario

Hospitals: St Josephs Hospital, Victoria Hospital

Length of Program: Three years—post internship

Size: One resident per year

Accreditation: RCPS (pending)

Program Director: Dr RV Gerace, Department of Emergency Medicine, Victoria Hospital Corporation, 391 South Street, London, Ontario N6A 4G5, phone (519) 432-2352.

Deadline for Application: October 1, 1984.

Montreal, Quebec

University: McGill

Hospital: Royal Victoria Hospital

Program Director: Dr M Dupré, 687 Pine Avenue West, Montreal, Quebec H3A 1A1

Length of Program: 3 years

Prerequisites: At least 1 year mixed or rotating internship

Number of Residents: Accepted per year – 4 residents

Deadline for applications: October 30, 1984

Accreditation: RCPS(C) (pending)

Calgary, Alberta

University of Calgary

Hospital Affiliation: Foothills Hospital, Alberta Children's Hospital

Program Director: RV Johnston, Div. Emergency Medicine, Foothills Hospital, 1403-29th Street N.W., Calgary, Alberta T2N 2T9

Number of residents accepted per year: one

Length of Program: 3 yr post internship

Deadline for application: October 30

Accreditation: RCPS approved

CFPC

Toronto, Ontario

University of Toronto, Department of Emergency Medicine & Family/Community Medicine

Hospitals: Toronto Western, Sunnybrook Medical Centre, Hospital for Sick Children

Program Director: Dr Calvin Gutkin, Toronto Western Hospital, c/o 751 Dundas Street West, Toronto, Ont. M6J 1T9

Length of Program: A three (3) year post MD program, the 1st two (2) years of which meet the requirements of the Department of Family & Community Medicine and a 3rd year structured in Emergency Medicine

Number of positions: three – third year positions available

Accreditation: The Special Certificate of Competence in Emergency Medicine will be awarded to those residents successfully completing the Emergency Certification Examination of the College of Family Physicians of Canada. Deadline for application October 15.

Hamilton, Ontario

University: McMaster University

Hospitals: Chedoke/McMaster Hospitals, St Joseph's Hospital, Hamilton Civic Hospitals, affiliated to the Department of Family Medicine.

Program Director: Dr David Maxwell, McMaster Hospital Emergency Department, 1200 Main Street West, Hamilton, Ontario L8N 3Z5

Length of Program: 3 year post MD integrated program with both Family Medicine and Emergency Medicine, or free-standing 3rd year post CCFP. Candidates may enter at 1st, 2nd, or 3rd post-graduate year levels.

Accreditation: CFPC (pending)

Deadline for applications: November 30.

Ottawa, Ontario

University: Ottawa

Hospitals: Ottawa Civic Hospital, Ottawa General Hospital, Children's Hospital of Eastern Ontario

Program Director: Dr AF Henry, Chief, Emergency Dept, Ottawa Civic Hospital, 1053 Carling Avenue, Ottawa, Ontario K1Y 4E9

Length of Program: 3 years post MD, first two years as a trainee in the Family Medicine Program, leading to CCFP and third year in Emergency Medicine. The third year is also open to practising physicians.

Size: 4 residents per year

Accreditation: provided by CFPC. Trainees eligible to write Certificate of Emergency Medicine exam of CFPC.

Deadline for Applications: September 30.

Calgary, Alberta

University of Calgary

Hospital Affiliation: Foothills Hospital, Alberta Children's Hospital

Program Director: Dr. Robert V. Johnston, Div. Emergency Medicine, Foothills Hospital, 1403-29th NW Calgary, Alberta T2N 2T9

Length of Program: one year post CCFP

Number of residents per year: two

Deadline for applications: October 30.

Bookshelf

Pre-Hospital Emergency Pharmacology. Bryan E Bledsoe, Gideon Bosker MD, and Frank J Papa, DO; Robert J Brady Co, Bowie, Maryland, 1984, 260 pages.

E.M.S. Libraries have been lacking a useful pharmacology reference text until now. This book will fill that role, but not without some unfortunate weaknesses.

The book is sensibly organized, with the paramedic student in mind. After an introductory section outlining some relevant terms, definitions, legislation (American) etc., a second section follows with all the "how-to's" of different routes of administration. The third outlines drug dosage calculation. The remainder of the text focuses on specific drugs. These are organized according to their clinical uses, rather than their pharmacological classification – "Respiratory Emergencies, Cardiovascular Disorders, Ob-Gyn etc." This allows for quick and easy reference for paramedics on a case-by-case basis. It also makes some sense as a primary text – paramedics are highly skilled clinical technicians, and should think of drugs in terms of their clinical context rather than their chemical derivation.

Following the main text is an extensive series of appendices. These too are thoughtfully presented – paediatric dosage calculations, quick synopsis of all emergency drugs and fluids in point form, extensive listing of commonly prescribed medications patients might have at home etc.

A consistent problem in many paramedic texts to date relates to an "ambiguity of focus", and this book is no different. The authors have not clarified in their own minds the objectives of the text, and the depth of knowledge they expect in the readership. An example is seen in the discussion of "Racemic Epinephrine" where an attempt is made to introduce the concept of optical isomers. Other than stating that "racemic epinephrine is an equal mixture of both the d and l forms, which together are optically inactive", no rationale is provided to the discussion. Why bother? Who can sincerely believe that paramedics need to understand that?

A second very disappointing and annoying problem is the continual interchanging of brand names and generic names for medications. This is consistently done throughout the discussion of many medications, and no clear justification can be seen. The field is confusing enough without expecting students to remember 2 or more names for each medication.

Finally, again perhaps relating to the "focus" problem alluded to above, the discussion of many drugs is quite inadequate. Paramedics receive their orders either directly by radio or indirectly via protocol. Their understanding of the indications for the precise dosage of medications need not therefore be very extensive. However, they must have a very precise understanding of both the contra-indications and the adverse effects to be anticipated. The discussion of nitrous oxide inhalation analgesia (listed as and discussed only Nitronox®) lists the only contra-indication as head injury, and the only precaution as nausea and vomiting. Paramedics

must understand that because N_2O comes out of solution so much easier than CO_2 , its use in patients with pneumothorax and/or bowel obstruction can be catastrophic. This omission is inexcusable. Similarly, hypotension is not included as a "precaution" with nitroglycerine, nor are seizures with lidocaine – the list goes on. This is unfortunate, because a more complete discussion of adverse effects and complications would not have substantially lengthened the text.

Despite the aforementioned weaknesses, *Pre-hospital Emergency Pharmacology* is still a useful addition to the E.M.S. Library. Unfortunately, it cannot stand alone as the definitive reference text for paramedics on the topic.

Peter L. Lane MD,
Head, Emergency Physician,
Regional Trauma Unit,
Physician Consultant,
Metropolitan Toronto,
Department Ambulance Services

Emergency in the Streets Nancy L. Caroline MD. Little, Brown and Company, Boston, 1983 603 pages, illustrated, paperback

Dr. Nancy Caroline has had a strong interest in pre-hospital care for many years. She is the author of the National Training Standards for E.M.T. – Paramedics published in 1977 by the United States Department of Transportation. Her latest work is a comprehensive text on paramedicine that can be used not only by paramedics, but by the doctor, nurse, or administrator who deals with pre-hospital emergency medical care.

The first chapter of the book is entitled Role Of The Paramedic and Dr. Caroline's experience and depth of knowledge about the paramedic profession is masterfully outlined. This is the most accurately written description of the paramedic function that I have read to date.

Every chapter begins with a basic review of the topic area to be covered. The author assumes that the reader has already had some training, at least to the E.M.T. level, before advancing to this text. The writing style is easy to comprehend and quite straightforward and she has directed both the quality and quantity of each subject area to the paramedic level.

On page 46, under Patient Assessment, Dr. Caroline tells how to check for the dolls-eyes phenomenon in the unconscious patient. There is probably no justifiable rational for performing this maneuver in the field, the risks are staggering.

General pharmacology is well organized and presented in a comprehensive manner with the exception of the section on how the drugs work. Here the author's writing style digresses to a childish presentation (repeated again in Chapter 6 – Cardiovascular System) that is out of character with the rest of the book. It does however, get the point across.

Missing from the text is a description of the technique of Blind Nasal Tracheal Intubation in the trauma patient. The trauma patient who is still making ventilating efforts can quite readily be nasally intubated blindly with an "endrotrol type" tube without having to risk movement of the C spine. A

detailed description of the indications and methodology would be a welcome addition to any publication that deals with trauma.

Considering the entire text, Dr. Caroline has produced a timely, valuable addition to the library of any pre-hospital care program. It can be used as a primary text book for almost any paramedic program and the author has included good bibliographies at the end of each chapter for further reading.

Ken Murray,
Clinical Co-ordinator,
E.M.A. Program,
Sunnybrook Medical Centre

Noticeboard

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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.