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CAEP REVIEW

Contents

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President's Notebook

Vancouver in springtime! Our Annual Meeting this year is, of course, in Vancouver, April 2–5. Dr. Sheldon Glazer and his group have done a remarkable job planning and organizing the meeting, and by the time you receive your Review, many of you will have attended. I look forward to meeting you all again there.

The past few months have been busy ones for CAEP, and I want to spend a few moments outlining our activities. In early February, the executive met in Hamilton, and a Resident's Committee meeting was held in conjunction. Among the many issues discussed were some general impressions about the past and future directions of CAEP. There appears to be some ambiguity about the future role of CAEP. We have achieved certification now, and many of the "old guard" among the membership and executive feel we have reached our primary goal of the past six years. There is no doubt that our association played a very pivotal role in the process of reaching that goal. But, now our members have new concerns, new goals. In fact, we have many new members, who may not have been involved in the certification struggle Where do we go from here?

From discussions with members and the executive in Hamilton, and from the results of our "Future Directions Survey" published in CAEP Review 5(1), I have drawn a few conclusions. *Education*-undergraduate, postgraduate and continuing education—remains a high priority. CAEP can and will continue to play an important role in advocating standards. In terms of CME, I think that separating our scientific papers sessions from the CME program of our annual meeting will prove wise. As you know, we now have a half-day time slot at the Royal College meeting devoted to scientific papers in Emergency Medicine.

A second issue of some importance on the survey was that of '*image*'. I think there is little we can actively do to specifically enhance the image of Emergency Medicine. To a large extent, that will develop as a consequence of the activities of Emergency Physicians throughout the country. We have undertaken to construct a "CAEP Archives" however, to clearly document the role of CAEP over the past few years.

A third, and I feel very important area of concern is that of *economics*. The time is ripe for the Association to play a more direct and facilitative role in the area of the economics of Emergency Medicine. While the actual fee schedule negotiations must of course take place at the provincial level, the Association can facilitate this. I intend to propose at the Annual Meeting that we strike an "Economics Committee" that would serve as a forum for effective communication and the development of strategies between those involved at the provincial levels. In addition, I hope that as a regular part of our Annual Meetings, we can have a session specifically related to the business and administrative side of Emergency Medicine. This has already been planned for the Vancouver meeting.

The final area of concerns is best expressed as *policy initiatives* — issues such as drinking and driving, standards of care, citizen CPR, etc. It was for this reason that I established the "working groups" last fall, and I will look forward to the results of their activities.

Canada Health Act

On your behalf, I prepared and presented a brief on the Canada Health Act to the Commons Committee reviewing the legislation. The central focus of the brief was that the proposed act excludes Emergency Health Services as an "insured service". Dr. Jan Ahuja and I, in our presentation, emphasized that Emergency Health services in general, and ambulance services in particular should not be subject to deterrent fees or user fees. Surely there is no more essential form of health care.

The federal government's refusal to fund prehospital care in the same manner as it funds other health services underlines the cynical political motivations behind the proposed act. The text of our proposed ammendments is as follows:

SUGGESTED AMENDMENTS

2.1

The Canadian Association of Emergency Physicians recognizes that the focus of the Canada Health Act is that of funding. Therefore, while our concerns regarding the federal government's involvement extend beyond funding, the suggested amendments do not. Specifically, the amendments reflect the principle that access to emergency health care should not be subject to financial deterrence.

2.2

It is suggested that s.2 "Interpretation" of the Act be amended to include the new paragraph:

" 'Emergency health services' means any of the following services provided to patients, when necessary, namely

a) ambulance services, including neces-

sary equipment supplies, communications and transportation

b) basic and advanced pre-hospital care provided by ambulance attendants

c) the delegation of medical acts to pre-hospital workers by base hospital physicians

d) drugs when administered in pre-hospital setting

e) emergency health care provided in the disaster setting, including the necessary equipment

f) interhospital, interprovincial and repatriation ambulance services for emergency patients when necessary. "

2.3

It is suggested that s.2 "Interpretation" be further amended to include emergency health services under the definition of insured health services as follows: " 'insured health services' means hospital services, physician services, emergency health services, and eligible surgical-dental services provided to insured persons,..."

2.4

The Canadian Association of Emergency Physicians notes with surprise and disappointment that the government proposes, pursuant to subsection 22(c) of the Act to specifically exclude "transportation or ambulance services" as per 3(b) of the "Proposed Regulations" regarding Hospital Services Exclusions. This is seen as an unwarranted attack on the emergency patient in need, and CAEP requests its deletion. All prehospital care should be "insured services".

2.5

The simple deletion of the clause noted above in 2.4, while important, would not alone be sufficient to meet the needs of Canadian emergency patients. Many ambulance services are administered by agencies other than hospitals. A clear statement is required in the Act itself, as suggested herein, to ensure that critically ill and injured patients have access to emergency health care.

This was an educational process for ourselves as well as for the Committee. I don't think many of us had really appreciated before that one of the major impediments to the development of comprehensive E.H.S. systems across Canada is that they are specifically excluded from coverage by the federal medicare system. We received, I think, a very positive hearing at the Committee. Whether our amendments will be accepted, however, remains subject to the vicissitudes of Ottawa politics.

Peter L. Lane President

Resident's Corner

As my term as Chairman of the Resident's Committee is coming to a close and with our recent biannual meeting completed, I thought this a good time to summarize some of the efforts of the Resident's committee over the past year.

We have had the opportunity to meet twice this year, once in Toronto at the Scientific Assembly in June, 1983 and again in February, 1984 in Hamilton in conjunction with the C.A.E.P. Executive meeting. Between the two meetings a newsletter was published to keep residents up to date on C.A.E.P. activities.

One of our major successes in the past year is what now appears to be a formal liaison in the near future with C.A.I.R. (Canadian Association of Internes and Residents). We had been seeking an input into C.A.I.R. for a number of years now and things seem to be moving ahead at last. C.A.I.R. being a national body of which all residents are members (except Quebec), has access to many services we could not provide on our own. The first we are trying to explore with C.A.I.R. is a manpower study for Emergency Residents in Canada. They have also offered to us some space in their national newsletter which will be a very useful tool for us to inform and educate other physicians in Canada as to who exactly we are, the new "Emergency Physician".

Another more recent issue discussed as our last meeting was the discrepancies in the various programs regarding things from time and money for conference leave to numbers of practice examinations carried out for benefit of the residents. From dialogue between all the program representatives, the differences appear to be very great ($50./year for conferences in one program and $2000./year in another!). Geographical and economic factors will always dictate some differences but indeed some programs could change their format in some aspects to better the resident's experience. The C.A.E.P. executive on our advise is currently looking into a meeting between all program directors to address this issue.

The Elective Bank created 2 years ago is alive and well but in need of new submissions. It is only as strong as what variety of electives we can offer.

The Trauma Association of Canada is a newly formed society involved with setting standards for the research and care of the trauma victim in Canada. The C.A.E.P. Resident's Committee has already had representation at the inaugural meeting and is encouraging its members to join this very important society.

Finally, many residents have expressed a keen interest in entering a research period or elective. Up to now there has been no funding available via C.A.E.P. for such activities and it was up to the local universities to provide available funds. Now, as of this fall, C.A.E.P. has achieved charitable status and is able to receive donations towards a research fund. We hope this fund will grow quickly as as to aid in the growth of Emergency research in Emergency programs across Canada.

The following represents the highlights of the past year for the C.A.E.P. Resident's Committee. Both myself and the Committee would like to sincerely thank the C.A.E.P. Executive for their ongoing support of our group that we may continue to better residency training in Canada.

Respectfully submitted

Robert Foxford, M.D.
Chairman, Resident's Committee
C.A.E.P.

The Pathophysiological Basis for Appendiceal Pain

by M. A. Cherniwchan M.D.

Question: Recently, in a training examination as part of our residency, the following question was asked:

"What is the pathophysiological basis for the pain experienced in appendicitis?"

As the detailed answer to this question was somewhat difficult to achieve (and required use of disparate references), the following brief summary is presented to aid those in exam preparation.

Answer: Any answer to this question must first point out the following:

1. The anatomy of the sensory innervation to the viscera remains incompletely understood and described.[3,9]
2. Proven appendicitis (on pathology) is associated with many different clinical presentations of abdominal pain.[6,7,5]

However, with these qualifications in mind, an understanding of the embryology, anatomy, and physiology of the normal viscera and abdominal wall allows a rationale under which to subsume many of the disparate findings.

There are basically three types of pain associated with appendicitis[1]:

1. *true visceral* pain
2. *deep somatic* pain
3. *referred* pain.

All are explained in the following review.

Embryology, Anatomy, and Physiology of the Nervous System of the Gut and Abdominal Wall (as applied to the Appendix):

The vermiform appendix is a derivative of the embryological midgut.[4] Its sensory nerve supply consists of *visceral afferent fibers*. Histologically, the fibers are C-type (small, unmylineated, 1–2 μm in diameter, transmitting at 0.5–2.0 m/sec.). No specific pain fiber ending exists in the gut; rather, the sensory end organs consist of a combination of free nerve endings and enteric plexuses (nervous plexuses located in the subserous, myenteric, and submucous locale).[1] The visceral pain system is, overall, less well developed than the corresponding somatic sensory apparatus, in regard to both size and number of fibers.[1]

These visceral pain afferents respond to stretching and distension, strong smooth muscle contraction, inflammation and ischemia.[9] It also appears that the pain threshold is lowered under conditions of ischemia.[1] Stimulation of these receptors produces pain that is 'dull and aching' in quality, of gradual onset, and may be associated with nausea and autonomic symptoms and signs (i.e. sweating). A final pathway mediating the pain response here may be the release of kinins or proteolytic enzymes at the site of injury.[1]

The visceral afferent fibers pass with the sympathetic nervous system chain to the level of the superior mesenteric plexus (overlying the origin of the superior mesenteric artery). After reaching the midline, the pain fibers travel a complex course involving the celiac ganglion, thoracolumbar sympathetic trunks, and the aortic ganglion; eventually entering the spinal chord with the associated SNS fibers (and synapsing in cord substance after travelling cranially or caudally a variable distance).[1] The level of entry of the gut pain fibers into the spinal chord is at T6–L1,2 and the segmental innervation of any particular visceral organ is not known with certainty. Tentatively, the segmental innervation of the appendix is T8.[3] Visceral afferents associated with the PNS also exist; function unknown.[9]

Whatever the exact segmental innervation of the appendix, the visceral pain fibers are involved in a synaptic framework in the cord substance; with modifying input from higher neural centers superimposed. Included in this synaptic framework is, of course, the input from the corresponding segments of abdominal wall as these fibers enter the spinal cord at their respective levels. The result is that pain signals originating from the appendix are interpretated by the brain as arising from the general T8–T10 area of innervation; and this can be *perceived* as a periumbilical or central epigastric discomfort; corresponding to the abdominal wall innervation at the T8–T10 level.[2] Thus the pain may be *referred*.

The parietal peritoneum overlying the appendix in its normal position, however, derives its nerve supply from the abdominal wall[9] and the characteristic sensation associated with its irritation is that of *deep somatic afferent* pain.[1] This pain is perceived as intense and sharp in character, and is well-localized. The fibers once again enter the cord at the level of the corresponding dermatomes, which in the area of the RLQ are the levels T11,12–L1.[3] Again, the possibility of the pain being *referred* to the appropriate superficial dermatome (or myotome, see 'guarding' below) exist.

Appendicitis

In 'simple' appendicitis, the pathological progression of luminal obstruction (possibly associated with hyperperistalsis of the appendiceal musculature), vascular congestion, bacterial invasion, and inflammatory response leads to activation of the *true visceral pain* response (by the mechanism of distension of appendiceal serosa, local ischemia, and mechanical smooth muscle contraction. The result is the 'classic' pain of early appendicitis:

1. poorly characterized, felt deep in the abdomen[1]
2. of gradual onset
3. associated with nausea and autonomic signs (such as sweating)
4. possibly perceived in the periumbilical or epigastric region (in a *referred* manner, as stated above)

This early pain sensation is mediated by the visceral afferent pathway system described, in detail, above.

As the progression to gangrenous appendicitis with perforation and localized peritonitis occurs, the parietal peritoneum becomes irritated, responding with *deep somatic pain*, with the appropriate characteristics and pathways outlined previously. In the context of irritated parietal peritoneum, components of the painful sensation that are *referred* may be manifest in a number of different ways[1]:

1. *hyperalgesia* of the RLQ dermatome
2. *tenderness* of the RLQ superficial abdominal wall to palpation
3. *muscular rigidity or guarding* of the affected area (an 'effector', vs. affector, referral effect)[9]

If the area of parietal peritoneum involved is not RLQ, the corresponding deep somatic and referred pains may be different, yet still appropriate, such as[8] i.e.

position of appendix	possible area of referral
retrocecal	flank/back
pelvic	suprapubic/rectal
retroileal	testicular (irritation of spermatic artery and ureter)
high retrocecal (ie. late pregnancy)	flank/CV

In summary, this approach shows how the various presentations of appendiceal pain can largely be explained by the current understanding of the nervous system to the gut and abdominal wall.

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Meetings to note

Program: Cardiology '84
The 11th Annual Symposium on Advanced Coronary Care
Date: May 28–29, 1984
Location: Royal York Hotel
100 Front Street West
Toronto, Ontario
Canada
Fee: $150.00 nurses, interns, residents
$165.00 physicians
Contact Hours: 12
Sponsor: Humber College of Applied Arts & Technology
Enquiries: Joanne McLeod
Project Coordinator
Professional & Management Development
Humber College, North Campus
205 Humber College Blvd.
Rexdale, Ontario M9W 5L7
(416) 675-5077

Title: Emergency Procedure Workshop
Location: Bellevue Hospital Center, New York City
Course Director: Wayne Longmore, M.D., F.A.C.E.P.
Course Description: A one day, two part practical workshop for physicians, nurse practitioners, physician assistants, nurses and paramedics. Part I – Trauma Center Resuscitation Procedures: cricothyrotomy, chest tube placement, thoracotomy, pacemaker insertion, cut-down, Burr Hole and other emergency procedures. Part II – Minor Surgery Procedures: basic wound care, debridement, suture technique, special procedures "tricks", i.e. removal of fish hooks, foreign bodies, rings, et cetera.
Dates: On-going program, second and fourth Wednesday of each month. Six month schedule: January 11, 25; February 8, 22; March 14, 28; April 11, 25; May 9, 23; June 13, 27.
Fee: Resuscitation Workshop: $200. Minor Surgery Workshop: $100.
CME: 8 hours Category I Credit applied for from ACEP, ACFP, EDNA, NAEMT.
Information: Wayne Longmore, M.D., F.A.C.E.P., Course Director Emergency Care Institute, Bellevue Hospital Center, First Avenue and 27th Street, New York, NY 10016. Telephone: (212) 561-5651.

Interested in teaching, or research in Emergency Medicine? Then plan on attending the

1984 STEM/UAEM MEETING

Where: Louisville, Kentucky
When: May 21–25, 1984
For further info: UAEM
900 W. Ottawa
Lansing, Michigan 48915
Tel.: A/C (517) 485-5484
(STEM = Society of Teachers of Emergency Medicine)
(UAEM = University Association of Emergency Medicine)

Course: *Emergency Medicine 1984*
Dates: June 11–15, 1984
Site: NYU Medical Center, Post-Graduate Medical School, New York City
CME: 29 Category I credit hours – AMA (AAFP, ACEP pending)
Fee: $500
Description: This program provides a rational and systematic analysis of clinical decision making in the emergency department. Instructors emphasize their personal approach to practical management. The case-presentation method will be utilized in all lectures.
Contact: NYU Medical Center Post-Graduate Medical School
550 First Avenue
New York, NY 10016
(212) 340-5295

Ontario Assembly of Emergency Care
Dates: October 1, 2, 3, 1984
Place: Constellation Hotel, Toronto
Sponsoring Organizations: O.M.A., E.N.A.O., & A.C.C.P.
For further Info: Seneca College, Conference Centre, 1000 Finch Ave., E., M3J 2V5. (416) 491-5050

The College of Family Physicians of Canada Certification Examination in Emergency Medicine – 1983 Successful Candidates

Allen, Douglas L., Halifax, N.S.
Allen, Timothy V., Quebec, P.Q.
Beliveau, Peter I., Victoria, B.C.
Casson, Richard I., Baie Verte, Nfld.
Chan, Patrick B., Winnipeg, Manitoba
Crosby, John W., Cambridge, Ontario
Cunningham, John A., Strathroy, Ontario
Dunlop. Anthony G., Williams Lake, B.C.
Dunne, Michel I., Saskatoon, Sask.
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The College of Family Physicians of Canada Certification Examination in Emergency Medicine – 1982 Successful Candidates

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Dispatch Specific Medical Training

Robert V. Johnston, M.D.

Introduction

Until recently, prehospital care in Calgary has consisted of a fire department based, solely advanced life support (ALS) service. The problems involved in such a service were such that a reorganization task force recommended that: (1) the ambulance division be separated from the fire department; (2) the ambulance service would respond in a layered response pattern, i.e. separate advanced and basic life support units. This plan involved a significant alteration of the existing dispatch system which had previously been responsible only for ensuring that the nearest unit was mobilized.[1] A system will be described whereby, without previous medical training, dispatchers will send the appropriate response unit or units (ALS, BLS, Fire first response) with the appropriate priority code.

Introduction

Jusqu'à récemment, les soins pré-hospitaliers à Calgary consistaient en un service de soins avancés en réanimation posté au département d'incendie. Les problèmes à l'intérieur de ce service étaient tels qu'un groupe assigné à sa réorganisation a recommandé: 1) que le service ambulancier soit distinct du département d'incendie; 2) que le service ambulancier fonctionne selon une réponse étagée, i.e. avec des unités distinctes de soins de base et de soins avancés. Ce projet a nécessité une modification importante du système de répartition des appels dont la tâche jusqu'à maintenant avait été de s'assurer que l'unité la plus proche soit mobilisée.[1] On décrit un système où les répartiteurs, n'ayant reçu aucune formation médicale, envoient la ou les unités appropriées (soins de base, avancés, sapeurs pompiers) en utilisant le code de priorité approprié.

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Historical Perspectives

The dispatch centre in an E.M.S. system should act as a nerve centre: a point of access for the general public through a 911 number, a point of dispatch of the appropriate vehicle, a link between vehicles and hospitals, and between vehicles and other agencies. In addition, call screening, response mode assignment and pre-arrival instructions have been recently added to the requirements of a well-functioning E.M.S. communication centre. These additional demands have been met in a variety of ways. In 1974, the Phoenix Fire Department's "Lifeline" program was started as the first organized attempt to provide pre-arrival or self-help instructions to caller prior to the responding team's arrival.[2] In 1977, the Illinois Division of E.M.S. developed a card system whereby callers in need were given general instructions after a series of indexed questions. In 1978, Stockholm introduced a system of triage for a differentiated medical transport service, wherein, after a brief patient history, every call was coded as to priority. Calls difficult to assess were transferred to a physician on duty in a hospital. In addition, pre-arrival instructions could be given by the ambulance team, itself.[3] In 1980, Dallas instituted a call screening program staffed by nurses in addition to the team of dispatchers.[4] In the same year, New York approached the matter somewhat differently by sending an R.N.-staffed triage car to the questionable calls or when prolonged ambulance response times were anticipated.[5] In 1982, Salt Lake City instituted an integrated system of call screening, response mode assignment and pre-arrival instructions. This system has been adopted as the U.S. Department of Transportation standard for dispatch training. This latter program's effectiveness has been described[2] but, due to differences in the system of response patterns, comparisons in Calgary are difficult. In Salt Lake City, paramedic-staffed fire vehicles respond, but do not transport. A private BLS system provides transportation. In Calgary, city paramedic-staffed ambulances are responsible for all functions (response, treatment and transport). In spite of these differences, the Salt Lake City experience suggests a decrease in ALS responses and a rise in BLS responses after implementation of the priority dispatch system. In Dallas, with nurse-performed call screening, ambulances were not dispatched at all in 65% of cases of obviously non-life-threatening calls handled by the nurse.[6] It should be noted that the system to be described in this paper is not one dealing with reducing calls, but rather sending the appropriate unit with the appropriate priority code and providing basic pre-arrival instructions. This program is part of a trend in North America, the common theme of which is apparent from the terms used, e.g. call screening, priority (urgency, severity) screening, no-response screens, selective dispatching of system abuse reduction programs.

Necessity of the Program

The major rationale for the implementation of such a program in Calgary has been mentioned, i.e. the change from a unidimensional ALS response to a layered response (ALS, BLS and Fire first response). The decision not to respond an ALS unit to all calls required that the dispatch centre perform triage, as well as pre-arrival instructions. The pre-arrival instructions consist of primarily basic first aid with the exception of the cardiac arrest situation in which telephone CPR instructions are given. This more aggressive approach to cardiac arrest was necessary because citizen CPR in Calgary is not prevalent. The question of who should perform dispatch triaging and pre-arrival instructions was not an issue in the reorganization of the Calgary system. The presence of a separate union (International Brotherhood of Electrical Workers) whose members have significant seniority and accrued benefits dictated, both economically and occupationally, that current dispatchers would perform these new tasks. A similar situation was faced in Salt Lake City.[7] Since the Calgary dispatchers had little or no medical training or expertise, a protocol system and a tested

Figure 1

ADDITIONAL INFORMATION

DIABETIC PROBLEMS

Diabetes Mellitus (Diabetes) is a condition caused by a lack of insulin (a hormone) in the body. Problems may be caused by either over or under treatment of this disease.

Insulin Reaction – too much insulin, very low blood sugar, rapid onset. Patient usually feels hungry & anxious. Patient may then exhibit bizarre behavior, seizures or may lose consciousness. (This is one of the most common and severe problems of a diabetic – may have appearance of alcohol intoxication with slurred or belligerent speech.)

Diabetic Coma – (gradual onset). Unconscious or decreased level of consciousness secondary to the body's inability to use available blood sugar for fuel when sufficient insulin is not given. Without accurate history, this problem may be difficult to tell from insulin shock.

NOTE: Level of consciousness is the key to determining pre-hospital response selection.

KEY QUESTIONS	PRE-ARRIVAL INSTRUCTIONS
1. Conscious? (able to talk)	a. If unconscious and not breathing normally, SEE CPR CARD.
2. Breathing normally?	b. If unconscious but breathing, SEE UNCONSCIOUS CARD.
3. Seizure?	c. Gather medicine for rescuers.
4. Alert? (aware of surroundings, clear speech)	d. Nothing by mouth.
5. Approximate age of patient?	

DISPATCH PRIORITIES THE CITY OF CALGARY

DETERMINANT	RESPONSE	
Conscious and alert	*Basic Life Support	Code 2
Conscious but not alert	*Advanced Life Support	Code 1
Unconscious (but breathing)	*Advanced Life Support	Code 1

*NO FIRE INVOLVEMENT FOR FIRST RESPONSE

19 DIABETIC PROBLEMS

Table 1

Categorization of Caller Complaints

1. General considerations
2. Abdominal pain/problems
3. Allergies/hives/med reactions
4. Animal bites
5. Assault/stab wounds/GSW
6. Back pain
7. Breathing problems
8. Burns
9. Cardiac/respiratory arrest
10. Chest pain
11. Convulsion/seizures
12. CO poisoning/inhalations
13. Diabetic problems
14. Drowning
15. Electrocutions
16. Eye problems
17. Falls
18. Headaches
19. Heart problems
20. Hemorrhage
21. Multiple complaints
22. Overdose/poisoning/ingestion
23. Pregnancy/childbirth/ miscarriage
24. Specific diagnosis as chief complaint (sick person)
25. Stroke/CVA
26. Traffic injury accident
27. Traumatic injuries, specific
28. Unconsciousness
29. Unknown problem (man down)
30. EMS call back
31. Transfer

training program was necessary. The system developed by Salt Lake City, and modified by Aurora, Colorado, is currently being instituted in Calgary after modification to meet local requirements. Input was sought from members of the medical community, the fire and police departments and the Heart Foundation.

Description of the Priority Dispatch Program

The basis of the program is a flip card file containing 30 sets of 8″ × 5″ cards (Figure I). Each complaint is listed alphabetically as a symptom or incident categorization rather than as a diagnosis (Table I). Additional information is presented on the top of each card; the bottom half of the card is divided into three separate areas. Key questions always include the status of consciousness, breathing, age and limited additional questions which are necessary to determine the priority of response. The second section lists the details of the appropriate response based on answers to the key questions. The response may be any combination of ALS, BLS, Fire first response, either lights and siren or at a more leisurely rate. The third section details the pre-arrival instructions which are meant to complement not replace an ambulance response. These instructions range from simple first aid to complex telephone CPR instructions. The latter pre-arrival instructions were developed and tested by King County (Seattle) Emergency Medical Services Division.[8]

Training

The dispatch training program is approximately 25 hours in length. It consists of an overview of the dispatch priority card system, BLS training, orientation to radio protocols, pre- and post-course written examinations and simulation practice.

The whole training programs was provided by in-house staff augmented by the Salt Lake City program (video tapes, simulation package and examination material). Senior paramedics were given the course and then subsequently acted as teachers. Considerable reluctance by the dispatch group to the overall training program was encountered. This may have arisen for

several reasons: (a) the lack of involvement of dispatchers in the initial revision process; (b) dispatcher concern regarding the perceived threats to their job security; and (c) the lack of direct physician involvement in the training program.

Medical–Legal Implications

Concern has been expressed from a medical–legal point of view with regards two aspects of the priority dispatch system. The first is that of the triage involved. A worst-case scenario can be imagined where an EMT-manned (BLS) ambulance is dispatched without red lights and siren on an apparent nonurgent call only to find that a rapid, tiered BLS, ALS response would have been more appropriate. However, it has been stated that "an upfront, clearly articulated written policy in support of telephone screening of emergency calls would provide a ray of light in an otherwise murky area of heavy potential liability."[9] It is felt that such a "clearly articulated" written policy is found in the form of the card system.

The second issue is that of pre-arrival instructions. These instructions were so formulated that the caller would not endanger either himself, e.g. burns or explosions, or the victim, e.g. inappropriate mobilization of a trauma victim prior to the arrival of the paramedic team. However, more controversial is the issue of the telephone CPR instructions. This issue has been raised by the Aurora, Colorado group when instituting the telephone CPR section of the Dispatch Specific Medical Training Program (legal opinion offered to Aurora Fire Department, 1981). The legal opinion offered was that a person who needs CPR meets the criteria of clinical death, i.e. pulseless and non-breathing. Thus, even if the effort to direct CPR by phone fails, the victim is no worse off than when the dispatcher received the call and offered assistance. The Law Department of the City of Calgary is currently formulating their advice in this matter prior to full institution of the program.

Review and Evaluation

Unfortunately, Calgary may never be in a position to fully evaluate the effectiveness or efficiency of the system for a variety of reasons. First, data does not now exist as to the present effectiveness of the prehospital care system. Second, since the Dispatch Specific Medical Training is only one part of the reorganized Calgary E.M.S. Department, its effect may be difficult to analyze separately. It is contemplated, however, that the system will be evaluated as a whole much more thoroughly than has been done in the past. The dispatch centre will play a critical role in the overall functioning of the E.M.S. Department.

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CAEP Leadership

Continued from page 51

Common Hand Injuries and Infections. A Practical Approach to Early Treatment. by P. R. Carter, W. B. Saunders, Co., 1983, 224 pages, (approx. $63.00).

This text seeks to provide, in a readable and readily referable format, a concise review of both the common and serious hand conditions likely to appear in the Emergency Room.

In short, it does the job admirably and is the best text on this subject the reviewer has seen to date. Though written by an orthopedic surgeon, it is specifically designed for emergency physician use and directs itself largely to those problems that surface time and again in the E.R. setting.

The foundations of good hand care are first laid out. Then functional hand anatomy is covered in a manner that foregoes complicated diagrams or lists of musculature and instead concentrates on the key clinical points required for normal function (along with how to test for them in the emergency setting). Separate chapters are devoted to proper splinting, hand dressing, and edema control. Where applicable, the guidelines for appropriate followup are presented. Further chapters cover joint ligamentous injuries, injection injuries, human "bites", and serious hand infections. Compartment syndromes and the types of emergency patients at risk of these are stressed. All of this is in addition to full coverage of tendon, neurovascular, and bony trauma. A clear and highly practical approach is given for diagnosis of wrist ligamentous instability, in a manner capable of being remembered.

All separate sections are self-contained and laid out in a manner to provide needed information in a hurry. The text is supplemented by one of the best color hand atlases this reviewer has seen (including pages devoted to displaying felon and paronychial drainage procedures). Throughout the chapters, use of diagrams is extensive and much X-ray material is included.

However, some limitations to the text are evident. Pediatric management covers general management and then is limited primarily to a discussion of epiphyseal fractures. X-ray reproduction is less than adequate and reference material is limited to a few chosen works. The opinion of the author in areas of controversy does appear from time to time (i.e. in the use of silver sulfadiazine in outpatient burn management, for example).

These limitations not withstanding, the book remains of immense benefit to emergency physicians at all levels; as a basis reference text, an immediately useable aid to bedside diagnosis and case management, and as resource material for teaching. It rarely remains on the shelf for any length of time in our library and was a joy to read.

M. A. Cherniwchan, M.D.

Toxicology Forum

Hydrocarbon Ingestion: a Not So Slippery Problem

by Milton Tenenbein, M.D., F.R.C.P.(C), A.B.M.T.*

The great hydrocarbon debate leaves many emergency physicians confused. Should stomachs be emptied after the ingestion of a hydrocarbon? The answer is a very clear yes and no. The difficulty is not with the answer but with the question. Such a query is similar to ... "Should an antibiotic be used to treat pneumonia?" The answers to both questions are simpler if the pneumococcus and kerosene are identified as the agents of concern.

Just as we classify infectious agents we should also classify hydrocarbons. This could be done on the basis of various physical or chemical properties. However, from the clinician's point of view, the most useful classification is based upon patient outcome. For hydrocarbons we are concerned with either systemic toxicity or pneumonia – outcomes that may not be mutually exclusive for a specific agent.

The ease with which a hydrocarbon can cause pneumonia is related to its viscosity. Low viscosity products such as kerosene, mineral seal oil and mineral spirits are more likely to be aspirated and cause pneumonia than the negligible risk high viscosity agents such as lubricating oils, greases and waxes. In the past it was postulated that systemic absorption of the hydrocarbon with subsequent hematologic spread to the lungs could cause pneumonia. However there is now a convincing body of experimental evidence that refutes this hypothesis. For this reason, maneuvers directed towards emptying the stomach will not lessen the likelihood of developing pneumonia and if the ingested hydrocarbon will not cause systemic toxicity, they should not be attempted.

Which hydrocarbons are systemic toxicants? That is to say, which hydrocarbons, after systemic absorption can cause specific organ damage and dysfunction. In fact, many hydrocarbons are non-toxic. The simple nonsubstituted linear hydrocarbons (the aliphatics, for those who recall the organic chemistry of their undergraduate years) cause no systemic toxicity.

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These are the chief constituents of gasoline, kerosene and mineral spirits. The hydrocarbons which do cause systemic toxicity are the halogenated (carbon tetrachloride), aromatics (benzene) and camphors. A hydrocarbon containing product may be toxic by virtue of its being a solvent for a toxic solute such as a pesticide or a heavy metal. These should be removed from the stomach to prevent organ damage and/or dysfunction. One must remember that most hydrocarbon products are not pure solutions. In fact the various solutes they contain may be in very low concentrations. Be sure that the absolute amount of specific toxic agent that has been ingested is potentially high enough to cause toxicity before attempting to remove it from the stomach. For instance, gasoline contains approximately 1% benzene, an amount not high enough to cause toxicity in a typical siphoning accident.

How safe are gastric evacuation procedures for the patient who has ingested a hydrocarbon? In the past, they were considered contraindicated in this clinical situation – an opinion still held by some authorities. Does induced emesis or lavage put the patient at increased risk for aspiration pneumonia? Careful review of the literature does not provide an answer to the question. Although aspiration pneumonias are well known to occur in patients who have been lavaged or who have had emesis induced, one cannot be certain whether the aspiration occurred during the initial ingestion or as a result of the gastric evacuation.

If gastric evacuation is decided upon, which technique should be employed? The choice between ipecac induced emesis and gastric lavage has been an unresolved controversy for the past two to three decades. I favour ipecac induced emesis for all patients provided that the usual contraindications of caustic or corrosive ingestant (quite uncommon for hydrocarbons) or of depressed level of consciousness are not present. In general, both procedures are of approximately equal efficacy in terms of percentage of ingestant removed. However, this has not been studied in the context of a hydrocar-

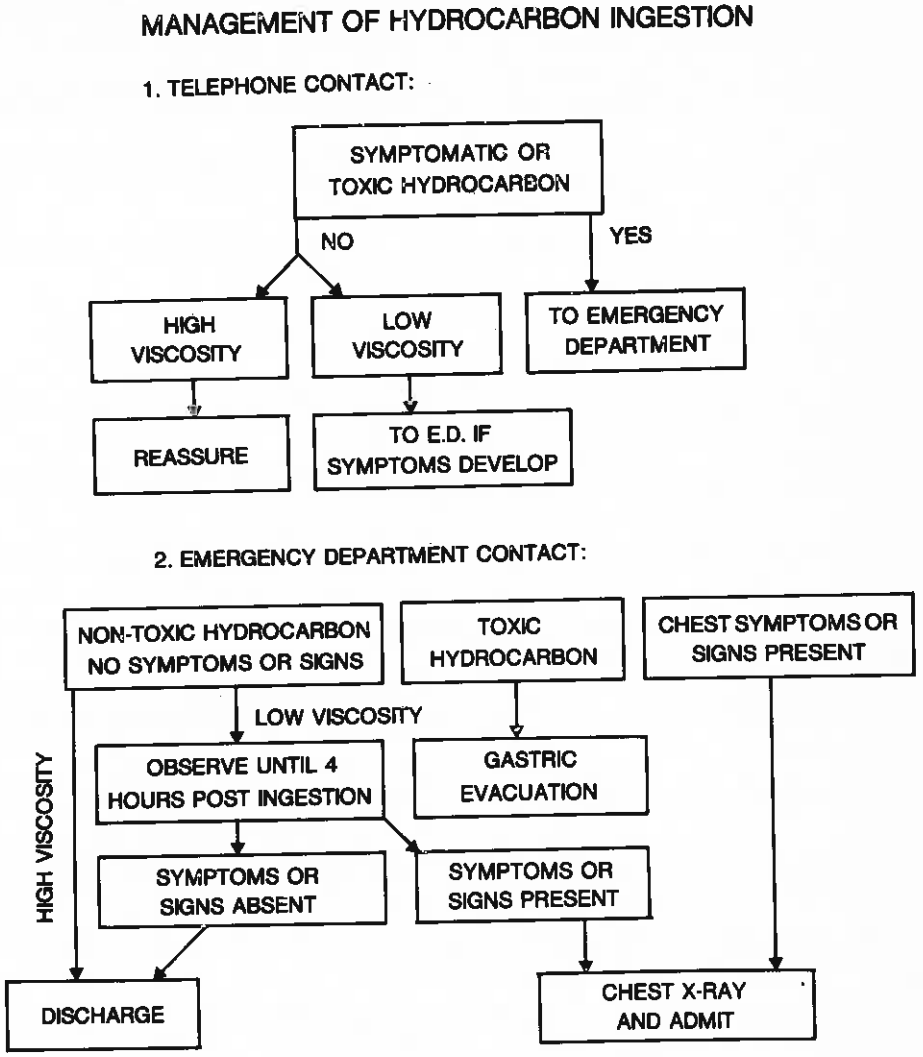
bon ingestion. A point not addressed in the literature is the fact that these substances are not soluble in aqueous solutions leaving one to question the amount removed during gastric lavage. Length of time taken to complete these procedures is approximately equal. However, ipecac induced emesis is less labour intensive. Also passage of the lavage tube often initiates vomiting and most patients require at least some degree of physical restraint in the supine position during this procedure. The potential complication of vomiting while supine is well known.

After gastric evacuation, the second confusing issue is whether to order a chest X-ray. As mentioned previously, only the low viscosity agents, those with the physical consistency of kerosene, cause aspiration pneumonia. In most of these ingestions pneumonia does not occur. In the majority of patients who do develop pneumonia clinical evidence of this condition is present within four hours of ingestion. Although in some cases pneumonia develops many hours after the event, these are simply a radiologic diagnosis and are not clinically significant. The treatment of hydrocarbon aspiration pneumonia is supportive. Prophylactic antibiotics and steroid therapy have not been shown to be efficacious, and there is evidence that they may in fact worsen the outcome. Therefore chest X-rays are not indicated in patients who do not experience respiratory symptoms or who do not demonstrate pulmonary signs.

Hydrocarbon ingestions are common. The potential complications of aspiration pneumonia and systemic toxicity are well known. The fact that the majority of patients suffer neither is also widely appreciated. Therefore, the need for a rational management plan is evident. My approach is outlined in the accompanying algorithm and may be summarized as follows.

If the initial contact is by telephone and the substance is of high viscosity and lacks systemic toxicity then the caller can be reassured providing the patient is asymptomatic. If the substance is of low viscosity, lacks systemic toxicity and the patient is asymptomatic (no choking, vomiting, coughing or dyspnea) he need not be brought to the emergency department unless any of the above symptoms develop. If the patient has ingested a toxic substance in sufficient quantity to cause toxicity or if he has symptoms he should be brought to the hospital immediately.

MANAGEMENT OF HYDROCARBON INGESTION



For emergency department patients who have ingested a hydrocarbon that can cause systemic toxicity in an amount great enough to cause damage, a gastric evacuation procedure is indicated. If the patient lacks both the symptoms and the signs of aspiration pneumonia chest X-ray is not indicated. However observation for four hours post ingestion should be considered if the hydrocarbon is of low viscosity. If he remains asymptomatic he can then be discharged. The patient or his family must be cautioned to return if symptoms develop. There is no need for a 24 hour chest X-ray in the asymptomatic patient. If either symptoms or signs of pneumonia are present, a chest X-ray should be ordered. All patients with hydrocarbon aspiration pneumonias presenting in the first few hours post ingestion should be admitted to hospital.

Table 1

Toxic Hydrocarbons

Halogenated
Camphor
Aromatic
Toxic Solutes Present
Pesticides
Heavy Metals

Scientific Section

Methemoglobinemia – a Cyanotic Patient

by John F. WHELAN, M.D.*

Abstract

Presented is a female who developed sudden onset of shortness of breath and cyanosis. The diagnosis of methemoglobinemia was suspected from her history, symptoms, signs and investigations.

Methemoglobinemia's etiology is varied but should always be entertained in the differential diagnosis of a cyanotic patient when other common etiologies are ruled out.

Key Words

Methemoglobinemia, Cyanosis, Aniline, Methylene Blue

Résumé

On présente le cas d'une malade qui a manifesté un épisode soudain de dyspnée et de cyanose. À partir des antécédents, des symptômes, des signes et des études paracliniques, le diagnostic de méthémoglobinémie est évoqué.

Les causes de la méthémoglobinémie sont variées, mais cette affection doit toujours être considérée lors d'un diagnostic différentiel chez un malade cyanotique alors que d'autres causes courantes de la cyanose ont été écartées.

Méthémoglobinémie, cyanose, aniline, bleu de méthylène

Introduction

Methemoglobin can result from hereditary causes and a wide variety of chemicals including prescribed medications. Nitrates and aniline derivatives are the chemicals which most frequently produce methemoglobinemia.[2]

Case Report

A 28 year old female presented with severe shortness of breath, cyanosis and confusion. She had participated in a car rally with her husband, and during the event, had complained of fatigue and dizziness. Her husband noted that she was blue and short of breath. She denied chest pain, or taking any medication including birth control pill. Her past medical history was unremarkable.

On physical examination the patient was lethargic, fatigued, dyspneic, peripherally and centrally cyanotic with a darker color than blue. Her respiration rate was 42 and pulse rate was 148. However, her chest exam conveyed normal air entry bilaterally, trachea midline, mild indrawing and normal heart sounds. The rest of the physical exam was within normal limits.

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Chest x-ray was reported as normal. The patient was started on 100% O_2 and arterial blood gases were drawn. The blood specimen was noted to be murky brown.

pH 7.42 $HNaCO_3$ 19 PCO_2 32 PO_2 264

Further questioning revealed a spill of High Octane Booster Fuel occurred during the rally. The patient was exposed by sitting on a seat which had been saturated with this substance. It was then ascertained that the fuel, sold under the brand name of Moroso, contained aniline. Hence, the diagnosis of methemoglobinemia was suspected and she was treated with 2 doses of 1 mg/kg of methylene blue. Subsequently, the methemoglobin level was reported as 53%. The patient was discharged 24 hours later in good health.

Discussion

When the iron molecule of hemoglobin is in the ferric (Fe^{3+}) state rather than the ferrous (Fe^{2+}) state, the abnormal hemoglobin is methemoglobin. Methemoglobinemia occurs when the concentration of methemoglobin in erythrocytes is greater than 1%. This can result from hereditary presence of an abnormal hemoglobin structure, hereditary deficiency of the enzyme methemoglobin reductase, or exposure to various drugs or chemicals that increase the rate of oxidation beyond the reductase capacity of the erythrocytes. Nitrates and aniline derivatives are the chemicals which most frequently produce methemoglobinemia.

Symptomatology is determined not only by the concentration of methemoglobin but also by its rate of formation and duration of action. Even though cyanosis may appear with methemoglobin levels of 15%, most patients experience ill effects such as headache, fatigue, tachycardia, weakness and dizziness when the levels reach 30–40%. Upon reaching 60%, oxygenation of tissues becomes severely compromised, resulting in dyspnea, acidosis, bradycardia, paralysis, stupor and coma. Lethal concentration is probably greater than 70% and is usually due to heart failure from hypoxia.

Several characteristics of cyanosis of methemoglobinemia were evident in this patient to help distinguish it from cyanosis of oxygen desaturation. The lips and mucous membranes were brown rather than blue in colour. This chocolate cyanosis is the hallmark of methemoglobinemia. Secondly, upon administration of O_2 by mask, her cyanotic condition improved very little. In addition, both venous and arterial blood appeared murky brown. Finally, when the chocolate cyanosis continued despite high arterial oxygen tension (264), the diagnosis of methemoglobinemia was strongly suspected and confirmation test ordered.

One of the unique features of this case was the route of exposure. skin absorption of aniline has been reported in children[2], however, a review of current literature failed to demonstrate recorded cases of adult toxicity.

The possibility of a concurrent inhalation exposure was ruled out when the following facts were considered: the vehicle was well ventilated and the patient's husband remained asymptomatic. The route of exposure, rapid rate of absorption and severity of symptoms, in this case, merit special mention.

Along with general supportive care and skin decontamination, the drug of choice

Journal Club

by Ian W. Cordon, MB. FRCP(C)

for treating severe methemoglobinemia (greater than 30%) is methylene blue. The usual dose is 1 to 2 mg/kg of a 1% solution given over 5 minutes.[1,2,3]

If cyanosis and hypoxia persist after an hour, a second dose can be given. The total dose should not exceed 7 mg/kg as this could result in increased levels of methemoglobin. Two important possibilities should be considered if treatment fails – congenital deficiency of methemoglobin reductase and associated glucose – 6 – phosphate dehydrogenase deficiency. Ascorbic acid and hyperbaric oxygen are of little or no value in the treatment of acquired methemoglobinemia.

Conclusion

Methemoglobinemia, even though a rare entity should be considered in the cyanotic, distressed patient when symptoms and signs do not suggest oxygen desaturation from common etiologies.

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The International Collaborative Study Group. N. Engl. J. Med. 1984; 310: 9–15. Reduction of infarct size with the early use of timolol in acute myocardial infarction.

Modern treatment of myocardial infarction hinges on the concept of minimizing myocardial damage (e.g. by providing O_2). It is thought infarct size is directly related to prognosis.

This is the report of a randomized, doubleblind, placebo-controlled, multicentre study on the effect of the beta-blocker timolol on infarct size when given intravenously within five hours of onset of symptoms. Contraindications to beta-blockade were bradycardia, hypotension, severe left ventricular failure, QRS greater than 0.11 second, A-V block, or history of bronchospasm. Various parameters were used to judge infarct size. These were pain and need for analgesia, cumulative creatinine kinase release, and vectorcardiographic assessment (e.g. rapidity and size of ST changes and maximum QRS vector change).

Myocardial ischemia and infarct size were statistically significantly reduced in the timolol group.

The postulated mechanism is a reduction in myocardial metabolic requirements due to the combination of a reduction in both blood pressure and heart rate. Beta-blockers are of proven value in reducing re-infarction and sudden death after MI, perhaps the time to begin treatment is in the emergency department.

J. Nordehaug & G. von der Lippe. British Heart Journal 1983 vol 50: 525–9. Hypokalemia and ventricular fibrillation in acute myocardial infarction.

This study is an offshoot of the Norwegian multicentre postinfarction trial of timolol. The 1074 patients with proven acute myocardial infarction were divided into groups upon admission according to their serum potassium concentration. This was then correlated with the incidence of ventricular fibrillation in the first 48 hrs. Among patients who were hypokalemic ($K^+ < 3.5$) 17.2% developed V.Fib., and of a sub-group with $K^+ < 3.0$ 33.3% (8/24) developed V.Fib.

The incidence of V.Fib. in normokalemic (3.5–5.1) patients was 7.5%. In another subgroup (patients with K^+ 4.1–5.1) the incidence was only 5.9%. Thus the graph of rate of V.Fib. versus K^+ concentration showed an impressive downward curve. Hypokalemia as in other studies was here more common in women (17.3% versus 9.2% in men).

The authors conclude there is a statistical association between K^+ and likelihood of V.Fib. The next step is to determine whether early correction of the serum K^+ will reduce the risk of V.Fib. If so this may become the role of the emergency physician, since V.Fib. occurred earlier as well as more frequently in the hypokalemic group (median time 0.3 hrs vs 7 hrs).

J. Kennedy et al. N.Engl. J.Med. 1983. 309: 1477–82. Western Washington randomized trial of intracoronary steptokinase in acute myocardial infarction.

This article reports the initial clinical results and mortality for 250 patients participating in a randomized multicentre trial comparing intracoronary streptokinase with conventional therapy for acute myocardial infarction.

The criteria for admission were rather strict in an effort to exclude patients with an already completed infarction. All eligible patients underwent cardiac catheterization. If a coronary thrombosis was present the patient was then entered into the study and on a random basis given intracoronary artery streptokinase by infusion. The infusion was continued until all the visible thrombi were lysed and there was reperfusion of the vessel, or until 350,000 units had been given.

The mean time from onset of symptoms to randomization (i.e. completion of initial cardiac catheterization) was 276 minutes. 68% of patients with complete occlusions had reperfusion during the procedure, as did 81% of those with subtotal occlusion. Angiographic complications occurred in 14.2% of the streptokinase group and

7.8% of controls. Length of hospital stay was similar in both groups, but on discharge there was statistically less angina in the streptokinase group.

Most striking was the six month mortality rate of 3.7% in the streptokinase group compared with 14.7% in the control group.

A. Yellin et al. Arch. Surg. 1983, vol 118: 1378–83. Diagnosis and control of post-traumatic pelvic hemorrhage.

Pelvic fractures and penetrating injuries can be associated with a very high mortality from hemorrhage. This is explained by the extensive network of major blood vessels which are quite adherent to the bony pelvis and render surgical attempts at precise ligation or pelvic packing often unsuccessful.

Yellin et al. describe 14 cases (6 blunt trauma, 6 penetrating, and 2 iatrogenic) of post-traumatic pelvic hemorrhage diagnosed and treated with transcatheter angiographic embolization. The indications and technique are discussed. The average transfusion requirement prior to embolization was 12 units. 13 of the 14 patients survived.

Complications of transcatheter embolization are those of diagnostic angiography, plus distal tissue ischemia or infarction, passage of emboli through traumatic arteriovenous fistulae into the pulmonary circulation, and possible renal toxicity due to excess contrast medium (especially in the hypotensive). Nevertheless the authors claim a success rate of 90–95% with this technique. Clearly for it to be much help in the early management of a trauma patient the radiology department needs to have aggressive and cooperative staff.

A. Joffe et al. Am J Dis Child. 1983 vol 137: 1153–56. Which children with febrile seizures need lumbar puncture?

Many authorities state all children with a first febrile seizure require a lumbar puncture as a routine part of their investigation. However by comparing the incidence of febrile seizures and of meningitis it can be concluded the probability of a child with a seizure and a fever having meningitis is about 0.1%.

From a retrospective review of the emergency department records of 241 children with their first febrile seizure five items associated with an increased risk of meningitis were identified. These were: abnormal neurological finding, suspicious physical finding (e.g. petechial rash), focal seizure, seizure in the department, and having seen a physician in the previous 48 hrs. If none of these were present the negative predictive value was 100%. If LP. was performed only on those children with one or more risk factors, then 62% would have been spared LP. and all those with meningitis would have been identified.

As a retrospective study this can only be used as the starting point for a prospective study. The authors are careful to emphasize the risk factors should not become a checklist but should rather assist in approaching the child with a first febrile seizure in an organised manner. I'm sure they want to avoid the kind of controversy generated by high yield criteria and skull X-rays.

L. Morgenstern & R. Uyeda. Surgery Gyn. and Obst. 1983, vol 157: 513–7. Nonoperative management of injuries of the spleen in adults.

It is well accepted that a nonoperative approach is desirable in the management of children with splenic injuries. This series demonstrates success with the nonoperative approach in young adults (average age 27).

I think this article well illustrates the difference in scientific quality of surgical journals and medical journals. This basically consists of a series of case reports followed by a discussion containing totally unsupported statements. The authors conclude that since most of their patients were young adults, splenic hemostasis is superior in the young. They set arbitrary limits on blood transfusion, length of bed-rest and of hospitalization and conclude these are appropriate on the basis of their 17 successes. The definition of when to treat a splenic injury nonoperatively is "... experienced surgical judgement in a setting when close clinical and laboratory observations are possible". This type of discussion (i.e. "we do X because it works") is of no value.

This article is of value in as much as it documents the successful nonoperative treatment of 17 young adults with splenic injury, with no delayed bleeding and resolution of splenic scintiscan defects over several months. It would have been of much more value to demonstrate the selection of patients involved some logical process.

D. P. Hartley et al. 1983 Arch Surgery vol 118: 1384–87. Myocardial dysfunction following blunt chest trauma.

Myocardial contusion is a frequently missed diagnosis. In fact clearly defined criteria for the diagnosis do not exist. In this study 35 patients who were admitted with blunt chest trauma were assessed using radionuclide ventricular angiography (RNA) and left ventricular segmental wall studies, both noninvasive procedures.

Seven patients had ECG abnormalities, and all of these also had RNA changes, but none had increased myocardial CPK. Altogether 26 (74.2%) of the 35 patients had evidence of reduced right or left ventricular function. 8 of 9 patients with documented steering wheel injuries had reduced ventricular function.

Of the 16 patients with chest wall fractures 55% had right ventricular depression and 25% had left ventricular depression. Presumably this proportion is a consequence of the right ventricle's anterior position.

In summary, radionuclide ventricular angiography identified depressed ventricular function in 26 of 35 patients admitted with blunt chest trauma. Of these only 7 had ECG changes.

M. Bracken et al. JAMA. 1984, vol 251: 45–52. Efficacy of methylprednisolone in acute spinal cord injury.

This study reports the results of a massive multicentre double-blind randomized trial of high (1 Gram/day) versus low (0.1 Gram/day) dose methylprednisolone in 330 patients with acute spinal cord injury. There are eight pages here crammed with all kinds of data and multivariate analysis. Simply put; the only difference between the two groups was in terms of side-effects.

The rationale for giving steroids in this situation is based almost exclusively upon animal studies. There are obvious parallels to the steroids in head injury debate where animal studies were encouraging but clinical studies have consistently failed to show any neurological benefit over placebo. As an accompanying editorial points out, perhaps a similar amount of energy and expense could be (better) directed at answering the question of whether steroids have any place at all in spinal cord trauma before considering in what dosage. Steroid related side-effects (delayed wound healing, all kinds of infection and sepsis, G-I bleeds, and fluid and electrolyte alterations) are not inconsequential.

N. Netto. Urology 1983 vol 22: 601–3. Traumatic rupture of the female urethra.

Traumatic rupture of the female urethra is quite unusual. (The common causes are secondary to obstetrical complications and

vaginal surgery.) Three cases of traumatic rupture from pelvic fractures are briefly described. Bleeding from the urethra and difficult catheterization are the major signs, while the presence of a pelvic fracture should make one at least consider the possibility. It is because the female urethra is short and relatively mobile that its injury from trauma is rare.

Continence in women is dependant upon the bladder neck. Consequently distal injuries are usually easily repaired while urethrovesical separation requires reconstructive surgery often in two stages over about three months. One of the commonest complications, urethro-vaginal fistula formation, can be best prevented by meticulous examination and repair of any vaginal tear at the time of injury.

W. C. Abbott. Surgery Gyn. & Obst. 1983 vol 157: 585–93. Nutritional care of the trauma patient.

There is no point in trying to condense this review article. It is well written, and gives a good account of what happens to various organ systems after the initial period of resuscitation and treatment. Late mortality in trauma is mostly due to sepsis and organfailure. Aggressive nutritional support can help decrease the overall mortality from trauma.

R. Latham et al. JAMA 1983 vol 250: 3063–6. Urinary tract infection in young women caused by Staphylococcus saprophyticus.

The most frequent organism cultured in UTI's in healthy young women is Escherichia coli. It is found in about 80% of cases. In Europe the next most common organism is Staphylococcus saprophyticus accounting for 7–26% of infections. Until recently this has been such an unusual cause of UTI in North America that most urine cultures growing Staphylococci would be considered contaminated.

In this study from Seattle, S. saprophyticus was found in 11% of UTI's with Klebsiella (3%) and Proteus (2%) third and fourth. 41% of S. saprophyticus infections involved the upper urinary tract compared with only 16% of E. coli infections. Almost all (86%) S. saprophyticus isolates were resistant to Pen G making it highly probable they were also resistant to ampicillin although this was not specifically tested.

The authors suggest Staphylococcus saprophyticus will be appearing more frequently in our urine microbiology reports as laboratories here look for it, especially at low colony counts (the generation time is longer than that of enteric bacteria) which might otherwise be called contamination or mixed gram positive infection.

Scientific Section

Research – So What!

by Bruce M. T. Rowat, M.D., FRCP(C)

1983 was an important year in Emergency Medicine in Canada. The year end saw the Royal College's first written and oral examinations in Emergency Medicine. A bench mark of clinical competence and excellence was established.

Although a landmark of the specialty development in this country – let there be no misunderstanding that there is still work to be done in the "prepetrating" of Emergency Medicine.

One of these areas in that of research in Emergency Medicine. But research – so what! Why research? Why bother? The question has been asked before and is now being posed again.[1,2,3,4]

Research is the child of the enquiring mind, but a tool for the satisfying and maintaining of an inquisitive spirit. It is to the scientific method, as is the teacher to the student. It is, in the words of Baraff "a basis for rational medical practice and an effective means for defining the discipline and the credentials for those in the academic community".[5]

The specialty in this country will not continue its appropriate development as long as this young discipline encourages but research consumers rather than active researchers, "takers" rather than "givers".

After years of having criticised the lack of significant published research in Family Medicine in this country, and its lack of "academic stature" in Canadian Medical Schools – it is now time for Emergency Medicine to "put up or shut up".

Where there is no research, the specialty perishes.[6] The academic imperative is clear – the troika of service, teaching and research applies equally to Emergency Medicine as well as to other specialties. The University has set these rules and it is by these rules that Emergency Medicine will be judged.

What are the barriers to research in Emergency Medicine and what are some of the solutions?

The first is lack of interest as, "where there is a will, there is a way". How many fugitives from academia are to be found in Emergency Departments? "I like the University setting and its stimulation, but I am doing Emergency Medicine because I don't want to do research".

And what about time? "Who has got time these days?" We all have time, it just depends on how it is used. If your clinical service load is significant and you are trying to remember which day of the week it is when coming off three midnight shifts in a row – who has the time and energy to put together a worth-while research project and then to follow it through. You cut back, however, on your clinical time and unless you have grant support or an arrangement of mutual support within your hospital's Emergency Department – it is going to cost the researcher. Bad news perhaps for the entrepreneur-minded Emergency Physician.

A third obstacle is perhaps the mind-set of many Emergency Physicians. Does the episodic kind of care provided in the Emergency Department effect our ability to think over time about the research potential of a given idea. For the individual who has but an occupationalist's view of his work – "I do my shift and when I'm off, I'm off" – (? off where?) – the "spectre" of an ongoing commitment to a project is not likely to be greeted with much enthusiasm. And yet for Emergency Medicine to not acquire this horizontal mind-set which is critical to the research endeavour, this will only result in the specialty's failure as an academic discipline. Should an Emergency Physician in a University setting not be willing to make such a commitment, perhaps he would do better in furthering his career in a community hospital where these demands are not made.

Yet another difficulty may lie in the very **variety** of **clinical problems** seen in an Emergency Department. The traditional specialist has an advantage, perhaps, in that his patient population is more defined, thus allowing for a more time-and-effort-intensive study of a given problem. On the other hand, the Emergency Department affords us the opportunity of seeing a large number of unselected patients – and this, with diligence and the use of the computer, should afford Emergency Medicine significant opportunities in clinical research.

A fifth barrier to research is clearly the **lack of research-competent and research-trained Emergency Physicians**. Here, the concept of Faculty Development should be introduced. If your Emergency Department does not have someone with a research background – then take someone from within the group and support him, if necessary, financially, while he acquires the tools necessary to do the job. At the same time, valuable University resources can be tapped – with Emergency Medicine drawing on the expertise of colleagues in research design, biostatistics, etc. in other departments. It is through this faculty development that we will develop a role-model of the researcher in Emergency Medicine for the Resident to emulate. The use of Journal Clubs as well can contribute to a more critical approach to medical literature and a greater appreciation of the importance and the "How's and How Not's" of research methodology. The appointment of a research co-ordinator to each Emergency Medicine Programme along with the development of a research curriculum for both faculty and trainees alike have been found to be of help in promoting research in this specialty.[7]

The question of **funding**. I have left this to the end as I believe that it is only when the above-cited obstacles are overcome that funding really then becomes an issue. In this regard, Emergency Physicians need to acquire the skills of preparing grant proposals, and learning where to apply for funding. In other words, how to compete with one's colleagues in other specialties in obtaining research funds.

Finally, in promoting research as a priority, this should in no way be at the expense of the discipline's recognition of the importance of the clinician-teacher in Emergency Medicine. Emergency Medicine needs both the researcher and the teacher.[8]

Research in Emergency Medicine is the responsibility of those involved in its practice. What remains to be seen is how responsible we really are.

by Bruce M. T. Rowat M.D., FRCP(C)
Director of Emergency Services
Toronto General Hospital

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Emergency Medical Training Programmes

Toronto, Ontario

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

University of Toronto

Length of Programme: four (4) years after graduation, three (3) years after rotating interneship.

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.

Programme Director: Bruce M. T. Rowat, M.D., C.M., F.R.C.P.(C), Director of Emergency Department, Toronto General Hospital, 101 College Street, Toronto, Ontario M5G 1L7

RCPS

Kingston, Ontario

Hospitals Kingston General Hospital, Hotel Dieu Hospital

University Queen's

Programme Director Dr. L. E. Dagnone, Emergency Dept., Hotel Dieu Hospital, Kingston, Ontario, K7L 3H6

Length of Programme 4 yr post-M.D. or 3 yr post internship

Size maximum of four (4) residents per year.

Accreditation RCPS (pending)

London, Ontario

Hospitals: St. Josephs Hospital, University Hospital, Victoria Hospital

University: University of Western Ontario

Length of Program: Three years–post internship

Size: One resident per year

Accreditation: R.C.P.S. (pending)

Program Director: Dr. R. G. Anthony, Department of Emergency Medicine, Victoria Hospital, 375 South Street, London, Ontario N6A 4G5, Phone: (519) 432-2352

Deadline for Application: September 15th

Montreal, Quebec

University McGill University

Hospital Royal Victoria Hospital Emergency Department

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

Length of Programme 3 years

Prerequisites at least 1 year mixed or rotating internship

Number of Residents Accepted per year: 4 residents

Deadline for applications October 30, 1983

Accreditations: RCPS(C) (pending)

Calgary, Alberta

University of Calgary

Hospital Affiliation Foothills Hospital, Alberta Children's Hospital

Programme Director R. Abernethy, 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: Oct. 30

Accreditation: RCPS(C) (pending)

CFPC

Toronto, Ontario

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

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

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 M.D. 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 Oct. 15.

Hamilton, Ontario

University McMaster University

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

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

Length of Programme 3 year post M.D. integrated programme with both Family Medicine and Emergency Medicine, or free-standing 3rd. year post CCFP. Candidates may enter at 1st., 2nd., or 3rd. postgraduate year levels.

Accreditation: CFPC (pending)

Deadline for applications; Nov. 30.

Ottawa, Ontario

University Ottawa

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

Programme Director Dr. A. F. Henry, Chief, Emergency Dept., Ottawa Civic Hospital, 1053 Carling Avenue, Ottawa, Ontario K1Y 4E9

Length of Programme 3 years post M.D., 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

Programme Director R. Abernethy, Div. Emergency Medicine, Foothills Hospital, 1403-29th N.W. Calgary, Alberta T2N 2T9

Length of Program: one year post CCFP

Number of residents per year: two

Deadline for applications: Oct. 30.