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CAEP REVIEW

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Resident's Corner

Why Do Research?

In the specialty training requirements for Emergency Medicine published by The Royal College of Physicians and Surgeons of Canada, research is listed as an elective rotation. To note that research is important appears self-evident, but I wish to examine this statement more thoroughly.

Research forms a major component of virtually all established specialty training programs, both at the postgraduate level, and at that of the attending physicians; to date, research in Emergency Medicine in Canada has, with a few notable exceptions, been absent. What are the implications of the development of research to residents, to Emergency Medicine training programs, and to Emergency Medicine, in general, in Canada?

First hand experience in research allows one to develop insight into not only statistical analysis, but experimental methodology and design. How one asks a question, and how one goes about attempting to answer it in a research protocol often influences the answer which is finally arrived at. This experience is important to the development of an ability to intelligently and critically handle the constant onslaught of new information characteristic of medical practice. Many aspects of medical care are based upon poorly designed or inadequate studies which have become entrenched as dogma; only the ability to critically analyze research papers allows one to separate valid conclusions from non-truths.

The potential for an individual to make a significant contribution to the bulk of new medical knowledge often appears slight. Nevertheless, although an individual's research may be criticized as trivial at the time of its publication, retrospective analysis often reveals that an important role was played in the resolution of a particular problem. There are, however, notable exceptions; Goldman was still a resident when he published his landmark paper in the area of preoperative risk assessment(1).

In addition, most useful papers are an outgrowth, either directly or indirectly, of questions related to patient care. The "publish or perish" syndrome is prevalent in medical literature, and the failure of authors and editors to examine material submitted for publication critically, in the context of these questions, has led to the loss of credibility of research in many medical circles.

Research may lead to both intangible and tangible personal rewards. All aspects of clinical practise become, in time, repetitive. Research offers an opportunity to ask new questions and one avenue for personal creativity. This may, in part, account for the enthusiasm and intensity with which some researchers approach their work, often to the amazement of an independent observer.

In addition, over the next few years, as residency training programs in Emergency Medicine become more uniform in quality, and as prime jobs become more scarce, applicants for jobs will be more critically examined, and on a greater number of planes. Experience in, and ability to do, research will undoubtedly become one of the criteria by which job applicants will be assessed, particularly for academic appointments.

Finally, although The Royal College of Physicians and Surgeons of Canada, and The Canadian College of Family Physicians now recognize Emergency Medicine as a unique entity, and residency training programs are now firmly established, it still lacks credibility and acceptance among our colleagues in other specialties. This situation will presumably improve somewhat with time; however, it will never be completely resolved until the practitioners of Emergency Medicine demonstrate the interest and the ability to engage in worthwhile original research related to their specialty. This is a characteristic of virtually all other groups of medical practitioners, and it is critical to the definition of Emergency Medicine as a unique and credible specialty.

Donald Livingstone, M.D.

References:

(1) Goldman L, Caldera DL, Nussbach SR, et al: Multifactorial Index of Cardiac Risk in Non-cardiac Surgical Procedures. N Engl J Med. 297:845, 1977.

Letters to the Editor

Dear Sir:

I read with interest the recent article on Disseminated Gonorrhoea by Dr. Charles Ramesar.

I noted, not without a certain wry amusement, the profound and scienfitic observation that "this disease will not diminish until people stop having sex for fun." I wonder when Dr. Ramesar considers this enviable, nay utopian, situation will occur?

Yours faithfully,
David Lewis
MB, BS, DRCOG

Dear Sir:

I have just finished reading the present CAEP Review of January 1983. Once again I must congratulate you and the staff for putting out a most informative journal. The articles seem complete as does all the information regarding Emergency care at home and abroad.

I would, however, like to comment regarding the article entitled "President's Notebook". Throughout this article you bring reference to the fact that "certification" is a reality and residency training programs seeking accreditation are proliferating across the country. CAEP has been active in promoting the views of Emergency physicians throughout this process. One of the main reasons that CAEP is able to present such a strong voice in Emergency medicine, is the extent and distribution of its membership. It is quite disconcerting to me as well as a great number of my colleagues, that the only reference regarding this accreditation program is to the Royal College of Physicians and Surgeons.

Seeing that a large number of your membership are actually Family Physicians who are either full time or part time Emergency physicians, I cannot understand your biased approach by only mentioning the Royal College certification and intentionally not referring to the certification route via the College of Family Practice of Canada. As you are aware, this certification program is well underway and the first year of examinations have now been completed. I feel that the College of Family Practice Residency Program as well as practice eligible examination process is valid and those physicians who have attended their certification by this route should be given adequate recognition for their achievement. When one looks at the requirements in order to obtain certification via this route, one would have to agree that these physicians must be well qualified to work as Emergency physicians within any Emergency centre.

Again, I congratulate you on your active participation in promoting Emergency care within our communities and feel that you have done a great deal to enhance both the status as well as educational standards required by Emergency physicians working in Emergency departments. But please remember Family Physicians are definitely an intergral part in community Emergency practice and if these Family Physicians choose to upgrade their educational and procedural standards, they are equally adept at manning Emergency departments as those physicians who have achieved certification by any other route.

Possibly I have over-reacted to feeling that you are demonstrating a definite bias in your reporting but if in fact my perceptions are accurate, I ask you to consider this bias and consider all individuals involved.

Thank you for taking time to consider my comments.

Your very truly,

B. Borden, M.D., C.C.F.P.
Director
Emergency Medical Services
North York General Hospital
Toronto

Reply to Dr. Borden

Dear Sir:

In a reply to Dr. Borden's letter, having participated in the initial exam for the Certificate of Special Competence in Emergency Medicine of the College of Family Physicians of Canada (CFPC), I am aware of the effort undertaken by them in promoting a means of upgrading skills and training for family physicians with a special interest in Emergency Medicine. Hopefully, this should lead to an enhancement of the quality of care provided by those physicians. The College indeed deserves credit for its role in promoting a higher quality of emergency care in Canada.

However, Dr. Borden should probably address his concern regarding the participation of family physicians in the Emergency Department to his own College. Following a request for information from the Section of Emergency Medicine of the Ontario Medical Association, the Executive Director of the College of Family Physicians of Canada stated that the major disciplines relating to Family Medicine are: Medicine, Pediatrics, Obstetrics/Gynecology, Psychiatry, and Surgery. Why is Emergency Medicine not considered a major discipline related to Family Medicine when the College has initiated residency programs and established a certification exam and when its members feel adamant that there is an on-going role for Family Physicians in the practice of Emergency Medicine?

In my article, I certainly intended no offence to those many family physicians providing exemplary care in Emergency Departments across the country. Nor did I intend to negate the effort of the College of Family Physicians of Canada in promoting a higher standard of emergency care across the country.

Yours sincerely,
Rocco Gerace, M.D.
President

To The Editor:

The article by Flannigan and O'Connor in your latest journal serves to emphasize better prehospital care. There is no doubt that improved and early management by both the public sector and ambulance services will improve cardiac arrest survival.

In August, 1982 we did a prospective study to assess the efficacy of our cardiac arrest procedures. However, we found some interesting statistics pertinent to prehospital care.

We were able to review 84 consecutive arrests seen in our Emergency Department. We divided these arrests into two groups — those who arrested in the Department and those who arrested prior to arrival with C.P.R. in progress. For our purposes we considered the patient resuscitated if a rhythm and blood pressure were established. The arrest was deemed successful if the patient was discharged home or to another facility with approximately the same quality of life as prior to arrest. We followed all resuscitated arrests until they either were discharged from hospital or died while in hospital.

The results of our findings are outlined in Figure 1. Of the 26 patients who arrested in hospital, 57.8% were resuscitated and 42.2% went home. However, if the patient was brought in with C.P.R. in progress, only 22.4% were resuscitated with 3.4% finally going home.

Both groups were comparable for age and previous cardiac history. In addition, the Emergency Room physicians conducting the arrests are all certified in B.C.L.S. and A.C.L.S. and generally follow a standard protocol in arrest situation.

The poor results of prehospital cardiac arrest must be assessed. Certainly by the time the ambulance personnel arrive at the scene either because of a poor history as to length of time of loss of conciousness or because adequate or inadequate C.P.R. is already in progress, the ambulance attendants must initiate their own B.C.L.S. Many of these patients are already dead but are given "benefit of the doubt". This is justifiable but is probably a contributing factor to the poor success of these "arrest" situations.

These "no win" situations in some instances might be avoided if the ambulance attendants could more accurately assess these patients with telemetry. Undoubtedly some of the patients are in a salvagable arrhythmia or arrest in the ambulance. Our ambulance personnel once again are bound by lack of training and equipment. Given the ability to perform A.C.L.S. (i.e. defibrillation, intravenous medication) the success rate will increase. One has only to look at the major U.S. cities with paramedics to see the efficacy of improved prehospital care.

The public sector can be a major contributor to improving the success rate. Seattle, Washington is a model for community education and its benefits to the cardiac arrest patient. It is so frustrating to hear a relative say they "tried mouth-to-mouth but it didn't help". Perhaps if that relative added proper cardiac compressions to the "mouth-to-mouth" that patient might be alive. How many choking victims might have been saved if somebody knew how to do the Heimlech manoeuvre? All these are the fundamental aspect of B.C.L.S. and must become a part of the community's education.

The in-hospital care of cardiac arrest is well examined and reviewed by most hospitals. Certainly medical and paramedical personnel are well trained in this aspect of patient care. It is considered fundamental for a doctor to manage a cardiac arrest.

Our statistics confirm that a patient who arrests in our hospital has a good prognosis comparable to any large American centre. The obvious problem is in the prehospital cardiac arrest. The dismal prognosis of these patients behooves us to examine means of improving prehospital care. It is up to the governments to find funding and to the medical community to promote a better system.

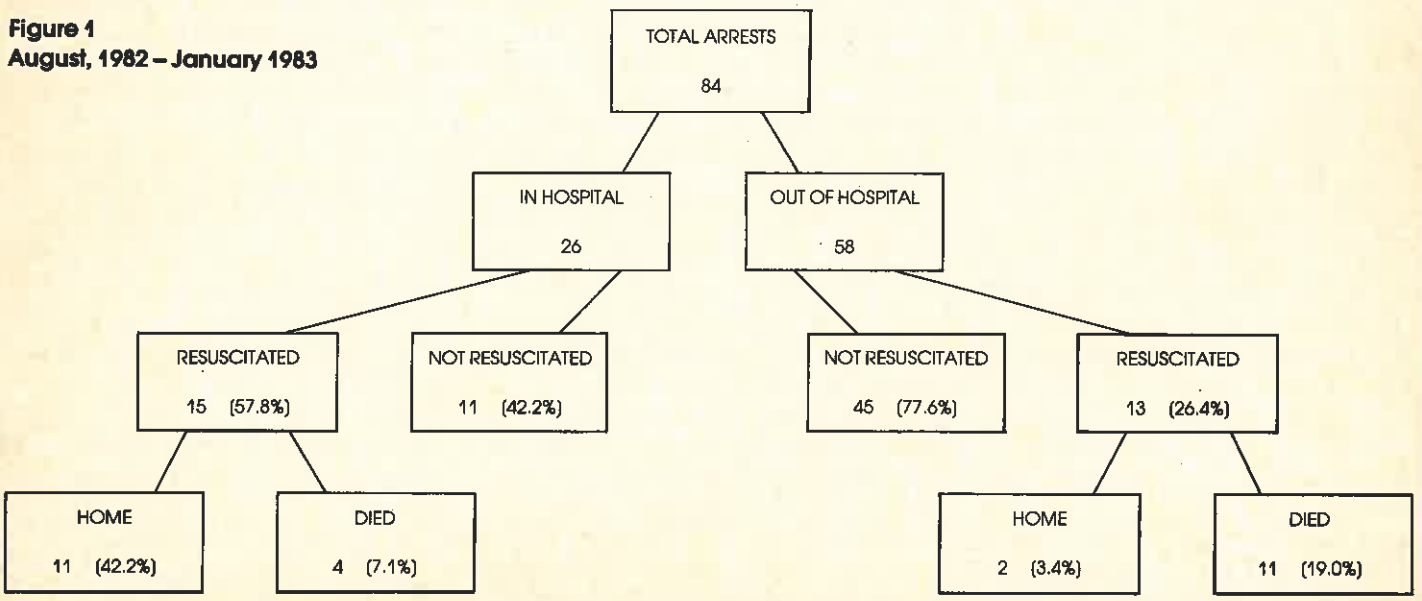
We submit, the following three points should be a priority in improving the health care system:

1. Education of the community in prehospital care and B.C.L.S.,
2. Improvement of the ambulance personnel to
   a) better assessment of the patient,
   b) improved equipment for monitoring and resuscitation,
   c) re-education and upgrading to A.C.L.S.,
3. Hospital Emergency Department should be in a position to provide adequate backup for ambulance personnel who are at the scene.

Unless these steps are taken, we do not see EVER improving the statistics we have provided.

Respectfully yours,
I. Fefferman, M.D.
L. LeBlanc, R.N.

Figure 1
August, 1982 – January 1983



Editorial

To Mecca Medical Centre, STAT!

It's still all too familiar. The critically ill or injured patient arrives in your emergency department, unannounced, inadequately stabilized, without adequate records or x-rays. You have little or no idea what's happened to the patient, what care they've had, how their condition has been affected. You have no time to summon the medical and paramedical staff needed, no time to ready the Operating Room, X-ray and labs, and you've got a busy shift on your hands with several other sick patients demanding your attention. You fight to contain your rage while assessing and managing the patient. An hour later, when the dust settles and you can finally get to a cup of coffee, you sigh and say "what's the use?" If you phone the referring M.D., you'll be lucky to find him and even if you do, you worry that if you really give him a piece of your mind, you'll never get another referral from there. Yet if you don't, how's he to know? What can you do to get him to call you first next time?

It's still all too familiar. A relatively quiet Saturday evening shift is suddenly turned into barely organized chaos by the arrival of a critically ill or injured patient. All of a sudden, you have to intubate, put in the chest tube, start the I.V.'s and get the x-rays done, and you're the only M.D. in the hospital. When you do finally get your consultant on the phone, he says transfer the patient stat, because your hospital isn't equipped and staffed to handle it. So while you're desperately trying to stabilize the patient, you have to try to find someone to accept the patient in transfer. The consultant at one hospital is no where to be found — Locating thinks his pager is broken. Everybody at the other hospital is in the Operating Room for the next two hours. Faced with a 45 minute transfer, you know it's best to get your patient on his way as fast as possible, or you may lose him. So you send him in the ambulance with a nurse while you make yet another phone call. Eventually, you get somebody, but they don't have any beds, so you have to reroute the patient. When the dust settles, and you finally get to a cup of coffee, you scratch your head — "This is supposed to be a *system*?" But what can you do? A nasty letter would only fall on deaf ears. A phone call would only bring meaningless sympathy and apologies that won't make a bit of difference next time.

In September 1979, CAEP adopted a Position Paper on Patient Transfer guidelines. In committee and on paper, they looked great — physician-to-physician contact, discussion regarding responsibility in transit, appropriate vehicles and equipment, adequate professional personnel, written guidelines, transfer agreements, etc. All very nice, but they're not working. Patients still arrive unannounced with no prior arrangements. Very few hospitals have written transfer agreements. Sending physicians are still frustrated by red tape, multiple phone calls and road blocks trying to arrange transfer. And these are not isolated instances — it happens regularly, all across the country, I'm told.

We need to re-think our approach to patient transfer. Emergency physicians are usually the ones on at least one end of these transfers, so we should be the ones to do the re-thinking. This has to be done at the local level, through district health councils, committees of emergency physicians, medical association sections, etc. Each hospital should identify a small core of attending staff who are authorized to receive patients and arrange transfers. The staff or his/her resident must be fully available at all times for a phone call. If residents are to be involved, they must discuss the transfer with the staffman prior to acceptance. In some centres, it may be practical to have *only* emergency physicians accepting all transfers — we are always on-site, always available within moments, always aware of the "bed situation", fully familiar with the capabilities of pre-hospital workers, etc.

Sending physicians must understand that the most vulnerable and dangerous period for the critically ill and injured patient is the time *in transit*, not that prior to transit. It's an odd paradox that the patient that was brought speeding to the hospital with "lights and sirens" is the same patient that we're so eager to put back out on the road, at risk again.

Physician-to-physician contact must be seen as essential prerequisite to safe transfer. If consultants are unavailable, the emergency physician should be phoned — contact must be made. It should be understood that the sending physician's responsibilities do not end once the patient leaves the department. Unless an ALS vehicle is used with base hospital physician involvement, the sending physician is responsible for that patient until arrival at the receiving institution.

Another aspect of patient transfer that needs to be addressed is the role of the nurse in this situation. All too often, it's the "float nurse" called down from the pediatrics ward that ends up accompanying the patient. She gets a brief report from the emergency nurse and then she's out on the road. She's not trained nor equipped to do much in transit. Usually, she's never tried to take a blood pressure in an ambulance, let alone bag a patient or do CPR while moving. If nurses are to be used in transfers on a regular basis, they must be specifically trained for the job. They probably should be emergency nurses, preferably who have already been involved in the care of the patient. Adequate "on-call" protocols must be developed to replace the nurse in the emergency department.

These are all frustratingly important issues that have not yet been adequately dealt with. It's time we all had another good hard look at Patient Transfer Policies, whether we're at Mecca Medical or North Overshoe General.

Peter L. Lane, M.D.

Pre-Hospital Advanced Life-Support: Ontario

A pilot training project in Metro Toronto will give Ontario advanced life support workers in the pre-hospital phase in 1983.

At present most ambulance attendants in Metro Toronto are certified by the provincial E.M.C.A. (Emergency Medical Care Assistant) process and function at the E.M.A. I level as defined by the C.M.A.

They are, however, grossly over trained for their present level of functioning. The current Ambulance and Emergency Care Programs offered in the community colleges involve nineteen courses over three semesters; totalling 1,110 hours. This compares with 160 hours as the recommended minimum training required to produce a level I E.M.A. by the C.M.A. guidelines.

The proposal is to train pre-hospital workers to the E.M.A. II level. This individual will be an intermediate between the basic and complete advanced life support worker. Training will be mainly directed toward impacting on the care of the trauma victim.

On completion of the level II program a graduate will have acquired additional skills including:

1. Application of P.A.S.G.
2. Advanced airway management including intubation
3. Intravenous therapy
4. Nitrous oxide analgesia administration
5. History taking and systems assessment.

The program will meet the requirements for the E.M.A. II as established by the C.M.A., and be based and administered through the Toronto Institute of Medical Technology.

Admission requirements;

1. E.M.C.A. certification + B.C.L.S. certification
2. Minimum two years experience on ambulance.

The program is divided into three blocks:

- **A.** Didactic and clinical instruction — 9 weeks.
- **B.** Ambulance rotation and introduction to Base Hospital — 6 weeks.
- **C.** Base Hospital Ambulance Service — 30 weeks.

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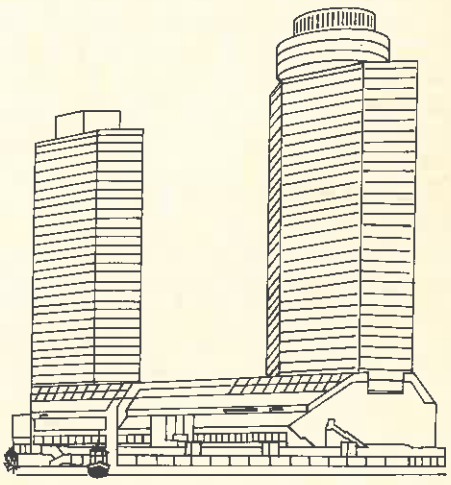
1983 Scientific Assembly of Emergency Medicine
Harbour Castle Hilton, Toronto
June 27 - July 1

- ***Programme:*** *Plenary Sessions*
  MD/RN Mini-courses
  ATLS/ACLS
  Spouse/Social Events
- ***Sponsors:*** *Canadian Association of Emergency Physicians*
  National Emergency Nurses Affiliation
- ***Further Information:*** *Ted Darby, Asst. Adm., Educational Services Sunnybrook Medical Centre, 2075 Bayview Ave., Toronto, Ontario, M4N 3M5*



President's Notebook

What is Emergency Medicine? All of us have heard varying definitions regarding the true entity of this beast. A specialty of breadth and depth. A specialty which crosses the bounds of all the traditional specialties to deal with emergent problems over the first hour or first four hours. Certainly these definitions describe what the emergency physician does, but does it make him a specialist?

Many of the perceptions of emergency physicians are rapidly changing. I am sure many of you remember when emergency departments were staffed by physicians who were seeking breathing time before going into practice, who wished to supplement their income, or who just could not "make it" elsewhere in the medical community. These motives for practicing emergency medicine are becoming the exception to the rule.

A questionnaire distributed to physicians involved in emergency medicine in the Province of Ontario has demonstrated that career oriented emergency physicians are indeed a stable group. Although young (30-40 years old), they average seven years in the practice of emergency medicine with the three years immediately preceding the questionnaire in the same hospital. Although firm figures are not available, it can be estimated that about 400 physicians are career oriented in emergency medicine in Canada. Experience and commitment to the discipline is a reality.

Most of you are aware of the activity in Emergency Medicine by both the College of Family Physicians and the Royal College of Physicians and Surgeons. Having established the concept of Certificate of Special Competence, the Canadian College of Family Physicians conducted the first Canadian certification exam in emergency medicine in Canada late last year. The Royal College of Physicians and Surgeons having recognized Emergency Medicine as a primary specialty is planning the first certification exam leading to a fellowship in the fall of 1983. The inherent recognition of emergency medicine as a discipline by these two bodies is clear.

In Canada, specialty training in emergency medicine is now being offered by at least seven university centres at the level of either the Royal College, the College of Family Physicians, or both. These institutions are now in the process of seeking accreditation from the appropriate certifying body.

In addition to this progress in post-graduate education, emergency physicians in several academic centres have been participating increasingly in the undergraduate medical curriculum. Very credible continuing medical education programs are being organized and to a great extent taught by emergency physicians.

Can we now say that with the significant advances made in the past several years that emergency medicine has achieved the stature of a true specialty? The answer to this question continues to be a resounding NO!

Allow me to quote an often repeated phrase: "In order for emergency medicine to survive it must gain a sound research base and be accepted in the medical schools as a discipline." In Canada, there has yet to be formed a full department of emergency medicine at the university level. Although some research is in progress in some institutions, it has not yet reached the credibility achieved by the other traditional specialities.

The challenge has been made and the ball is in our court. It is now our responsibility as emergency physicians to pick it up and run with it. The opportunity is now here to be on the ground floor of the building of a specialty and we must not allow this opportunity to pass. It is critical that emergency physicians begin to take an active interest in promoting research amongst their peers and even more importantly amongst the trainees, the emergency physicians of the future.

What will be the role of the Canadian Association of Emergency Physicians in this final hurdle? In order to carry out credible research, emergency physicians will need a vehicle to present the research, expertise in research methods and a sound source of funding. A forum has already been created to present scientific papers at the annual meetings. The CAEP review, as a scientific journal, stands ready to receive manuscripts to be considered for publication. The Annual Scientific Program Committee must develop educational programs devoted to the teaching of research methods.

Finally, CAEP has made a major commitment in the coming year to the formation of a research fund in emergency medicine. Although still in the planning process, we will apply to Revenue Canada for a charitable number allowing donations to be more easily solicited from the private sector to gain sound financial footing. The executive will undoubtedly be calling upon your assistance and support in this endeavor.

The challenge and opportunity to achieve true specialty recognition is here. It must not go unanswered.

Rocco Gerace, M.D.

Rapport du president

Qu'est-ce que la médecine d'urgence? Nous avons tous entendu une variété de définitions concernant la vraie identité de cette bête. Une spécialité tant par l'etendu que par la profondeur des ses intérêts. Une spécialité qui traverse les frontières de toutes les spécialités traditionnelles pour traiter des problèmes urgents pendant la première heure ou les quatre premières heures. Ces définitions décrivent certainement ce que fait le médecin d'urgence, mais celà en fait-il un spécialiste?

Bien des perceptions sur les médecins d'urgence évoluent rapidement. Je suis sûr que nombre d'entre vous se souviennent de l'époque où les départements d'urgence employaient des médecins à la recherche d'un peu de répit avant d'entrer en pratique, de ceux qui voulaient augmenter leur revenu ou encore de ceux qui ne pouvaient tout simplement pas "réussir" ailleurs dans la communauté médicale. Ces raisons pour pratiquer la médecine d'urgence sont en voie de devenir l'exception à la règle.

Un questionnaire distribué aux médecins travaillant en médecine d'urgence en Ontario a démontré que ceux qui ont choisi de faire de l'urgence leur carrière constituent en effet un groupe stable. Bien qu'ils soient jeunes (entre 30 et 34 ans), ils pratiquent la médecine d'urgence en moyenne depuis sept ans, dont les trois années précédant le questionnaire au même hôpital. Bien qu'on n'ait pas de chiffres exacts, on peut estimer qu'environ 400 médecins ont orienté leur carrière vers la médecine d'urgence au Canada. L'expérience et la dédication dans cette discipline est réelle.

La plupart d'entre vous sont au courant de l'activé du Collège des médecins de famille et du Collège Royal des médecins et chirurgiens dans le domaine de la médecine d'urgence. Ayant établi le concept de Certificat de compétence, le Collège des médecins de famille du Canada a organisé le premier examen de certification en médecine d'urgence au Canada à la fin de l'an dernier. Le Collège Royal des médecins et chirurgiens, ayant reconnu la médecine d'urgence comme une spécialité fondamentale compte tenir le premier examen de certification conduisant au "fellow" à l'automne 1983. La reconnaissance inhérente de la médecine d'urgence en tant que discipline par ces deux groupes est claire.

Au Canada, la spécialité en médecine d'urgence est maintenant offerte dans au moins sept centres universitaires par le biais soit du Collège Royal, soit du Collège des médecins de famille, ou les deux. Ces institutions recherchent présentement l'accréditation par le College concerné.

En plus des progrès enregistrés dans le domaine de l'éducation post-graduée, les médecins d'urgence dans plusieurs centres académiques participent de plus en plus à l'enseignement au niveau des études pré-graduées. Des programmes d'éducation médicale continue très ralables sont organisés et sont donnés en grande partie par des médecins d'urgence.

Peut-on maintenant dire que grâce à ces progrès importants au cours des dernières années que la médecine d'urgence a atteint le statut de spécialité? La réponse à cette question demeure toujours un *NON* retentissant!

Permettez-moi de citer une phrase souvent répétée: "Pour que la médecine d'urgence survive, elle doit acquérir une base de recherche solide et être acceptée par les écoles de médecine comme discipline". Au Canada, il n'existe pas encore de département de médecine d'urgence comme tel au niveau universitaire. Bien que des recherches soient en cours dans certaines institutions, cette discipline n'a pas encore reçu la crédibilité que possèdent d'autres spécialités traditionnelles.

Le défi est lancé, il n'en tient qu'à nous, médecins d'urgence, de le relever.

Nous avons maintenant la chance de jeter les fondations pour bâtir une spécialité et nous ne devons pas laisser passer cette chance. Il est essentiel que les médecins d'urgence manifestent un intérêt actif dans la promotion de la recherche parmi leurs confrères et encore plus important, parmi les résidents, les médecins d'urgence de l'avenir.

Quel sera le rôle de l'Association Canadienne des médecins d'urgence pour franchir ce dernier obstacle? Afin de procéder à des recherches valables, les médecins d'urgence auront besoin d'un véhicule pour présenter ces recherches, de conseils quant aux méthodes de recherche et d'une bonne source de financement. On a déjà créé un forum pour la présentation d'articles scientifiques lors de la réunion annuelle. La revue CAEP, en tant que journal scientifique, est prête à évaluer des manuscrits en vue de leur publication. Le Comité des assemblées scientifiques annuelles doit élaborer des programmes d'éducation afin d'enseigner des méthodes de recherche.

Finalement, CAEP s'est engagé à former un fond de recherche en médecine d'urgence dans l'année qui vient. Bien qu'encore à l'état de projet, nous ferons une demande auprès de Revenu Canada pour l'obtention d'un numéro de taxe permettant de soliciter plus facilement des dons auprès du secteur privé afin d'acquérir une base financière solide. Le Comité exécutif solicitera sans aucun doute votre aide et votre appui pour réaliser ce projet.

Le défi est lancé, la reconnaissance de notre spécialité est à notre portée. Ne laissons pas passer cette opportunité.

Rocco Gerace, M.D.

"There's Nothing Serious Wrong"

by Ian W. Cordon, M.B., CH.B.*

Recently, a patient of mine became quite irate at my suggestion that there was no serious cause for his chest pain.

"What do you mean, there's nothing wrong?" he cried. (The crucial word, serious, has already been forgotten). "If you can't find anything, then I'd better find someone who can!"

It used to be that if a doctor could find nothing serious this was a reason to rejoice. Now it is a reason to question the doctor's competence. "You'd better refer me to a specialist" is the request, direct or by implication. We all know that the bulk of patients seen in the Emergency, as elsewhere, have nothing major wrong but how do we tell them?

There are a number of ploys available to the pragmatic physician that may help him with this task.

First, there is the fake diagnosis. In its purest form this is the invention of a diagnosis to prevent the lengthy and mutually unsatisfactory discussion that has to take place if no diagnosis can be made. There are two important rules to follow. The diagnosis should have at least five syllables and end in "itis". Scapular pericapsulitis is far more acceptable than a strained shoulder from the season's first baseball game. However, such pure examples are rare. Much more common is the old lady who suffers from "rheumatics" or "fibrositis". A "touch of appendicitis" that doesn't require surgery will buy more time in a child crying with nocturnal tummy pain than abdominal pain N.Y.D. An even more grey area is entered in "costochondritis" for all those hard to interpret chest pains. (This diagnosis is facilitated by bony probing fingers). Fake diagnoses are also important for the relatives, for it is much nicer all around if your wife has migraines instead of tension headaches. It is most important, however, that the physician realize when he is making a fake diagnosis and why.

The next ploy is the "early diagnosis". This, I feel, is no less common but is much less defensible. There are similarities to the "fake diagnosis" manoeuvre but the fundamental difference is that the physician deceives himself instead of the patient. He uses the diagnosis as an excuse to prescribe treatment, as opposed to doing nothing at all. An example of this approach is the child

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with "an early ear infection", and nothing to find save a high fever and a prominent capillary or two on a tympanic membrane. Treat with Amoxil! Better use Amoxil for that red throat, persistant cough, or slight dysuria after intercourse, too. It is probably "early" strep throat, bronchitis or cystitis. Cimetidine (like Amoxil) is among the ten most prescribed drugs in Canada and is frequently given without proof of an ulcer. Maybe an "early" case?

The next approach is that of absolute logic and honesty. This involves meticulously detailing to the patient the possible diagnoses and explaining how by the absence of certain signs, symptoms and laboratory data each can be excluded. This time-consuming technique is usually met with considerable resentment, and is doomed to failure because your patient sees this as a form of trickery and deceit. He believes that you not only don't know what's wrong with him, but you haven't even got the guts to admit it!

One of my colleagues feels that Emergency Physicians have an advantage over Family Practitioners when treating patients with minor complaints. We can be blunt (we call it frank) for we have no need to pander to the sensitivities of patients we may see over and over again. We do not have to face the mother and daughter who, having already seen a specialist that found "nothing wrong", pay weekly office visits for the daughter's persistent and vague abdominal symptoms that keep her from school. Nor do we face the child with a cough of several months duration that disappears when he's distracted. My colleague's "call a spade a spade" attitude is fine and well, but it does not really address the problem of those patients in whom no definite diagnosis can be made.

Finally, there is guilt. Much has been written on the subject of how a patient's guilt may present itself but little on how to make use of the guilt. The most obvious example is that of smoking. Smoking can be blamed for almost any ill and the public is willing to accept this. Consequently if there is no clear diagnosis of treatment in the case of a smoker then one should at least take the opportunity to blame cigarettes and do some good. Of course, it is futile to try this approach with an alcoholic.

I cannot conclude by producing an answer to the problem of the patient who is frightened by his symptoms or his parental responsibility and needs the crutch of an iron-clad diagnosis or a penicillin prescription. The approach has to be as individual as the patient and physician involved. The Emergency Physician should be aware of these approaches as they are pieces of the jigsaw that often is the practice of medicine.

Scientific Section

Ethanol-Induced Hypoglycemia

by Amin A. Nanji.* M.B., Ch.B., F.R.C.P.(C)

Abstract

The association of symptomatic hypoglycemia with alcohol ingestion was first recorded forty years ago. Ethanol (alcohol) causes hypoglycemia by a variety of mechanisms. These include (i) alcohol induced fasting hypoglycemia, (ii) alcohol potentiation of drug induced hypoglycemia, (iii) "essential" reactive hypoglycemia in chronic alcoholics and (iv) alcohol promoted reactive hypoglycemia. Fasting hypoglycemia is due mainly to inhibition of gluconeogenesis by alcohol both directly and also by altering the intracellular redox potential. However, counter-regulatory mechanisms responsible for glucose homeostasis may also be impaired. Alcohol also augments the hypoglycemic action of insulin and sulfonylureas. The insulin response to glucose may be enhanced by prior exposure of pancreatic beta cells to alcohol.

Resume

L'association de l'hpoglycémie symptômatique à la consommation d'alcool fut rapportée pour la première fois il y a de celà quarante ans. L'éthanol (alcool) cause ne hypoglycémie par une variété de mécanismes. Ceux-ci comprennent (i) L'hypoglycémie de jeûne provoquée par l'alcool, (ii) la potentialisation par l'alcool de l'hypoglycémie en présence d'hyproglycémiants, (iii) l'hypoglycémie réactionnelle "essentielle" chez l'alcoolique chronique et (iv) l'hypoglycémie réactionnelle exacerbée par l'alcool. L'hypoglycémie de jeûne est due essentiellement à l'inhibition de la néoglcogenèse par l'alcool qui agit directement et qui altère également le potentiel redox intracellulaire. Cependant, les mécanismes compensatoires responsables de l'homéostase du glucose peuvent également être altérés. L'alcool augmente aussi l'action hypoglycémiante de l'insuline et des sulfonylurées. La réaction de l'insuline au glucose peut être intensifiée par la présence d'alcool dans les cellules bêta pancréatiques.

The ability of alcohol to produce hypoglycemia has been recognized since 1941[1]. Only in the past 20 years has attention been paid to the various mechanisms by which hypglycemia occurs. Although its occurrence is world wide, most cases occur either in underfed communities or where alcohol consumption is high. Appreciation of the setting in which this syndrome occurs should prompt a vigorous clinical and laboratory assessment of potential victims that present to the Emergency Department with a variety of often unexplained symptoms and signs.

There are four clinically and elologically distinct types of alcohol induced hypoglycemia[2]. These include (i) fasting hypoglycemia (ii) alcohol potentiation of drug induced hypoglycemia (iii) essential reactive hypoglycemia in chronic alcoholics and (iv) alcohol promoted reactive hypoglycemia. The various mechanisms by which ethanol (alcohol) causes hypoglycemia are summarized in Figure 1. A discussion of these various mechanisms and illustrative case presentations are provided.

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(I) ALCOHOL INDUCED FASTING HYPOGLYCEMIA.

This is the best recognized and most studied of the four varieties. The patient, when first seen, is usually comatose. Most patients are chronic alcoholics and hypoglycemia typically develops 6-24 hours after ingestion of alcohol. Physical signs include a rapid bounding pulse, profuse perspiration, hyperventilation and hypothermia. Alcohol may or may not be detectable in the blood in these patients; laboratory abnormalities of liver dysfunction may also be present. Plasma insulin levels are characteristically low.

Case History

A 44 year old male was brought into the emergency room in a semicomatose state having been found at home by a friend. The patient has been a heavy drinker for 12 years. He had stopped drinking for twenty-four hours prior to his arrival in hospital. He had not eaten for a couple of days because of nausea, vomiting and abdominal pain. History obtained after recovery of the patient did not suggest ingestion of drugs or toxins. Physical examination revealed a malnourished man who responded minimally to verbal and pain stimuli. He was markedly diaphoretic and there was a smell of alcohol on his breath. Heart rate was 115/min, blood pressure 110/70 mm. Hg. and respirations were 28/min. Abdominal examination revealed an enlarged liver. Blood was drawn for glucose estimation prior to the patient being given a bolus of 100

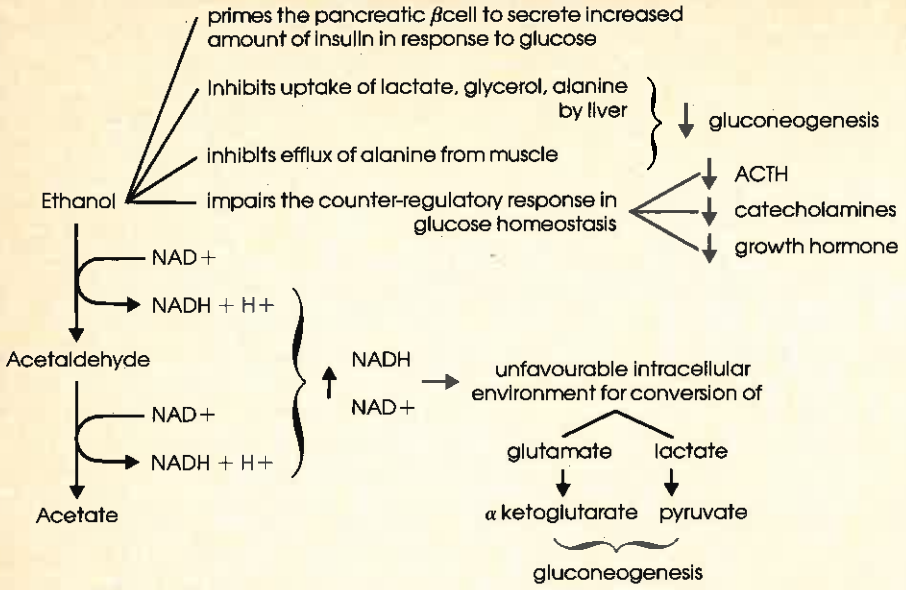

Figure 1 *Legend:* Mechanisms by which ethanol causes hypoglycemia.

grams of dextrose in 200 ml. of water. The serum glucose was reported as 20mg/dL. Blood ethanol was not detected. A qualitative 'toxicology screen' on urine was negative. The patient was given glucose, multivitamins and saline intravenously and discharged 48 hours later.

Discussion

The above case demonstrates the most well known and most thoroughly investigated variety of alcohol induced hypoglycemia. However, of the four types, it is probably the least common. This is fortuitous since significant mortality occurs in these patients[3]. The hypoglycemia usually develops in malnourished or starved individuals whose glycogen stores are depleted. It is widely believed that this variety of alcohol induced hypoglycemia is due exclusively to the inhibition of gluconeogenesis[4].

Apart from some minor metabolic routes e.g. glucuronidation, ethanol is oxidized primarily by three distinct pathways[5]; a) in the cytosol, alcohol dehydrogenase catalyses the oxidation of ethanol to acetaldehyde and acetate, b) catalase in the presence of hydrogen peroxide catalyses the oxidation of ethanol to acetaldehyde and, c) a microsomal ethanol oxidizing system (MEOS) which also oxidizes ethanol to acetaldehyde. The major ethanol oxidizing system, ADH, produces 2 moles of reduced nicotinamide adeine dinucleotide (NADH) per mole of ethanol oxidized. During ethanol metabolism, the rate of production of NADH exceeds its rate of mitochondrial oxidation resulting in an increased NADH: NAD+ ratio in the cytosol. This shift in redox potential decreases the availability of pyruvate, glycerol and alanine for gluconeogenesis by creating an unfavourable intracellular environment for their oxidation[6]. An increased NADH: NAD+ ratio is also an effective suppressant of the tricarboxylic acid cycle[7].

In addition to the alteration of the above intracellular processes, ethanol inhibits gluconeogenesis at two other sites. In the presence of ethanol, uptake of lactate, alanine and glycerol by the liver is reduced (8-10). Moreover, the efflux of alanine from muscle is reduced, thereby decreasing the availability of alanine for the formation of new glucose[11]. Also, impaired glycogenolysis in response to hyperglucagonemia may play a minor role in the pathogenesis of hypoglycemia[12].

Whereas the above effects of ethanol on intermediary metabolism are probably the most important factors in the pathogenesis of fasting hypoglycemia, other factors such as impaired counter-regulatory humoral responses may also play a role. A deficient ACTH response to hypoglycemia occurs in over 25% of chronic alcoholics[2]. Contrary to this, the plasma ACTH and cortisol levels increase in normal individuals in response to ethanol[13]. This has been attributed to a direct stimulatory effect of ethanol on the hypothalamic-pituitary-adrenal axis[14]. Impaired growth hormone (GH) secretion in alcohol induced hypoglycemia had been confirmed in a number of studies[15,16] and is probably related to the elevated levels of free fatty acids commonly seen in this condition[17]. The adrenergic response to hypoglycemia may be absent since repeated exposure to ethanol may deplete catecholamine stores[18].

It is important to note that persons consuming a low calorie diet[19] children[20] and patients with diabetes, Addison's disease and thyrotoxicosis are especially susceptible to alcohol-induced fasting hypoglycemia whereas obese individuals are usually resistant[21].

This type of hypoglycemia responds immediately to glucose infusion[22]. An important point worth noting is that most patients like the one presented above may not have detectable levels of alcohol at the time of hypoglycemia. In fact, hypoglycemia may occur whilst blood ethanol levels are declining[22]. A probable reason for this is that the altered redox state can persist in the livers of alcoholics for up to 4-5 days[23].

(II) ALCOHOL POTENTIATION OF DRUG INDUCED HYPOGLYCEMIA.

The development of severe hypoglycemia following the ingestion of alcohol by insulin-treated diabetics was first described by Arky et al[24]. The mechanism by which alcohol causes hypoglycemia in these individuals is not known. It is however believed to be independant of its ability to suppress gluconeogenesis.

Case History

A 42 year old man with known insulin dependant diabetes mellitus was brought into the Emergency Room deeply comatose. The patient was also a chronic alcoholic as evidenced by several previous hospital admissions for treatment of alcohol related problems. Physical examination revealed a comatose man responding only to pain stimuli. His pulse rate was 112/min; BP 110/60mm Hg, and respiration 28/min. Relevant laboratory studies disclosed a serum glucose of 0 mg/dL (confirmed on a separate instrument) and a blood ethanol of 40 mg/dL. A qualitative toxicology screen was negative. No barbiturates were detected in serum. The patient was given several boluses of intravenous glucose. No change in his mental state occurred. Eventually the serum glucose rose to 110/dL. At the same time there was some evidence of neurological deficit (confusion and hyperreflexia). There was no clinical

evidence of any residual neurologic dysfunction one week later. It was believed that the neurologic problems were secondary to the protracted hypoglycemia.

Discussion

The above case, similar to those previously reported[24], demonstrates that alcohol may augment the hypoglycemic action of insulin. It has been shown in normal volunteers that alcohol does not alter the rate of the insulin induced decline in serum glucose but delays that phase of rebound of serum glucose levels back to normal. This delay in the "rebound" phase is thought to be related to inadequate stores of catecholamines[18] and an impaired growth hormone response induced by alcohol[16].

Patients on sulfonylureas who combine its use with that of alcohol also develop hypoglycemia[25]. However, the effect is less dramatic than the one seen with insulin. This may be related to the fact that patients taking sulfonylureas for the treatment of diabetes also tend to be obese. This may protect them from fatal hypoglycemia. Alcohol also potentiates the fall in blood glucose that occurs with exercising in the cold[16]. This is probably related to further enhancement of peripheral glucose utilization by alcohol.

(III) "ESSENTIAL" REACTIVE HYPOGLYCEMIA IN ALCOHOLICS".

It is generally recommended that patients with "essential" reactive hypoglycemia should not drink alcohol. Because the criteria employed for the diagnosis of essential reactive hypoglycemia are poorly documented, it is possible that this phenomenon occurs no more frequently in chronic alcoholics than it does in the normal population. A few studies, however, have documented the increased occurrence of symptomatic reactive hypoglycemia in chronic alcoholics[27,28] The hypoglycemia in these studies was not accompanied by an excessive rise in serum insulin and the expected rise in serum cortisol did not occur. The exact mechanism responsible for the reactive hypoglycemia is not known.

(IV) ALCOHOL PROMOTED REACTIVE HYPOGLYCEMIA

Alcohol, in addition to its previously described actions on intermediary metabolism has the ability to increase insulin secretion in response to an oral glucose load. Thus, the tendency to develop reactive hypoglycemia is enhanced. Food does not protect against the reactive hypoglycemia induced by alcohol. In such patients, a meal consisting of easily digestible carbohydrate may make them more susceptible to alcohol induced reactive hypoglycemia[2].

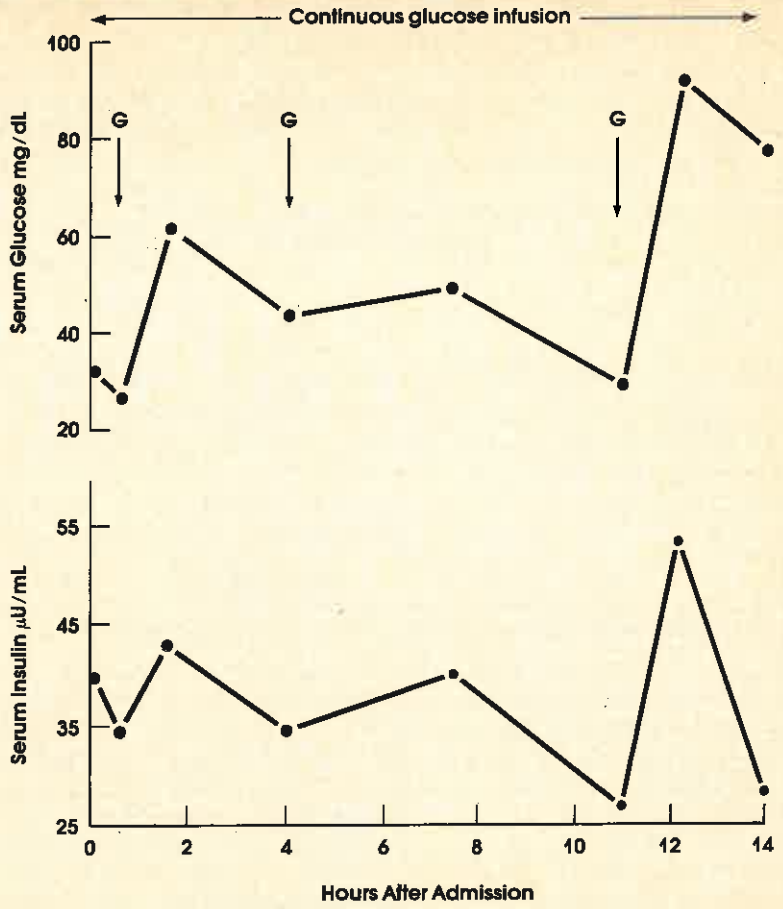

Figure 2

Legend: Serum glucose and insulin levels in Case 3 in the first 15 hours after admission. G represents boluses of intravenous glucose given during the first 14 hours after admission. Each bolus contained approximately 100 G of glucose. Upper limit of normal for plasma insulin is up to 15 mU/ml.

Case history

A 58 year old man was brought into the emergency room after being found comatose at his home by a friend. Physical examination revealed an obese man responding only to pain stimuli. No other abnormalities were found. Subsequent history obtained after recovery revealed that he had eaten little in the past two days and had drunk mainly alcohol containing beverages. The last event he remembered before becoming comatose was that he had a cola drink. Serum levels of glucose and insulin in the first 15 hours after admission are shown in figure 2. Massive amounts of glucose were required to bring the serum glucose level to normal. The patient was subsequently fasted to rule out an insulinoma. The results for serum glucose and insulin were within normal limits.

Discussion

Ethanol has the capacity to potentiate the action of glucose in stimulating insulin secretion[29]. The pancreatic beta cells are primed after exposure to alcohol and secrete greater amounts of insulin in response to glucose. Kuhl et al showed that the pancreatic beta cells must be exposed to ethanol for at least four hours before the priming effect becomes manifest[30]. However, Marks et al showed that this length of prior exposure is not necessary[31]. The above potentation of insulin secretion by alcohol is observed only when glucose, arginine and tolbutamide are used as stimuli[30].

There are some important clinical implications of the above phenomenon. Normally, the insulin

levels are appropriately suppressed with hypoglycemia and the response to intravenous glucose is immediate (Case 1). If the insulin levels are abnormally elevated as occurred in Case 3, increased peripheral uptake of glucose occurs[32]. Massive amounts of glucose may then be required to bring the serum glucose level to normal. Also an erroneous diagnosis of an insulinoma may be made in these patients since the calculated immunoreative insulin to glucose ratio (IRI/G) is well above the upper limit of normal and into the range seen in patients with insulinomas[33].

From a social standpoint, a number of individuals in preparation for the drive home will sober up with a cup of sweetened coffee. Because of the subsequent probability of severe hypoglycemia occurring, this may prove to be catastrophic. Furthermore, the hypoglycemia will also enhance the clinical features of alcohol intoxication[34]. Finally nocturnal hyperinsulinemia leading to lower serum glucose levels can occur in individuals who take alcohol with their evening meals[35].

In conclusion, it is important to recognize the various distinct types of alcohol induced hypoglycemia and the different clinical presentations. Symptoms produced by hypoglycemia are easily confused with those of alcoholic intoxication and often occur simultaneously. It is important to avoid attributing progressive loss of consciousness to deepening alcoholic stupor.

Typically the alcoholic who develops hypoglycemia will be malnourished and will have been drinking for several hours or days prior to the onset of hypoglycemia. However, weekend drinkers can also develop hypoglycemia and ethanol may be a potentiating factor in insulin dependant diabetics and patients taking oral sulfonylureas.

In most cases, intravenous glucose will reverse the neurologic manifestations. Nevertheless, the mortality for alcohol induced hypoglycemia remains high[12] and if possible, the patient should be persuaded to reduce his alcohol intake and eat more regularly. The emphasis is on making an early diagnosis and to provide appropriate management and follow up.

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Calcium Channel Blockers in the Emergency Room

by Robert J. Brison, M.D.

Abstract

Calcium Channel Blockers are being used in the treatment of cardiovascular disorders in the inpatient and outpatient population with accelerating frequency. The emergency physician must become aware of their actions, interactions and adverse effects as more outpatients are prescribed these drugs. The uses, potential uses and areas of possible research into these drugs' actions by the emergency physician are discussed. An approach to management of an overdose of these drugs is described.

Resume

On utilise de plus en plus les antagonistes du calcium pour traiter les atteintes cardiovasculaires tant chez les malades hospitalisés que chez les malades de consultation externe. Le médecin d'urgence doit se familiariser avec les actions, les interactions, et les effets nocifs de ces médicaments qu'on prescrit de plus en plus en consultation externe. Cet article présente les indications, les indications potentielles de ces médicaments de même que les domaines de recherche possibles sur leur action par le médecin d'urgence. On décrit la façon de traiter une intoxication aux antagonistes du calcium.

Calcium Channel Blockers (C.C.B.'s) have enjoyed a tremendous surge of popularity in the treatment of cardiovascular disease and disorders of smooth muscle. Their accelerating use brings with it a need for a greater understanding of these drugs by Emergency Physicians.

C.C.B.'s are not new drugs. They have been used extensively in Japan and Europe for a decade, primarily in the treatment of variant angina. The three predominant C.C.B.'s currently in use include verapamil, nifedipine and diltiazem. In Canada, verapamil and nifedipine are both available in oral and intravenous preparations. At present, diltiazem is available only as an experimental drug.

At the present time, these are infrequently prescribed to outpatients by the emergency physician. Their widespread use, however, requires that their actions, interactions and adverse effects be understood. The C.C.B.'s already have an established role in the acute treatment of supraventricular arrhythmias. There are a number of exciting new potential uses for C.C.B.'s in the Emergency Room. These include cerebral resuscitation and preservation, decreasing myocardial infarct size and decreasing the incidence of sudden death after myocardial infarct. The management of an overdose of these agents must also be anticipated.

Ion Channels

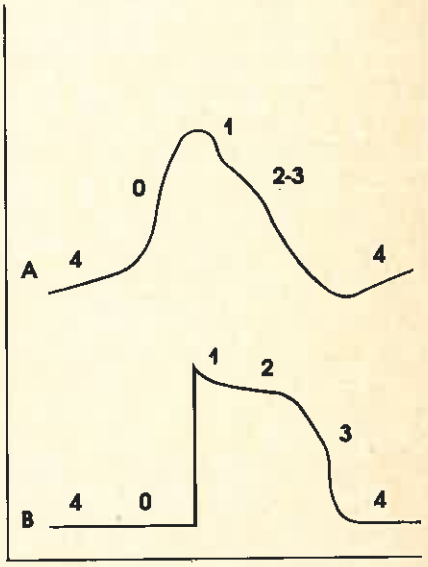
C.C.B.'s act by inhibiting the transmembrane flux of calcium through the "slow" channel during the transmembrane action potential. This effect seems most pronounced, and has been best studied, in the cardiac conduction system, cardiac muscle and vascular smooth muscle. In these tissues, there are two predominant ion channels; the fast, sodium channel and the slow, calcium channel.

The sodium channel regulates the rapid influx of sodium ions into the cell during phase 0 depolarization. It is the ion channel which generates the rapid upstroke in the action potential of atrial and ventricular muscle and the His-Purkinage system. These tissues conduct impulses relatively rapidly and do not normally possess automaticity. (Figure 1)

The slow, calcium channel is responsible for the action potential in the sinoatrial (S.A.) node and the atrioventricular (A.V.) node. These are tissues with slower rates of phase 0 depolarization, spontaneous phase 4 depolarization (automaticity) and long refractory periods. In fast channel contractile tissues, calcium influx via the slow channel is responsible for maintaining the plateau phase of the action potential as well as initiating muscle contraction through excitation-contraction coupling.

The sodium channel is retarded to a much greater degree than the calcium channel by disease or ischaemia. The calcium channel may then become the

Figure 1



Time

Representative Action Potentials From:

A Calcium Channel Dependent Tissue (SA Node), and

B Sodium Channel Dependent Tissue (Ventricular Muscle Fibre).

* ["Direct refers to the drug's effect on an isolated tissue preparation devoid of reflex input.]

predominant channel in the initiation of the action potential giving these cells a degree of automaticity. This may play a role in the development of ectopic contractions.

The interplay of calcium and the calcium ion channels has other important effects. Smooth muscle tone, particularly vascular smooth muscle of the various arterial beds, is dependant on alterations in the influx of calcium. The intracellular actions of catecholamines are largely mediated by calcium. Adrenergic agonists are thought to work by opening a great number of calcium channels resulting in increased calcium influx. Uncontrolled influx of calcium leads to cell injury and death. Inappropriately high intracellular levels of calcium due to leaky cell membrances appears to be a major cause of the extensive damage seen following reperfusion of ischaemically injured myocardium. The interaction of C.C.B.'s with these calcium dependant actions will be illustrated below.

Actions of the Calcium Channel Blockers

Although nifedipine, verapamil and diltiazem each work by antagonizing calcium influx, they have quite different molecular structures [1] and affect the calcium channels of various tissues differently. This suggests some structural variation in the calcium channel receptor sites in different tissues. [2] It is understandable then that nifedipine, verapamil and diltiazem have differing therapeutic uses and differing side effects. [3]

All of the calcium channel blockers have the following actions:

i Direct* Negative Chronotropic Effect (decrease in S.A. node discharge)

ii Direct Negative Dromotropic Effect (prolonged refractory period of A.V. node)

Figure 2

Direct Effects of Nifedipine, Verapamil and diltiazem

	Nifedipine	Verapamil	Diltiazem
Negative Chronotropic Effect	–	+++	+
Negative Inotropic Effect	+	+++	++
Peripheral Vasodilatory Effect	+++	++	+
Reflex Increase in β-adrenergic Tone	+++	++	+

iii Direct Negative Inotropic Effect

iv Decrease in Vascular Smooth Muscle Tone

v Indirect or Reflex Effects

The net effect of these actions differs for each of nifedipine, verapamil and diltiazem. This is also dependant to some degree on the dosage used.

In the usual clinical dosages [Table 1], the direct effects of these agents are as follows: [Figure 2] (These dosages are generally the doses required to dilate the coronary vascular bed.)

- The negative chronotropic and dromotropic effects are seen most with verapamil. Diltiazem is less potent and nifedipine has little or no effect on the S.A. or A.V. nodes.
- Negative inotropic effects are also most seen with verapamil. Diltiazem is again less potent and nifedipine has the least effect on cardiac contractility.
- Nifedipine is by far the most potent in producing peripheral vasodilation by diminishing vascular smooth muscle tone. Verapamil is less potent here. Diltiazem has little effect peripherally.
- The reflex response is primarily a baroreceptor-mediated increase in β-adrenergic tone secondary to peripheral vasodilation. This results in an increase in heart rate and in contractility. It may even override the direct negative inotropic and chronotropic effects. In fact, this is usually the case with nifedipine which, as mentioned above, is the most potent vasodilator of the three.

The *net* effect of the C.C.B.'s varies somewhat from person to person and varies depending on the state of the myocardium and the drug dosage used. Their net effects on heart rate, peripheral resistance and cardiac output are presented in Figure 3. Net effect on contractility is variable with all three drugs.

Proven Therapeutic Indications

1. Variant Angina:

The coronary vasospasm associated with variant angina responds well to C.C.B.'s. All three drugs have been shown to greatly decrease frequency and severity of angina, as well as decreasing nitroglycerin use. [3-6] Furthermore, the frequency of ventricular tachyarrhythmias seen with this syndrome is diminished using C.C.B.'s.

Diltiazem may prove to be the best choice for this disorder, as it seems to be more selective in affecting coronary vascular smooth muscle with less significant effects on peripheral vessels and the cardiac conduction system.

2. Angina Pectoris:

All three drugs are effective in treating effort-induced angina. They appear to be effective even in the absence of coronary spasm. These drugs offer an alternative mode of therapy, particularly in those patients in whom nitrates and β-blockers are either not tolerated or have been unsuccessful. One must be wary of increasing A.V. nodal conduction block when combining β-blockers with C.C.B.'s.

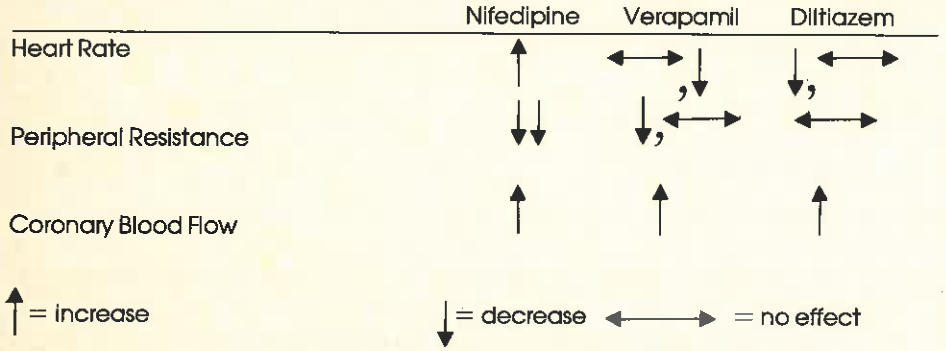
TABLE I

Clinical Doses and Half-Lives of Nifedipine, Verapamil and Diltiazem

Agent	Usual clinical Dose	Half-Life
Nifedipine	10-60 mg p.o. q.6h. 0.005 - 0.0015 mg/kg IV (available sublingually	4 hrs.
Verapamil	40-260 mg p.o. q.6h. 0.075 - 0.15 mg/kg IV	3-7 hrs.
Diltiazem	60-120 mg p.o. q.6h. 0.075 - 0.15 mg/kg IV	4 hrs.

*An IV bolus should be given slowly over several minutes.

Figure 3
Net Cardiovascular Effects Usually Seen With the Common Calcium Channel Blockers.



C.C.B.'s are usually tolerated in angina patients with C.O.P.D. in whom β-blockers may have harmful effects.

3. Dysrhythmias:
Verapamil is well known now for its effectiveness in treating paroxysmal atrial tachycardia (P.A.T.). Nifedipine has no antiarrhythmic effect. Diltiazem probably does, but has not been studied sufficiently. Verapamil is also useful in slowing the ventricular response in atrial flutter and atrial fibrillation. However, conversion to sinus rhythm is infrequent.

Caution is required using C.C.B.'s in the Wolff-Parkinson-White patient with supraventricular tachycardias. Verapamil has little or no effect on conduction through the anamalous tract. Therefore, in the W.P.W. patient with P.A.T., verapamil only retains its full effectiveness if the antegrade impulse to the ventricles is via the A.V. node. In the W.P.W. patient with atrial flutter or fibrillation, verapamil will increase the A.V. node block and enhance conduction via the anamalous pathways. This may result in either no change, or an increase in the ventricular response. Ventricular fibrillation might be more easily produced.

Ventricular ectopic beats and dysrhythmias associated with ischaemia may also respond to C.C.B.'s. This is more likely due to an increase in coronary blood flow than to a direct decrease in ventricular automaticity.

4. Other Indications:
Nifedipine and verapamil have both been shown to be at least as effective as β-blockers in the treatment of hypertrophic cardiomyopathy. C.C.B.'s offer some limited use in pulmonary hypertension. Although the effect is limited, initial reports are encouraging in the treatment of this otherwise refractory disease. The potent vasodilatory effects of nifedipine may be useful in the management of some patients with hypertension and congestive heart failure.

Potential Therapeutic Indications

There may be many exciting new indications for C.C.B.'s of special interest to the emergency physician.

1. Acute Myocardial Infarction:
C.C.B.'s may be useful both in limiting infarct size and in decreasing the frequency of life-threatening dysrhythmias. [8] There are a number of possible mechanisms for this. In the setting of ischaemia, ATP stores within the myocardial cell are depleted, allowing calcium influx. Excessive intracellular calcium is harmful to cellular metabolism and, if not corrected, will lead to cellular death. A C.C.B., by blocking the calcium channel, may retard this process. Secondly, their effects on the coronary circulation could increase oxygen delivery by opening up collateral supply vessels and also by decreasing coronary spasm, if present. Finally, C.C.B.'s may decrease myocardial oxygen requirements by decreasing afterload.

Certainly a decrease in infarct size has been adequately demonstrated for nifedipine and verapamil in the animal model after ligation of a coronary vessel. In practice, the potential of these drugs to hamper A.V. conduction, decrease contractility and drop blood pressure in the presence of acute ischaemia must be appreciated.

2. Cerebral Resuscitation:
This is perhaps the most exciting potential use of C.C.B.'s in the emergency room. By similar mechanisms to those cited above for myocardial preservation, C.C.B.'s may have a role in C.N.S. resuscitation from anoxia. These hopes have been supported by animal studies. [9] C.C.B.'s might also be an answer to the treatment of the hazardous vasospasm of subarachnoid hemorrhage.

3. Other Vasospastic Disorders:
Promising results are appearing in the treatment of Raynaud's and migraine. Other theoretical indications might be ischaemic bowel disorders and intermittent claudication.

4. Smooth Muscle Disorders:
Treatment of smooth muscle disorders other than of vascular smooth muscle is possible. These might include dysmenorrhea, irritable bowel and esophageal motility disorders.

Adverse Effects

Although initial studies suggested that nifedipine and verapamil were well tolerated orally, since being released for clinical use, their widespread use has shown that the frequency of side effects is much higher than the initial indications of about 10%.

Common side effects seen with all three agents include flushing, headache, postural hypotension, dependant leg edema and fatigue, with the first four symptoms most commonly seen with nifedipine. Verapamil is also associated with A.V. block and constipation. Diltiazem appears to be the best tolerated.

Precautions

Verapamil and diltiazem should be used with caution in the presence of an altered conduction system. Sinus bradycardia and arrest may be produced in sick sinus syndrome. A.V. conduction block may be produced or increased with C.C.B.'s in the presence of A.V. nodal disease or when used in the presence of β-blockers, digoxin and some antiarrhythmics (procainamide and disopyramide). These concerns generally do not apply to nifedipine. Verapamil may increase serum digoxin levels.

In the presence of a failing ventricle, nifedipine may increase cardiac performance by decreasing afterload

and by a reflex positive inotropic and chronotropic response. β-blockade would prevent this favourable response.

Nifedipine will augment the side effects of nitrates associated with vasodilation (headache, flushing, postural hypotension).

Giving verapamil intravenously as a *rapid* bolus in treating P.A.T. will increase the likelihood of hypotension, bradycardia, sinus arrest, A.V. block and ventricular fibrillation.

The Poisoned Patient

There are, as yet few reported cases of C.C.B. ingestion overdose. However, the opportunity to see such a case is increasingly likely. The treatment of such an overdose will be in part theoretical and will vary with the drug taken.

C.C.B.'s are 90% absorbed in the gastrointestinal tract and then are 80-90% protein bound in the serum. Their clinical doses and half lives are noted in Table 1. C.C.B.'s are in part metabolized by the liver and excreted mainly in the urine. Presumably, the use of ipecac, gastric lavage, activated charcoal and magnesium sulphate would be appropriate initially.

The specific challenges of the C.C.B. overdose would be in dealing with hypotension secondary to any or all of the following: S.A. nodal block, A.V. nodal block, decreased contractility and decreased peripheral resistance.

Reversing the electrophysiologic effects of the C.C.B.'s involves getting more calcium into the cells. Adding exogenous calcium may be of short term value, but the ultimate answer is to open more calcium channels. As the calcium channels are cyclic-A.M.P. dependant, drugs which will increase cyclic-A.M.P. would be appropriate. These include β-adrenergic agonists and glucagon. Atropine may also be needed in the case of hemodynamically significant bradycardia and/or in A.V. block.

Management of the profound decrease in peripheral resistance associated with hypotension may require a fluid load. Providing a fluid load, as well as increasing peripheral resistance could be achieved with the Military Anti-Shock Garment (M.A.S.T.). [10]

An adrenergic agent may be required to counteract the peripheral vasodilation and the negative inotropic and chronotropic effects of the C.C.B. overdose. The hypotension of a nifedipine overdose would likely be due to peripheral vasodilation and be best treated with an α-agonist. The negative inotropic and chronotropic effects of verapamil and diltiazem could be treated with a β-agonist, such as isoproterenol. If peripheral vasodilation was also a factor with verapamil and diltiazem, an agent with combined α and β effects, such as dopamine or epinephrine, would be indicated.

Summary

Calcium channel blockers are finding widespread use in the outpatient population for the treatment of cardiovascular disorders. Their increasing use dictates that emergency physicians should become familiar with their actions, interactions and adverse effects.

The final definition of the indications for calcium channel blockers will take more research. The initial contact of the emergency physician with critically ill patients will allow him to study the potential of these agents in myocardial and cerebral resuscitation and preservation.

Calcium channel blockers may have a greater therapeutic role in the future. As the structures of the calcium channels are better understood, "second generation" calcium channel blockers will be developed. These may be even more specific and varied in their modes of action than the "first generation" drugs, which are only now beginning to be understood.

TABLE II

Indications For Calcium Channel Blockers

Proven Indications	Potential Indications
Variant Angina	Myocardial Cell Preservation in Acute MI
Angina Pectoris	Decrease in Ventricular Arrhythmias with Myocardial Ischaemia
P.A.T. (Verapamil)	Decrease in Incidence of Cardiac Sudden Death
Atrial Flutter and Fibrillation with rapid ventricular response	Cerebral Resuscitation
Hypertrophic Cardiomyopathy	Subarachnoid Hemorrhage
Pulmonary Hypertension	Migraine
Systemic Hypertension	Raynaud's
Congestive Heart Failure	Intermittent Claudication
	Dysmenorrhea
	Irritable Bowel
	Esophageal Motility Disorders

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Noticeboard

Required 'Medical Director' Emergency

A Medical Director is required for the Emergency and Ambulatory Department of a 310 bed acute treatment general hospital. The Department deals with approximately 62,000 cases a year and is the 24 hour emergency centre for the Region.

There are presently 5 full-time with the support of part-time medical postions in the Department, with back-up support of specialists in all available specialties and diagnostic modalities.

The successful candidate will be a physician eligible for licensure in Ontario with experience in Emergency Medicine.

It would be considered an asset for the applicant to have had previous administrative and teaching experience including formal training in Emergency Medicine as the Director will assume management responsibility for all medical matters pertaining to emergency services and will provide training to associated medical, nursing and paramedical staffs.

Please forward written application and inquiries in confidence to:

Chief of Staff
Sudbury General Hospital
700 Paris Street
SUDBURY, Ontario
P3E 3B5
Telephone: (705) 674-3181
Extension: 170

Emergency Physician

The Division of Emergency Medicine, Department of Surgery at Queen's University, invites applications for a full-time faculty position in emergency medicine. Applicants should have at least three years of postgraduate training relevant to emergency medicine and experience in emergency medicine teaching and research. Applications should be addressed to:
Dr. L.E. Dagnone, Chairman, Division of Emergency Medicine, Department of Surgery, Queen's University, Kingston, Ontario, K7L 3N6.
Closing date for applications May 1, 1983.

Emergency-Intensivist Physician

Emergency-Intesivist physician required for low volume Emergency Department in a specialty hospital affiliated with McMaster University to form a group, along with four others, to provide services to both Emergency Department and in-hospital patients.

Requirements

- CPSO general license and CMPA membership or equivalent.
- Training experience (two years post M.D.) in Emergency or Critical Care Medicine required.
- Successful attendance at ACLS within last three years desired.
- Preference will be given to those with Royal College Fellowship or Family Medicine certification with competence in Emergency Medicine.

Additional inservice training in Critical Care Adult Medicine will be provided.

Remuneration

Contract will provide for a level of guaranteed remuneration plus an additional billings capability, four weeks vacation, two weeks study leave, and a fringe benefit package.

Apply in writing to:
Dr, Frank Baillie
Head of Section, Emergency Services
Chedoke-McMaster Hospitals
Box 2000, Staion 'A'
1200 Main Street West
Hamilton, Ontario
L8N 3Z5

Emergency Medicine Academic Position

The Department of Emergency Medicine, Victoria Hospital, LONDON, Ontario, has an opening for a full-time Emergency Physician, commencing July 1, 1983.

The successful applicant will be eligible for Certification by the Royal College of Physicians and Surgeons of Canada in Emergency Medicine, and should have an interest in Emergency Medicine teaching and research.

Salary and expense allowances are as per University of Western guidelines for Emergency Medicine.

Please send letter of application with curriculum vitae and the names of three references to:

Dr. R.V. Gerace, Chairman
Emergency Physician Selection Committee
Department of Emergency Medicine
Victoria Hospital
LONDON, Ontario

Meetings to note

Director of Emergency Services

The Mount Sinai Department of Family and Community Medicine is currently looking for a Director of Emergency Services. The Director of Emergency Services should be responsible for the organization and functioning of the Emergency Service and for all the teaching and research activity in the Emergency Service and for the general supervision of clinical work.

The candidate should be eligible for the certification in Emergency Medicine either from the College of Family Practitioners or the Royal College of Physicians and Surgeons.

The Director will hold an academic appointment in the Department of Family and Community Medicine, University of Toronto.

Send resume or for more information write or call Dr. Yves Talbot, Family Practitioner-in-Chief, Mount Sinai Hospital, 600 University Avenue, Toronto, Ontario, M5G 1X5, Telephone number is (416) 596-4633.

Faculty Emergency Physician
McMaster University

To provide service and academic activity by working a moderate number of hours 'on-line' in the Emergency Unit at McMaster Division of Chedoke-McMaster Hospital. The academic activity includes providing education to allied health professionals and undergraduate and postgraduate medical students. Additional academic activities will be encouraged in research and/or development of improved pre-hospital care.

Applicants should be 2 years post M.D. with specific experience in Emergency Medicine, including A.C.L.S., A.T.L.S. or certification in Emergency Medicine from the Royal College or Canadian College of Family Medicine.

Applications should be submitted to:

Dr. C.A. Moore
Professor & Chairman
Department of Family Medicine
McMaster University Medical Centre
1200 Main Street West
Hamilton, Ontario, L8N 3Z5
Tel: (416) 525-9140 ext. 2547

First Canadian Emergency Underwater Conference

Maui — Hawaii November 4-18 1983.
Presented by The Emergency Medicine Section of the Hamilton Academy of Medicine.
Contact Chairmen:

Dr. L.A. Hargot,
McMaster University Medical Centre,
1200 Main Street West,
Hamilton, Ontario L8N 3Z5
Telephone (416) 525-9140 ext. 2964.

UNIVERSITY HOSPITAL

EMERGENCY DEPARTMENT DIRECTOR

The University Hospital and the Department of Family Medicine of the University of Saskatchewan invites applications for the position of Director, Department of Emergency Medicine.

Applicants should have, or be eligible for, the Certificate in Emergency Medicine of the Royal College of Physicians and Surgeons or the College of Family Physicians of Canada, and have had experience in emergency medicine, teaching in the emergency setting, and administration of a department.

Major responsibilities will include the provision and coordination of patient care, undergraduate and postgraduate teaching, and administration of the department.

Salary will be negotiable depending on academic attainment and experience, and is complemented by a benefit package including:

- 4 weeks paid holiday
- 1 week paid educational leave
- Group insurance and other benefits

Application accompanied by a curriculum vitae and names of two references should be forwarded to:

Mr. I. D. Sutherland
Assistance Executive Director
— Medical Support Services
University Hospital
Saskatoon, Saskatchewan
S7N 0X0