TAMPA BAY

EMS

JOURNAL

Volume 1, Number 1 July-September, 1987

PREMIERE ISSUE

A Publication of the Acute Care Foundation
A Non-Profit Academic Organization Serving the Emergency and Critical Care Professional

Continuing Education Programs

presented by the

ACUTE CARE FOUNDATION

1987 AGENDA

The Acute Care Foundation is proud to announce its offering of the following academic programs during 1987. Specific dates, times and locations will be announced in direct mailings to Tampa Bay area EMS agencies and hospitals within the near future.

SYMPOSIUM ON PREHOSPITAL MEDICAL CONTROL

This one day symposium will hear dicussion and debate regarding the philosophies and methods for medical control and quality assurance in prehospital EMS systems. Abstracts of 1000 words or less are now being accepted from persons, groups or agencies interested in presenting a paper at this event.

SYMPOSIUM ON PREHOSPITAL CONTINUING EDUCATION

The unique logistics of prehospital EMS poses a significant challenge to inservice continuing education programs. This one day symposium will explore the continuing education needs of the veteran field clinician and how they differ from the paramedic and EMT student. Particular attention will be given to self-directed post-graduate education programs. Abstracts of 1000 words or less are now being accepted from persons, groups or agencies interested in presenting a paper at this event.

PROTOCOL ROUNDTABLE - ACUTE CONGESTIVE HEART FAILURE

The Protocol Roundatables are an on-going series of symposia which explore a specific emergency disorder with a distinguished panel of medical specialists, paramedics, emergency physicians and nurses. Their discussions will include a review of the pathophysiology, therapeutic objectives, current literature and local treatment protocols. This information will be used to synthesize a state of the art model protocol that may be used by EMS agencies and hospitals as a resource in their own respective protocol maintenance efforts. Partcipation of those attending these sessions is encouraged in the dialog.

Tampa Bay EMS Journal

A Jouranl of The Acute Care Foundation, Inc.
A Non-Profit Academic Organization Serving the Emergency and Critical Care Professional

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Tampa Bay EMS Journal
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Volume 1, Number 1
July-September, 1987

A Refereed Academic Journal For EMS

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EDITOR

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The refereed medical journal plays a vital role in medicine, providing a respected forum of research and academic debate. Acceptance of scholarly papers for publication requires compliance to standards of academic quality such as appropriate research study design, methodology, hypothesis testing and bibliographic referencing. A paper is first screened by a journal's editorial staff. Meeting with their approval, it is then sent out for a formal academic review by referees – highly respected researchers and clinicians in their particular disciplines. Their critique and commentary are published along with the response and closing comments of the authors, immediately following the original paper. Through this process, the true merits and significance of the paper to the discipline are explored.

Emergency Medical Services (EMS) are in a major transition. Administrative trends are shifting away from increasing the numbers of ambulances and critical care beds toward the refinement and sophistication of cost-effective services. Medically, there is a growing need for research and academics in EMS. The EMS community requires an interdisciplinary forum, encompassing the entire spectrum of EMS care from the field into the critical care units. The *Tampa Bay EMS Journal* is the first step in an innovative program of academics and continuing medical education for EMS.

The *Tampa Bay EMS Journal* is a unique publication. It provides EMS with a journal in a formal academic format. A major objective of this project is to catalyze development of much needed prehospital research and academic development. It is a regional publication, currently encompassing Hillsborough, Pasco, Pinellas, Polk, and Manatee counties. A regional scope permits discussions with references to specific agencies and hospitals. For example, a discussion of trauma care can address the encatchment areas of individual hospitals and the destination protocols of specific prehospital agencies. This approach will make the *Journal* an excellent forum in which regional concerns may be debated, where no such forum currently exists.

The project is being conducted under the auspices of a non-profit organization, the Acute Care Foundation. The Acute Care Foundation was started by a small group of local emergency medical technicians and paramedics wanting to develop collaboration between different agencies and hospitals, across political and territorial borders. This lack of interagency and prehospital-inhospital collaboration is a major obstacle to the academic growth of EMS. A key to resolving this problem is effective professional communication. It is a mechanism through which interagency collaboration may be developed.

FUNDING

A financial base to initiate this project will be sought through sponsorships, grants and donations. The recurring costs for printing and expenses associated with production of the *Journal* will be met with revenues from advertising. So long as it is financially practical, a specific target audience will receive the *Journal* without subscription fees. This subscription free target distribution includes all area prehospital EMS substations, their medical directors, administrators and training officers, emergency departments and critical care units, their head nurses and directors, in-service education departments, EMS training facilities, and the University of South Florida Colleges of Medicine, Nursing, and Public Health. Additional courtesy distributions will be made to area public officials, the State EMS Office, and EMS related professional organizations. This provides exposure of the *Journal* throughout the system. It assures a communication link. This approach also makes the *Journal* a very attractive advertising medium. Those persons having the major influences on purchasing decisions are within the target distribution. Paid subscriptions to the *Journal* will be available through membership in the Acute Care Foundation.

PUBLICATION LOGISTICS

Another unique aspect of the *Journal* is the technology used to produce it. The *Journal* uses "desktop publishing" tools to replace the traditional methods and expense of typesetting and paste-up. Draft pages are produced with a laser printer. Final pages are made using a new generation of phototypesetting devices. This equipment is currently borrowed and rented. Thus, access to the *Journal* staff is very limited. Acquisition of some components of this system will dramatically improve its production efficiency.

The articles are entered into personal computer word processors by each author or supervising contributing editor. The text is then transmitted via modem to a computer information network as electronic mail. The editorial staff then retrieves the articles into their personal computers for editing and review. After acceptance for publication, the articles are sent by electronic mail to a computer system used for electronic typesetting and layout. The final composed page information is again transmitted by modem to a phototypesetting service for production of camera-ready images. These logistics have helped to make it feasible to produce a publication of professional quality at a reasonable cost.

ADDITIONAL PROGRAMS

Access to computer information networks offering electronic mail is obtained by local call through hundreds of telephone exchanges in the U.S. and abroad. These networks also provide facilities for electronic forums that are sometimes referred to as bulletin board services (BBS) or special interest groups. Many such forums include general medicine and EMS. Negotiations will be conducted with these services to provide on-line publishing of general interest articles from the *Tampa Bay EMS Journal*, as is now done with many other magazines and newspapers. This will give immediate national and limited international distribution of the *Journal*. Through this electronic medium, discussions and debates in the *Journal* may continue on-line and be incorporated into subsequent printed issues. These networks can also facilitate live on-line medical conferences with participation of invited commentators. Formal publication of these on-line proceedings may follow in both the electronic and printed versions of the *Journal*.

The interagency collaboration initiated by the *Journal* may also be used to establish a network of area cable and microwave TV systems. This network will be used to broadcast continuing medical education programs and symposia for EMS. One of the first such programs planned for broadcast is the *Protocol Roundtable*, where panels of EMS clinicians and appropriate specialty physicians will draft specific emergency treatment protocols. This will include a review of current protocols of selected area agencies and an up-to-date review of the medical literature. Acquisition of protocol examples from outside the Tampa Bay area may be solicited through the computer BBS networks. The prototype protocols developed in the *Protocol Roundtable* will be published with complete bibliographic references. Agencies may then use the prototype and its bibliography as an aid in their own on-going protocol revision efforts. Programs on the EMS television network may also include rebroadcast of grand rounds, lectures, classes, and commercially produced education tapes. Negotiations will also be made to obtain satellite access for expansion into a nationwide EMS continuing medical education television network.

A program is under development to award continuing education units (CEU) to physicians, paramedics, nurses and EMT's for reading major *Journal* papers, viewing educational programs on the television network, or participating in the on-line computer conferences. For prehospital EMS, accreditation for CEU will be sought from individual medical directors, the Florida Association of EMS Medical Directors and the National Registry of Emergency Medical Technicians. Nursing CEU will be sought through collaborating hospitals and institutions. Continuing medical education credits for physicians will be sought through the Continuing Medical Education Office at the University of South Florida College of Medicine.

The *Tampa Bay EMS Journal* is a prototype in what is hoped to become a much larger network of sister publications for other regions throughout Florida and the U.S. These sister publications would share materials of general interest but retain exclusive publication of papers and proceedings of a primarily regional interest.

SUMMARY

The many components of this project, including printed and electronic publications, emergency medical television programming, symposia sponsorship, and CEU programs, will be coordinated by the Acute Care Foundation. Its objective is stimulation of formal academic activity in EMS with collaboration between the prehospital and inhospital sectors. Funding will be developed through grants, advertising, memberships and individual donations. The project is economically and logistically feasible through the use of new technology in personal computers and computer networks.

Participation is sought from EMS and critical care professionals at all levels - from the field, emergency departments, trauma centers, critical care units, continuing education departments and the academic community. The *Tampa Bay EMS Journal* and its associated projects are a reflection of the interest and dedication to EMS in this region. It requires your participation and financial support – consider it an investment in our EMS community.

Acknowledgement

The editorial staff would like to express their gratitude to the people that have contributed their time and resources to help initiate this project - Dr. Richard Connar, Chairman of the U.S.F. Department of Surgery, Dr. John Carr and Mr. Paul Higgins of the U.S.F. Department of Computer Sciences, Joe Massarelli and Helen Quinn of Florida Trade Publications in Dunedin. A special acknowledgement is given to Dr. George Watkins, and Dr. Alex Rosemurgy of the U.S.F. Department of Surgery for their unfailing support, guidance and inspiration.

Tampa Bay EMS Journal
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Volume 1, Number 1
July-September, 1987

Nifedipine

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Nifedipine is a calcium channel blocking agent gaining widespread acceptance in the treatment of hypertension and angina. It acts as an arterial vasodilator in coronary and peripheral vascular beds without major effects on cardiac contraction or impulse conduction. Continuing clinical investigations have consistently demonstrated the safety and efficacy of nifedipine in these situations, making it an increasingly important drug in emergency care.

THE ROLE OF CALCIUM IN MUSCLE CONTRACTION

A muscle cell is stimulated to contract by an electrical impulse from a nerve or an adjacent cell. The electrical stimulus sets off a chain of events, called depolarization, which moves specific ions in and out of the cell across its outer membrane.

In cardiac muscle cells, depolarization depends on the opening of special channels in the cell membrane for sodium and calcium ions (1,2). Once in the cell, calcium binds to troponin. When troponin is bound to calcium, the strands of muscle protein, actin and myosin, are free to link together. Actin and myosin strands may then slide together in an interleaving fashion (Figure 1). After depolarization and contraction, sodium and calcium are pumped back out of the cell. As calcium is pumped out, troponin is free to again wedge itself between actin and myosin, preventing linkage and contraction.

The vascular smooth muscle cell mechanisms are slightly different than those of cardiac muscle cells. Contraction of vascular smooth muscle appears to be dependent upon two different types of mechanisms (3). The first mechanism, electro-mechanical coupling, is mediated by electrically triggered calcium channels, which open in response to depolarization of the membrane. Extracellular calcium may then diffuse into the cell to initiate the contractile process. The second mechanism, pharmaco-mechanical coupling, involves a receptor site stimulation induced contraction that occurs without depolarization of the membrane. It results from the release of calcium retained in the cell, within the sarcoplasmic reticulum (3). This effect also results in an additional influx of extracellular calcium via the receptor-oriented channels on the outer cell membrane. The free intracellular calcium, brought there by either mechanism, results in binding of calcium to calmodulin. The calcium-calmodulin complex activates an enzyme, myosin light-chain kinase, that allows binding to occur between actin and myosin. These two separate but interwoven fibers, now linked, can then slide together to cause vascular smooth muscle contraction (4).

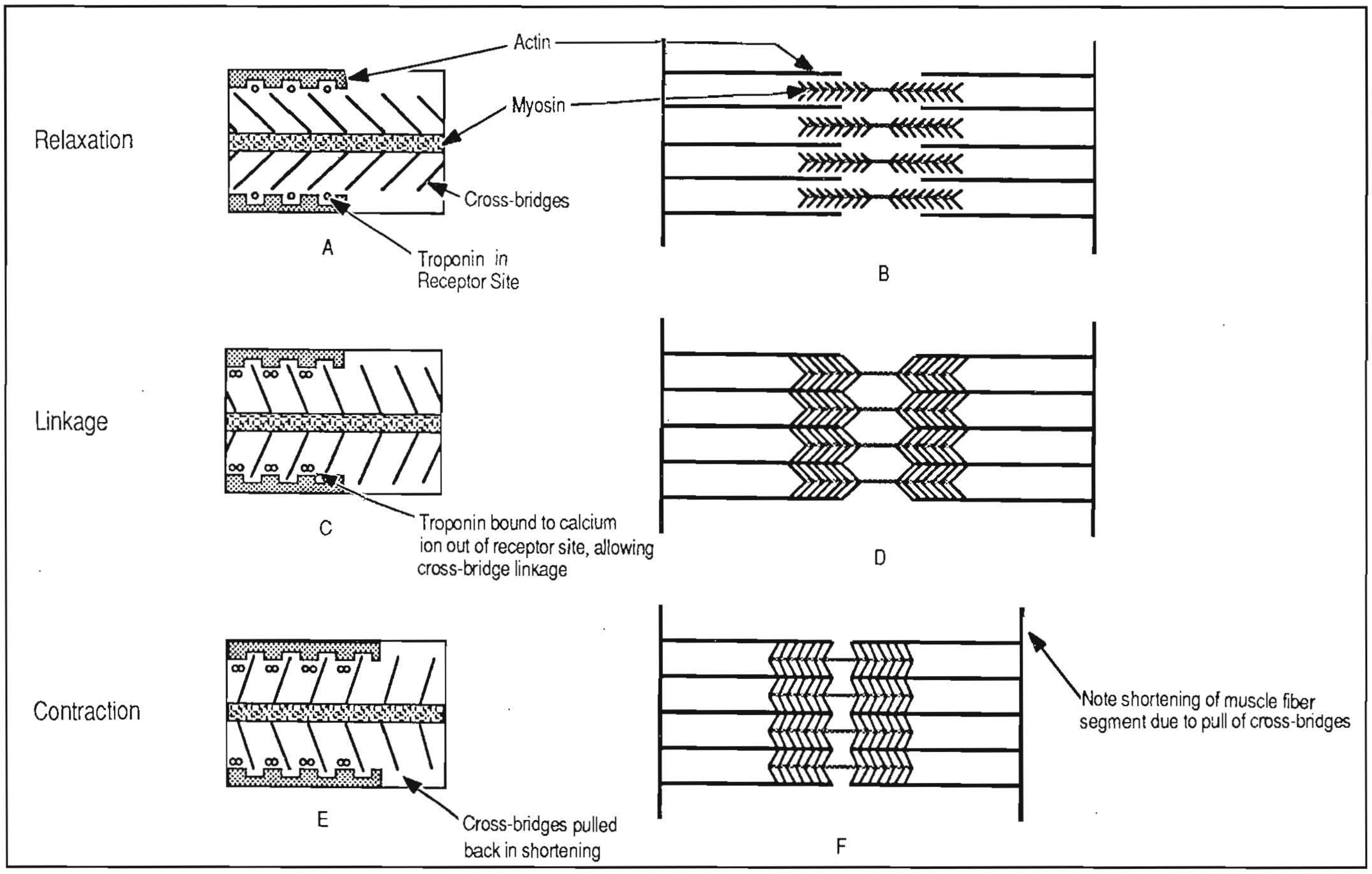
ACTIONS

The actions of calcium channel blockers have undergone extensive study and clarification, but the molecular basis of their actions remains unclear. Evidence regarding the presence of specific receptors for calcium channel blockers is incomplete. It has been suggested by Herscheler et al. that these drugs may have to cross the cell membrane to gain access to receptor sites on the inner (cytosolic) surface of the membrane to enable them to block the calcium channels (5). It is also unclear whether these drugs block calcium channels by entering the mouth of the channel, by interacting directly with the channel proteins, or by disordering or otherwise modifying specific regions of the membrane and thus altering the functional response of the calcium channel proteins within the membrane (6). Although the exact mechanisms of action calcium channel blockers have not been described, their therapeutic actions have been extensively documented.

Cardiac Effects

Intravenous and intracoronary administration of nifedipine has been shown to increase coronary sinus oxygen saturation and coronary artery diameter in normal and post-stenotic segments (7-9). Many studies have shown that administration of nifedipine to patients with coronary artery disease (CAD) results in increases in coronary blood flow (9-12). The greatest increases appear to occur following intracoronary administration, followed by intravenous, sublingual, and oral administration, in that order. Engel and Lichtlen showed nifedipine increased coronary blood flow to myocardial tissue served by both normal and stenotic coronaries. Relative to baseline values, percentage increases in coronary flow to myocardial tissue served by stenotic coronaries were greater than those in tissues served by normal coronaries (11).

Malagoff et al. suggested that the beneficial effects of nifedipine on coronary blood flow in patients with CAD may



M. Gunderson, H. Shuster

Figure 1 - Calcium in muscle contraction - Figures A (detail view) and B illustrate the relaxation phase of mucle fiber proteins. Note the presence of troponin molecules in A, blocking the receptor sites needed for cross-bridge linkage between actin and myosin. Figures C and D show establishment of cross-bridge linkage. In C, note the receptor site has formed a cross-bridge after the troponin was pulled out and bound by the calcium. Figures E and F show how cross-bridges pivot to move the actin and myosin together in an interleaving fashion. The contracted legnth of the muscle fiber is shown in figure F.

be related to a decrease in resting coronary vascular tone (13). The dilation of large epicardial coronary arteries after intracoronary administration of nifedipine is prolonged and suggests that nifedipine predominantly results in sustained relaxation of the epicardial conductance vessels, while intramural arterioles are only transiently dilated (7,10). Nifedipine has little effect on coronary sinus flow or myocardial oxygen consumption either at rest or during exertion. Thus, the overall effects of nifedipine are consistent with mild coronary vasodilatory action, reduced coronary arterial resistance and increased myocardial blood flow, usually without a significant reduction in myocardial oxygen consumption (14).

The acute administration of sublingual nifedipine has been shown to increase heart rate in cardiac patients and normal volunteers (15). The increases in heart rate have ranged between statistical insignificance to increases of up to twenty-eight percent over control values. These increases in heart rate are due to a baroreceptor mediated increase in beta-adrenergic tone secondary to systemic vasodilatation (6). Although nifedipine may induce a statistically significant increase in heart rate, it does not produce clinically significant changes in the maximum sinus node recovery time, sinoatrial conduction time, atrio-ventricular conduction time, QRS duration or the QT interval in patients with normal electrocardiac function (15). Thus, nifedipine has no direct chronotropic properties of its own.

Cardioprotective Effects

Myocardial ischemia and subsequent reperfusion may cause accumulation of excess free intracellular calcium which can lead to inhibition of adenosine triphosphate (ATP) production by the mitochondria. In turn, a depression of ATP synthesis can eventually affect the sodium and calcium pumps to ultimately cause even more calcium accumulation in the cell and mitochondria - a vicious cycle that may destroy the ATP generating capacity of the mitochondria. Increased intracellular calcium appears to be partly responsible for ischemic myocardial cell death (16). Inhibition of the inward transport of calcium by means of calcium channel blockers may therefore prevent mitochondrial calcium overload and consequently preserve the structure and function of myocardial cells, protecting against the deleterious effects of hypoxia and ischemia (12,17,18).

Animal studies have shown that nifedipine augments collateral flow into the ischemic myocardium after coronary occlusion and decreases infarct size while preserving ventricular function (18,19). There are conflicting data regarding the effect of nifedipine in limiting the extent of ischemic injury after acute myocardial infarction in humans (20, 21).

It is likely that other mechanisms may contribute to the cardio-protective effects of nifedipine. Other possible mechanisms may include inhibition of calcium dependent platelet aggregation (22), the effect of nifedipine on coronary hemodynamics and myocardial oxygen supply, and the ability of nifedipine to retard and/or prevent the onset of arrhythmias associated with ischemia (23).

Peripheral Vascular Effects

Sublingual nifedipine may elicit significant increases in cardiac output and decreases in left ventricular filling pressure as a result of the reductions in systemic vascular resistance (24). These increases in cardiac output and reductions in systemic vascular resistance are due to direct arteriolar vasodilatation with reductions in impedance to left ventricular ejection (25-27). Decreases in left ventricular filling pressure may be related to an improvement in ventricular emptying due to afterload reduction (27). Reductions in pulmonary vascular resistance associated with increases in the cardiac index further suggests a selective effect of nifedipine on arterial resistance vessels (28). Afterload reduction has also been demonstrated by the consistent lowering of left ventricular, systolic and diastolic volumes (26,29).

The magnitude of the decreases in systemic vascular resistance due to nifedipine administration has been inversely correlated with initial values, indicating that basal vascular tone is very important in modulating the response to arterial vasodilatation (26,29). Greater decreases in systemic vascular resistance have also been seen in patients with abnormal than in those with normal left ventricular function (24). Nifedipine reduces left and right ventricular end-diastolic pressures in patients with impaired left ventricular function (24,26) but does not alter the filling pressures of those with normal left ventricular function (24). Nifedipine may also effect a striking improvement in left ventricular ejection fraction and ejection rate in both subjects with normal or impaired left ventricular function (26). Likewise, cardiac output has increased significantly in patients with impaired left ventricular function but remained unchanged in those with normal left ventricular function (24). Thus, the reduction of left ventricular preload by nifedipine is not due to venous pooling but rather to improved left ventricular systolic function in response to afterload reduction. This is especially true in patients with impaired left ventricular function (24).

PHARMACOKINETICS

Close to one hundred percent of a single oral or sublingual dose of nifedipine is absorbed, although only forty-five to sixty-eight percent of the drug reaches the systemic circulation and is bioavailable (22). Considerable variations in peak plasma nifedipine concentrations have been measured after oral and sublingual administration. Time required to reach peak levels have been reported from thirty to one hundred eighty minutes (22,30). Presumably, these variations are due to differences between individuals in the rate of drug absorption and/or variability in the extent of nifedipine breakdown as it first passes through the liver.

The steady state volume of distribution of nifedipine in man after oral administration is 1.32 liters/kg and 0.62-1.12 liters/kg for intravenous administration (30). Nifedipine protein binding to albumin is concentration dependent and has ranged from 92 to 98% (22,30). Nifedipine undergoes almost complete (95%) hepatic oxidation via microsomal oxidases to three pharmacologically inactive metabolites which are excreted in the urine (22).

The elimination half-life of nifedipine is apparently dependent on the dosage form in which it is administered, with half-lives of 6-11 hours, 2-3.4 and 1.3-1.8 hours measured after oral tablet, oral capsule and intravenous administration, respectively (22). The total systemic and intrinsic clearances of nifedipine from plasma have ranged from 27 to 66 liters per hour and 33 to 37 liters per hour, respectively (22). Renal impairment does not significantly alter nifedipine pharmacokinetics.

At the present time, nifedipine is only available commercially as a sealed capsule filled with gel containing 10 mg. of the drug. The capsule may be taken orally with later absorption in the G.I. tract. Sublingual administration is more appropriate for emergency situations. To administer nifedipine sublingually, the capsule should be punctured with a 18 gauge or larger needle. The capsule may then be squeezed to deposit gel beneath the tongue. The small volume of gel makes it safe even in an unconscious patient.

EMERGENCY USE

Classic Stable Angina

Classic stable angina is characterized by the occurrence of sudden retrosternal chest pain which is precipitated by exercise of relatively short duration and is closely associated with severe atherosclerotic coronary artery obstruction. In the majority of patients with chronic disease, their chest pain and ST segment depression are reproducible at a given work load and this appears to be directly related to an imbalance between myocardial oxygen supply and demand (31, 32). In recent clinical investigations, patients diagnosed with chronic stable angina were administered nifedipine, 30 - 60 milligrams per day, for periods of up to six weeks. It was found that anginal attacks, assessed by consumption of glycerol trinitrate tablets, were reduced by 50-60%. Exercise tolerance was also markedly improved by nifedipine as indicated by significant increases in the time to onset of angina, maximal exercise duration and work performance at both the onset of angina and at maximal exercise (33). Sherman and Liang found that both systolic and diastolic blood pressures were significantly reduced at the onset of angina and at maximum exercise, whereas the rate pressure product (which is indicative of myocardial oxygen consumption) was not (33). Myocardial ischemia, as reflected by ST segment depression at peak exercise, was significantly reduced by nifedipine and there was a decrease in the magnitude of ST segment depression at specific levels of rate pressure product during moderate to maximal exertion (33). This data suggests that patients with chronic stable angina, the vasodilator properties of nifedipine reduce afterload while heart rate remains relatively unchanged. The drug offers clinical benefits to such patients by decreasing myocardial oxygen demand (33). Additional evidence indicates that nifedipine may augment myocardial oxygen supply by direct coronary vasodilatation, although this appears to be of lesser importance (31, 32).

Variant Angina

Variant (Prinzmetal's) angina is a clinical syndrome characterized by episodes of spontaneous and unprovoked ischemic cardiac pain at rest (usually at night or early morning) associated with ST segment elevation and relieved by sublingual glycerol trinitrates (34,35). Although the precise mechanisms are unknown, there is substantial evidence that coronary artery spasm (of sufficient severity to induce ischemia) plays a major pathophysiological role in the ischemia of variant angina (17). The clinical efficacy of nifedipine for treatment of variant angina has been extensively documented. Ebner and Donath, in a retrospective study of 439 patients with variant angina treated with nifedipine (10-160 milligrams per day, up to six years) indicated that an overall success rate of 87 percent was achieved (36). In a large multicenter study, nifedipine eliminated attacks of variant angina in 63 percent of the patients and greatly reduced the incidence of attacks in all but 7 percent of the remaining patients (37). Stone et al. noted that complete remission of anginal attacks or reduction in anginal frequency of more than 50 percent occurred more often in patients with variant angina than in those with stable angina (38). These results indicate that nifedipine can be expected to produce a beneficial response in most patients with a vasospastic component to their angina.

Unstable Angina

Unstable angina is a clinical syndrome which is usually diagnosed when episodes of angina increase either in frequency, severity, or duration. Such patients frequently report decreased responsiveness to anti-anginal medications or need to use them more often (39). The increased symptomatic manifestations of myocardial ischemia, with pain occurring at rest, (day or night), are often associated with reversible ECG changes in the ST segment and or the T wave. Although the vast majority of ischemic episodes abate, this is not always the case. The disease can exacerbate with development of an acute myocardial infarction. There are growing indications that coronary vasospasm may play a major role in the onset of ischemic attacks (39). This suggests that calcium antagonists may make an important contribution to their treatment. In several studies, 74% to 89% of patients with unstable angina, refractory to conventional therapy, responded successfully when nifedipine, 30 to 240 milligrams per day, was added to their treatment during short term hospitalization (9,40,41). In the largest of these trials, nifedipine at 10 milligrams, 6 times per day, was found to produce short-term stabilization of unstable angina in greater than 80 percent of patients. After two years, nifedipine, titrated between 30 and 80 milligrams per day, maintained excellent long-term benefits in 60 percent of patients (40). Although these initial results are encouraging, further studies must be made prior to absolute statements about the relative efficacy of nifedipine in unstable angina.

Myocardial Infarction

The differentiation between angina and myocardial infarction is a common problem, particularly in the field. Sublingual nitroglycerin is a first-line drug in the treatment of chest pain without hypotension. If there is favorable response to nitroglycerin, angina is implied. Should the relief be minimal or transient, nifedipine may be considered. Should there be a vasospastic component to the angina, the relief may be dramatic.

Patients who do not have relief of chest pain with nitroglycerin are more likely to be suffering a myocardial infarction. Nifedipine may offer some clinical benefits with its cardio-protective effects. However, the potential benefits of nifedipine are likely to be offset by enlargement of the infarction due to increased myocardial oxygen demand from reflex tachycardia associated with systemic vasodilation. If the infarction is due to intense coronary vasospasm, the response to nifedipine may be very good. Unfortunately, it is difficult to tell whether a developing infarction has a vasospastic component before nifedipine is attempted. More research is needed to address the consequences of nifedipine when vasospasm is not part of the clinical picture.

Hypertension

Approximately 25 to 30 percent of the U.S. adult population has hypertension. Of these people, 10 percent have severe hypertension (42). The primary hemodynamic abnormality in essential hypertension is an elevation of both peripheral and renal vascular resistance (43). Hemodynamic differences among patients are related to age, race, sex, and duration of hypertension (43, 44). In established hypertension, the cardiac output decreases by as much as 25 percent and there is a marked increase in systemic vascular resistance (43). In hypertensive crisis, there is a marked increase in systemic vascular resistance because of structural changes in the vasculature (45). Therefore, therapy should be directed at correcting this hemodynamic derangement. Hypertensive crisis if untreated, often quickly results in death, usually related to central nervous system events, cardiovascular events or renal failure (44). Some patients may tolerate very high blood pressures with few symptoms or signs, whereas others may manifest end organ damage at lower blood pressures. Thus, the definition of hypertensive crisis depends on the clinical assessment of blood pressure level and clinical and laboratory assessment of end organ damage. The absolute blood pressure in itself does not determine the seriousness of the clinical situation or the necessary expediency of treatment and monitoring within the critical care unit (44). Although treatment of hypertensive crisis is generally aimed at lowering the blood pressure, it is important not to lower the blood pressure precipitously or to a subnormal level. This is of particular importance in patients with some degree of end organ damage (42). Such treatment may critically reduce blood flow and perfusion to vital organs and exacerbate a cerebral or myocardial ischemia or renal failure (42,46). Smooth, gradual reduction of blood pressure is crucial to patient management. Thus, a return to normal blood pressure should be avoided in the first few days of therapy (44,46). The arterial blood pressure should be controlled at about 160/100 millimeters of mercury for the first few days until the body can adjust to lower blood pressure levels (44,46).

Hypertensive crisis may occur when the diastolic blood pressure is 120 to 130 millimeters of mercury in association with end organ damage that is either progressive or present at the time of the initial evaluation (42). Most patients with a hypertensive crisis should be treated expeditiously, within minutes to an hour (46,47). Administration of calcium channel blockers is an ideal approach to treatment of patients with hypertensive crisis. These drugs reduce blood pressure by decreasing the intracellular calcium available in vascular smooth muscle cells. Through vasodilatation, nifedipine reduces blood pressure in patients who have hypertension as well as high peripheral vascular resistance. However, the drug minimally affects blood pressure levels in subjects within the normal range and with normal vascular tone (48). Nifedipine's vasodilatory effect is proportional to vascular resistance prior to its administration (49). Moreover, cardiac output increases acutely after administration of nifedipine to patients with hypertension. This increase is a result of baroreflex activation which causes an increase in blood flow (49).

Many studies have shown that nifedipine can reduce blood pressure effectively with minimal side effects in patients with hypertensive crisis (46-48, 52). Bertel et al. showed that 10 to 20 milligrams of oral nifedipine markedly reduced blood pressures in a heterogeneous group of patients with severe hypertension, within 30 minutes (48). In the treatment of a group of patients with hypertensive crisis, Ellrodt et al. found that sublingual nifedipine produced a significant decrease in systolic, diastolic and mean arterial pressures within five minutes of administration. Although there were considerable variations in individual responses, 97, 90 and 67% of patients achieved diastolic blood pressures of less than 120, 110, and 100 millimeters of mercury, respectively, in the first sixty minutes (51). Haft, in treating 63 patients with diastolic pressures over 120 millimeters of mercury or systolic pressures over 200 millimeters of mercury, or both, found that all of the patients tested had blood pressures less than 200/100 in twenty to thirty minutes following 10 to 20 milligrams of nifedipine sublingually (52). He noted no side effect in any of the patients, even though 37 of them were taking at least one other anti-hypertensive medication. The reduction of blood pressure was prompt, smooth, and predictable (46-48, 50-52).

In hypertensive encephalopathy and acute cerebral vascular accidents associated with severe hypertension, the rapid reduction of blood pressure is critical in limiting tissue damage. In both of these conditions, the integrity of the microvasculature is compromised. Resultant increased capillary permeability causes interstitial edema, thereby contributing to cerebral cellular dysfunction and death. Excessive arterial pressures (exceeding cerebral autoregulation) leads to increased fluid pressure which exacerbates cerebral edema and its consequences (52,53). Similarly, inadequate cerebral blood flow leads to cellular hypoxia, capillary leak, and edema (53). A rapid fall in blood pressure in patients with severe hypertension may result in permanent ischemic brain damage because of disturbances in autoregulation of cerebral blood flow (53). Strandgaard et al. noticed a reduction in cerebral blood flow in chronically hypertensive patients with mean arterial pressures below 120 millimeters of mercury (54). Thus, an acute reduction of 30 percent from baseline is recommended to avoid underperfusion of the chronically hypertensive cerebrum (53). Bertel et al. found cerebral blood flow significantly increased after administration of nifedipine in patients with severe hypertension (48). Several other investigators have yielded similar results, showing the efficacy and safety of nifedipine in the management of severe hypertension without cerebral compromise (49-53).

The patient experiencing a hypertensive crisis may be encountered in the field. In order to minimize further end organ damage, expeditious reduction of the systolic and diastolic pressure must be achieved. Sublingual nifedipine administered in the field is very effective toward this goal. The advantage of nifedipine over other anti-hypertensives is that nifedipine will reduce blood pressure towards a point of normal vascular tone. It has little effect beyond that point (48,49). Thus, nifedipine may be administrated without the sophisticated hemodynamic monitoring that may be appropriate with other types of agents.

Systemic hypertension and ischemic heart disease frequently occur in the same patient (55). There are several reasons for this. First, hypertension is a well-known risk factor for coronary atherosclerosis. Second, several of the diseases or conditions associated with one of these two disorders are often associated with the other. For example, systemic hypertension often results in renal disease and uremia accelerates coronary atherosclerosis. An increased incidence of both hypertension and symptomatic coronary atherosclerosis is found in patients with obesity, diabetes mellitus, or a long history of cigarette smoking. In patients with severe coronary atherosclerosis and in those with mild to moderate coronary artery disease having considerable left ventricular hypertrophy, hypertension is an important determinant of increased myocardial oxygen demand (55). Patients with a combination of hypertension and ischemic heart disease frequently have depressed left ventricular function and often develop signs and symptoms of left-sided heart failure (53,55). In numerous studies reporting the results of nifedipine in patients with hypertensive crisis and associated left ventricular failure, nifedipine markedly reduced systemic and pulmonary arterial pressures and relieved pulmonary edema (26, 53, 56). Nifedipine induced a striking reduction in left ventricular pressure (26.5 millimeters of mercury to 17 millimeters of mercury) and increased cardiac index (2.76 liters per minute to 3.77 liters per minute), as reported by Cohan and Checcio, when administered to patients in acute cardiac failure. Olivari noted that nifedipine produced no intravascular volume expansion, no alteration in glomerular filtration rate and no changes in plasma renin activity in the treatment of his patients (57). The net effect of nifedipine in hypertensive patients associated with ischemic heart disease is reduction of blood pressure, increased coronary blood flow, marked improvement in left ventricular function, and relief of pulmonary edema (26,53,55,57).

SIDE EFFECTS

Analysis of composite studies of nifedipine usage worldwide has shown an overall incidence of side effects of about 20 percent (58). These side effects, which are generally extensions of the vasodilating effects of nifedipine, can be alleviated by either decreasing the nifedipine dose, or by combining the drug with a beta blocker (22). Side effects from nifedipine increase with larger dosage. Thus, oral tablets (slow release) are indicated for long term therapy (59). Ebner and Donath presented an analysis of side effects based on 4,863 patients (36). It was shown that side effects usually occurred within the first 14 days of chronic treatment. The most frequent side effects were headache, flushing, dizziness, gastrointestinal symptoms, and fluid retention, in that order. Cardiovascular side effects most commonly included tachycardia and hypotension (60). Tachycardia was generally transient due to the baroreceptor reflex of the acute vasodilatation. The incidence of hypotension was dose related, occurring in 5% of patients receiving greater than 120 milligrams of nifedipine a day (60). Stone et al. (38) reviewed 716 cases of patients with refractory angina and noted that 14% of the patients experienced an increase in angina frequency during chronic nifedipine treatment. He also noted that an exacerbation of acute anginal symptoms was more common in patients with classical exertional angina than in patients with either variant or unstable angina. It was reasoned that these patients had only a small vasospastic component in their ischemic heart disease. Schanzenbächer et al. described a worsening of cardiac ischemia in several high risk patients on beta blockers or nitrates when the addition of nifedipine caused hypotension (10). It was later shown that this could be prevented by an adjustment in the dosage of nifedipine (55). Several other potential side effects have been reported in a few patients but their relationship to nifedipine has not been clearly established (22). The reported frequency with which side effects, most commonly those related to vasodilatation, required discontinuation of nifedipine has been reported to be 2 to 6% (36, 60). This has led most investigators to consider nifedipine as a relatively safe agent in the treatment of ischemic heart disease and hypertension (14, 25, 28, 32-39, 46-53, 55, 56).

DRUG INTERACTIONS

Several pharmacokinetic drug interactions have been reported for calcium entry blockers. Many of these can be ascribed to the extensive hepatic metabolism of these drugs, but their clinical relevance remains to be evaluated (61). However, when other agents metabolized by the microsomal oxidative system are used concurrently, there can be potential interactions for either drug.

Cimetidine, a widely-used agent for gastrointestinal disorders, has been shown to reduce hepatic blood flow and inhibit hepatic microsomal oxidative functions (62). When either oral or intravenous nifedipine was administered to patients taking Cimetidine, the bioavailability was nearly doubled while the clearance was decreased by about 40 percent (62). It was also noted that Cimetidine exacerbated the hypotensive effects of nifedipine (61). Ranitidine has not been shown to interact with nifedipine and should be considered for use in place of Cimetidine in patients concurrently taking nifedipine (61). Cigarette smoking has been show to stimulate both the heart rate and the activity of hepatic microsomal oxidases. These effects would be antagonistic to the action of any anti-anginal agent, because increased heart rate increases myocardial oxygen demand, while increased drug metabolism results in lower plasma levels of the drug (61).

There are conflicting clinical reports regarding the interaction of nifedipine with digoxin. Recent studies have shown nifedipine having no effect on levels of digoxin in normal or cardiac patients (63,64). Although it appears safe to use nifedipine in digitalized patients, caution should be exercised when initiating this combination.

A pharmacokinetic interaction apparently occurs between nifedipine and quinidine in some patients. Farringer et al. reported two cases in which concurrent nifedipine therapy appeared to decrease serum quinidine levels (65). This continuation of nifedipine therapy resulted in an increase in serum quinidine concentrations from 2/2.5 to 4.6 micrograms per milliliter in one case and from 1.2 to 4.0 micrograms per milliliter in the other. Thus, this combination should be used with caution, particularly because of the rebound increases in serum quinidine levels after nifedipine is discontinued. Similarly, if adequate serum concentrations of quinidine cannot be obtained, concurrent nifedipine administration should be ruled out before larger doses of quinidine are ordered (61).

PRECAUTIONS IN SELECTED PATIENTS

Since nifedipine is extensively metabolized by the liver, liver disease would most certainly have the potential to modify the pharmacokinetic parameters with this drug, with resultant increases in half-life. Thus, for cirrhotic patients, half the normal intravenous dose is appropriate. The oral dosage should start from 25% of the normal oral regimen.

Elderly patients experience changes in both hepatic drug oxidation and hepatic blood flow during the aging process. Therefore, dosaging should be conservative (61). Until more data regarding the use of nifedipine in the elderly is available, patients over the age of 70 years should receive low dosages of the drug initially.

Nifedipine has not been approved for use in the lactating mother or in the pediatric patient.

Nifedipine should be used with caution in patients with renal disease or diabetes mellitus until further studies establish their safety in such patients.

COST CONSIDERATIONS

In the American medical practice, the course of therapy considered most beneficial to the patient has traditionally governed clinical management. The increasing popularity of HMO's, PPS's and other prepaid medical plans has led to an increasing pressure to constrain rising health care costs. Cost consideration of therapy has become an important aspect of patient management. In a recent study by Luce et al., the cost implications of the use of nifedipine as compared to nitroprusside in the definitive care of acute, severe hypertension was reviewed (66). The investigators found that patients who received nifedipine in the emergency department, when compared with those who received nitroprusside, showed a significant savings. These savings ranged from $270 for the first day to $1,437 for a four day period (66). The primary reason that was cited for this difference involved a less urgent need for close monitoring of patients on nifedipine who would otherwise be receiving a parenterally administered drug. The authors concluded that nifedipine was less costly than nitroprusside for the treatment of patients with hypertensive emergencies (66).

SYNOPSIS

Nifedipine has shown to be an effective and relatively safe agent in the treatment of all grades of angina and mild to severe hypertension. Its rapid onset and smooth predictable effects make it particularly useful in the emergency setting.

Hypertensive emergencies must be controlled within an hour in order to limit end organ damage. The therapeutic choices available to the paramedic and emergency department are limited by administration and hemodynamic monitoring requirements. Until recently, diuretics and beta blockers were the only reasonable treatments available. What was needed was an agent that could lower patients blood pressure quickly, safely and predictably. It also needed to be easily administered and without the need for extensive hemodynamic monitoring. Nifedipine meets these requirements. It is easily administered by piercing a 10 milligram capsule and evacuating its contents sublingually. It is absorbed quickly and near completely with its vasodilating effects seen within five to ten minutes and peak effects seen in thirty to sixty minutes. The decrease in blood pressure is directly proportional to pretreatment vasoconstriction and peripheral resistance levels. Therefore, the reduction in blood pressure is predictable. Side effects are seen in approximately 20 percent of patients treated with nifedipine but are generally limited to dizziness, flushing, and occasional tachycardia.

The coronary vasodilating effects of nifedipine are useful with various forms of angina, especially if vasospasm is a major underlying component. This drug has been used for years for this indication and with impressive results. Besides relieving the coronary vasospasm, nifedipine dilates coronary arteries thereby increasing the supply of blood to the heart. This can lead to a reduction in ischemia and improved cardiac function in some patients.

Nifedipine should prove to be a valuable and effective tool in the management of patients with angina or hypertension in the prehospital, emergency department or other acute care settings.

REFEREE COMMENTARY

Robert B. Pettyjohn, D.O. (Emergency Department, Metropolitan General Hospital) - Nifedipine has been used extensively in the Pinellas County EMS system with very good results. In our experience as a metropolitan EMS system responding to approximately 71,000 emergency calls per year, we have seen few failures or complications with nifedipine in the field. This paper points out the pharmacologic aspects of nifedipine quite well. I am not aware of any evidence that sublingual nifedipine has an advantage over sublingual nitroglycerin in treatment of classic angina. However, sublingual nitroglycerin has a much lower cost per dose than nifedipine.

Brian Cobb, M.D. (Internal Medicine PGY-4, University of South Florida College of Medicine) - Mr. Wilbur has provided an excellent and comprehensive review of the pharmacology and clinical applicability of nifedipine in emergency care. Nifedipine is useful in the treatment of angina, malignant hypertension, and hypertensive urgencies. Although the drug is quite safe and effective, it bears repeating that blood pressure should be carefully monitored after administration to detect excessive lowering, which could result in worsened cerebral or myocardial ischemia. We have had good results with the drug in both angina and hypertension. Emergency physicians should, however, consider the short duration of action of the antihypertensive effect and provide an appropriate regimen for sustained control before discharging patients where hypertension has been treated with nifedipine. While prompt relief of chest pain with nitrates suggests angina rather than myocardial infarction, one should bear in mind that the pain of MI can also be relieved with nitrates.

Mr. Wilbur's Response - I agree with Dr. Pettyjohn that that there is no evidence showing that sublingual nifedipine is preferable to sublingual nitrates in treatment of classic angina. Nitrates are still the drug of choice. However, if sublingual nitrates do not relieve acute cardiac pain of unknown etiology, nifedipine should be considered as it may be very beneficial if the etiology turns out to be vasospastic with variant angina or MI secondary to vasospasm. In the normotensive patient with thrombic MI, there is no evidence showing harm from nifedipine. If the thrombic MI patient is hypertensive, nifedipine may provoke a reflex tachycardia with an increase in myocardial oxygen demand that is theoretically deleterious, but this tachycardia is transient. In either case, the nifedipine is beneficial by limiting mitochondrial damage secondary to excessive intracellular calcium influx in the ischemic myocardial tissues. I agree with Dr. Cobb's remarks. I would like to thank both Dr. Pettyjohn and Dr. Cobb for their comments.

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Tampa Bay EMS Journal
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Volume 1, Number 1
July-September, 1987

Rationale for Changes in BCLS and ACLS

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Continuing research in resuscitation and acute cardiac care has prompted periodic conferences sponsored by the American Heart Association to develop consensus recommendations to the medical community. The latest such meeting was held in July of 1985. Previous meetings were held in 1973 and 1979. These meetings have each produced a major paper in the *Journal of the American Medical Association (JAMA)*(1, 2, 3) detailing methods advocated for basic and advanced cardiac life support (BCLS, ACLS) as well as related logistical and legal issues of their implementation. This latest publication, entitled "Standards and Guidelines For Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care (ECC), " was published in the June 6, 1986 issue of *JAMA (3)*.

The considerable attention given to these proceedings stems from the esteem held by the medical community for the personnel and organizations which participate. Persons involved in these proceedings include prominent clinical research scientists, educators and organizational representatives. Organizational participation at the 1985 meeting included, the American Academy of Pediatrics, American College of Emergency Physicians, American Medical Association, National Research Council -- National Academy of Sciences, Society For Critical Care Medicine and the United States Department of Transportation-National Highway Traffic Safety Administration.

The opening paragraph of the paper clearly defines the aim of their efforts. "This publication is the product of the 1985 National Conference on Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care (ECC) and represents an updating of the standards and guidelines that were published in 1974 and updated once since, in 1980. It is intended for use as a guide for the proper training in and performance of CPR and ECC. Like the 1974 and 1980 standards and guidelines, these represent a consensus of experts from a variety of disciplines. Cardiopulmonary resuscitation and ECC are dynamic disciplines, however, that continue to develop through ongoing experience and research. Thus, there is no intent to limit the adoption of new standards as they emerge; deviations from these standards and guidelines may occur when a trained physician proficient in CPR and ECC recognizes that such is in the best interest of the patient."

The purpose of this paper is to highlight some of the more significant changes in these BCLS and ACLS guidelines concerning direct patient care. Bibliographic citations have been intentionally limited to those in the 1986 JAMA paper (3), to assure that the research rationale behind these changes is based on those considered by the conference participants and not the authors of this article.

BASIC ADULT CARDIAC LIFE SUPPORT

Opening The Airway

The head tilt/chin lift technique is now the recommended lay rescuer method for opening the airway in cases without suspicion of neck injury. Suspected neck injury cases should still be managed with the jaw thrust. The head tilt/neck lift has been superceded by the head tilt/chin lift on the basis of research which found the latter to be more effective in alleviating obstruction by the tongue (4). However, it is recommended that professional rescuers learn both the neck lift and chin lift techniques.

Ventilation

Inflation of the stomach with air during the course of rescue breathing has the potential to cause regurgitation and aspiration with lethal consequences. The air pressure at the pharynx required to inflate the lungs is less than that which will distend the esophagus and thereby allow gastric inflation (5-7). Consequently, the new ventilation procedures are aimed towards reduction of high pharyngeal pressures by using slower and more sustained breaths lasting 1 to 1.5 seconds. This should reduce the likelihood of air entering the stomach. Initial ventilations will no longer be "staircased" for the same reason. Instead, the first ventilation will be followed by a pause before delivery of a second ventilation. This same sequence will be used for one rescuer CPR ventilations between each set of 15 chest compressions. In two rescuer basic CPR, a pause is made between sets of every 5 chest compressions to allow for the slower single ventilation. In the interest of simplifying training, 2 rescuer CPR will no longer be taught to the lay public.

In the event that another professional rescuer may be assisting with basic ventilation (without an endotracheal tube or esophageal airway), the assistant may apply firm downward pressure with their fingers on the cricoid cartilage. This procedure, sometimes called the Selick Maneuver, holds the esophagus closed between the cricoid cartilage anteriorly and the spinal column posteriorly (8, 9). It is intended to prevent air from entering the stomach and gastric contents from being regurgitated.

Obstructed Airway

The basic categorization of airway obstruction has been changed from complete and partial to those with good or poor air exchange. A good air exchange allows forceful coughing, while a poor exchange has a weak, ineffective or absent cough. A patient with good exchange should be allowed to cough on their own. A natural cough produces higher lung pressures to expel foreign bodies than does an artificial cough (back blow or thrust) (4).

Those with poor air exchange should be treated as if it were a complete airway obstruction. In such situations, a sequence of six to ten successive Heimlich maneuvers (abdominal thrusts) should be used. Patients with advanced pregnancy or marked obesity should receive chest thrusts in lieu of the Heimlich maneuver.

Controversy between the use of back blows and the Heimlich maneuver continues. A research study has reported that when back blows and the Heimlich maneuver are evaluated separately, the Heimlich maneuver had better efficacy than the back blows (10). On that basis and in the interest of simplification in training, the Heimlich maneuver alone is advocated for adults.

Near Drowning

The use of the Heimlich maneuver has been considered in treatment of the near drowning victim who requires rescue breathing. The Heimlich maneuver may be effective in expelling water from the lungs (11-13). However, due to the low incidence of actual lower airway aspiration of water (14, 15) and rapid absorption of aspirated freshwater (14), the Heimlich maneuver should only be applied if there is strong suspicion of foreign matter obstructing the airway or if the patient fails to respond appropriately to ventilatory support.

Chest Compression

A better understanding of the mechanisms generating blood flow during CPR have led to a recommendation for compression rates of 80 to 100 per minute (16-18). In addition to improving the cardiac output, the faster rate may better compensate for the interruptions in chest compressions during pauses for ventilation of non-intubated patients during two rescuer CPR.

Discussion was presented on the use of new techniques to improve blood flow during CPR, such as simultaneous compression-ventilation CPR (19), interposed abdominal compression CPR (20), MAST-assisted CPR (21) and CPR with abdominal binding (22). None of these techniques were specifically recommended on the basis that specialized devices are required and that their efficacy in long term outcome has yet to be established. Further research into these methods has been encouraged.

Statements are now being made about the application of CPR in the prehospital setting. It is recommended that moving a patient to a position of convenience be deferred until it can be done without interruption of CPR or until a pulse is restored. Interruptions in CPR for movement down stairs should not exceed 30 seconds. CPR should not be interrupted during movement of a patient to the ambulance by litter.

BASIC PEDIATRIC AND NEONATAL CARDIAC LIFE SUPPORT

The techniques for CPR are essentially the same in adult and pediatric patients with use of the head tilt/chin lift and a rate of 100 compressions per minute.

Airway obstruction in the child over one year of age is treated the same as for the adult, with the Heimlich maneuver. The concern for intra-abdominal injury in children less than one year of age has led to a decision to retain the incumbent sequence of back blows and chest thrusts with the head of the child held lower than the chest to gain gravity assistance in alleviating the obstruction.

For ventilations and chest compressions in the depressed neonate, an assisted ventiatory rate of 40 breaths per minute should be employed. After 15-30 seconds of adequate ventilation, a decision may be made on subsequent treatment. If the heart rate is over 100 with spontaneous breathing, support may be discontinued. With a heart rate less than 60, both ventilation and chest compression are applied. A heart rate of 60-100 and rising indicates continuation of ventilation without chest compressions. A heart rate of 60-100 and falling should prompt reevaluation of ventilation efficacy and application of chest compressions should the heart rate fall below 80.

The infant heart has been found to be comparatively lower in the chest than in the adult. Thus, the fingers are positioned lower on the sternum during performance of CPR. Using three fingers, the finger closest to the head should be positioned one finger width below a line between the nipples (23).

ADULT ADVANCED CARDIAC LIFE SUPPORT

Ventilation

Oxygen powered mechanical breathing devices using manually-triggered ventilation have had a propensity to produce gastric distention in patients due to the higher pharyngeal pressures associated with their gas flow rates exceeding 100 liters per minute. With the recognition of this potential complication, these devices are now recommended to provide flow rates of only 40 liters per minute with the venting of gases to the atmosphere if the airway pressure exceeds 60 centimeters of water. These specifications are appropriate only for adult use and therefore, these devices should not be used with children.

The esophageal obturator airway devices have previously been recognized as an acceptable airway adjunct. However, their potential for complications (24, 25) and possibly inferior efficacy in ventilation (26-28) prompted a clear statement favoring endotracheal intubation as the preferred method of advanced airway control. Specifically, endotracheal intubation should be a part of the armamentarium of all ACLS providers, including paramedical personnel (29-31). Thus, it seems that esophageal obturator airways are no longer acceptable first-line substitutes for endotracheal intubation in prehospital ACLS.

Concerns about developing gastric distention during ventilation are not applicable to the patient with a cuffed endotracheal tube. Therefore, a pause need not be made during two rescuer CPR to facilitate ventilation. Further, synchronization of ventilation between chest compressions has been found to be unnecessary when a cuffed endotracheal tube is in use. Research has demonstrated that ventilation remains adequate despite ventilation performed out of synchronization with chest compression (32).

Medical Anti-Shock Trousers (MAST)

The use of MAST in ACLS was discussed, with the suggestion that it not be applied in the routine care of the cardiac arrest victim, due to a lack of data demonstrating an improvement in survival. However, MAST was mentioned as a method to provide a reversible volume load in cases of congestive heart failure with pulmonary congestion. It must be remembered that MAST may provide an increased preload and aggravate pulmonary congestion and is therefore potentially dangerous in this setting. In pulmonary congestion cases where hypovolemia is suspected to be part of the clinical problem, MAST may be beneficial. If the clinical picture deteriorates with inflation, MAST can be deflated. It is emphasized that MAST should only be used in these circumstances by personnel familiar with this physiology and potential complications.

Drug Routes and Delivery

The reliability and speed of drug delivery to target sites when given under conditions of poor perfusion during CPR has been questioned (33-35). In patients without an I.V. prior to the start of CPR, the antecubital is asserted to be the vein of first preference. The distal veins of the hand, wrist and leg were specifically mentioned as the least favorable sites during CPR due to their poor perfusion.

Suggested measures to improve transport of drugs from the extremity veins into the central circulation include using a large volume of flush solution following I.V. drug administration. This would essentially flush the drugs out of the tubing as well as the peripheral vein up to the point where it empties into the larger central veins. Lifting up the extremity to promote gravity drainage into the central blood circuit and use of a longer I.V. catheter were also mentioned. Due to the inherent delays of drug transport from peripheral veins to target sites in the central circulation, a one to two minute period should be allowed following peripheral venous drug administration before assessment of its effects during CPR.

Situations occur where an endotracheal tube is placed the endotracheal route is mentioned as a suitable alternative for administration of epinephrine, lidocaine, and atropine (36). This may be of particular significance in the field, where intubation often preceeds establishment of an I.V. in cases of unwitnessed arrest. The intracardiac route for drug administration is mentioned as an alternative, appropriate only to cases where neither I.V. or endotracheal routes are available.

Central venous access is acknowledged as a drug route with faster central drug delivery and more consistent availability in situations with poor peripheral perfusion. Thus, if circulation is not rapidly restored after initial drug therapy through a peripheral I.V., a central I.V. should be placed. It is important to choose a central I.V. technique that minimizes any interruptions in chest compression or ventilation. With an intubated patient, the internal jugular may be cannulated with less interruption in chest compression than that required for the subclavian. External jugular cannulation can access the central circulation using the J-wire technique and do so without interruption in CPR when an endotracheal tube is utilized.

Drug Therapy

I.V. Fluids – It is acknowledged that electromechanical dissocation is common in cases of arrest secondary to severe traumatic blood loss. Aggressive fluid expansion is indicated. In cases with acute myocardial infarction and hypotension, cautious volume expansion may also be useful but should be done with appropriate monitoring of filling pressures and cardiac output. Volume expansion is not recommended in the cardiac arrest victim without specific suspicion of prior volume depletion, because it may result in an actual reduction of blood flow to the brain and heart (37).

Lidocaine – Lidocaine has been designated as the first-line anti-arrhythmic drug for management of ventricular ectopy. Previous recommendations had given a first-line option between lidocaine and bretylium in management of ventricular fibrillation (VF). This clear preference toward lidocaine is based on research showing lidocaine at least as effective as bretylium in VF (38, 39).

The dosage for lidocaine has been reconsidered. Lidocaine should now be given to the non-arrested patient with significant ventricular ectopy (40) with both an initial bolus of 1 milligram per kilogram and a constant infusion running at 2 milligrams per minute (40). Additional boluses of 0.5 milligrams per kilogram may be given at 2 to 3 minute intervals, as needed to a maximum bolus dose of 3 milligrams per kilogram, to achieve suppression of ventricular ectopy. With each repeat administration of a 0.5 milligram per kilogram bolus, the lidocaine drip rate should be increased by an additional 1 milligram per minute up to a maximum of 4 milligrams per minute (41) (Figure 3). In patients with advanced age (greater than 70 years) or liver disease, lidocaine dosage should be attenuated to half of the normal dosage (40).

When lidocaine is given during VF or pulseless ventricular tachycardia (VT), there is likely to be a decreased clearance of the drug by the liver, similar to that

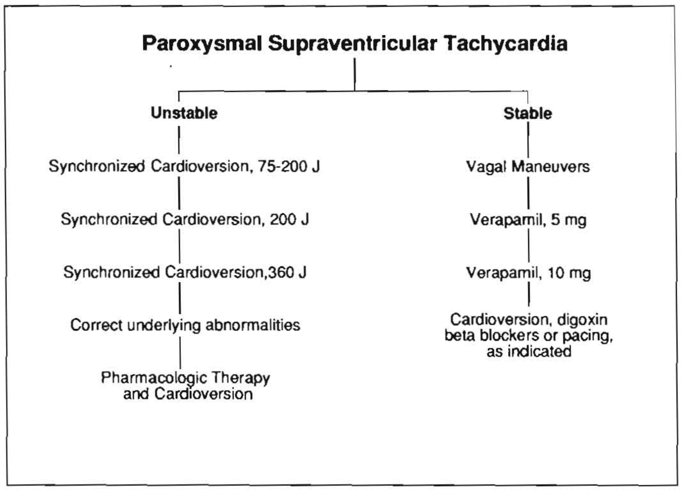

Figure 1 - Paroxysmal Supraventricular Tachycardia

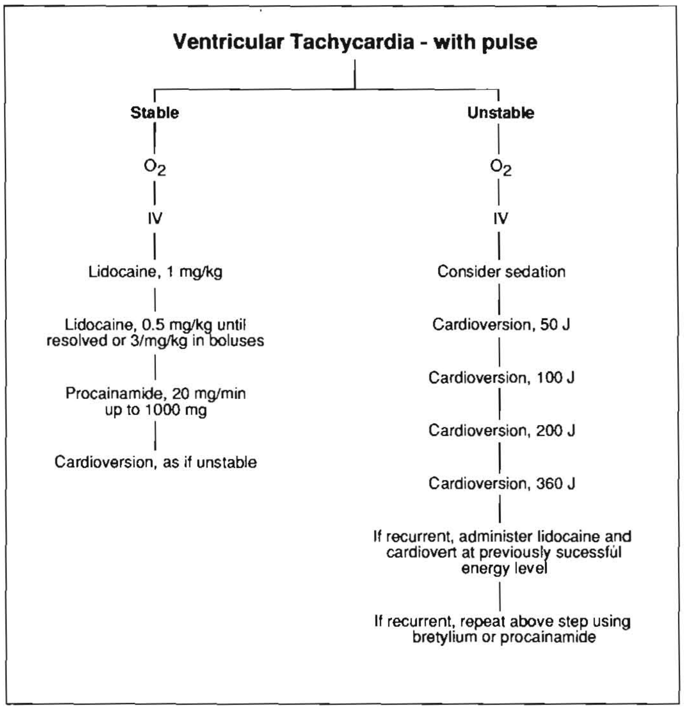

Figure 2 - Ventricular Tachycardia with Pulse

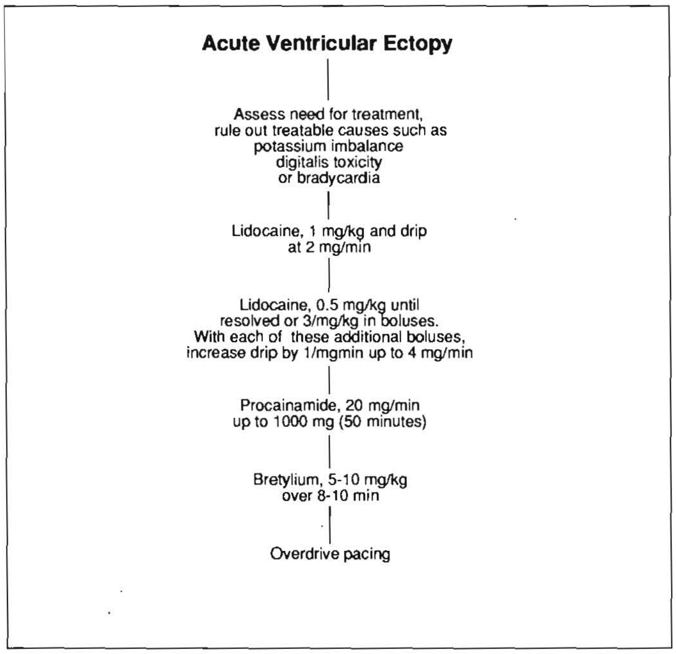

Figure 3 - Acute Ventricular Ectopy

seen with shock and liver disease (42). An initial dose of 1 milligram per kilogram is suggested. A lidocaine infusion is not necessary until the pulse is restored. If VF or pulseless VT remains after lidocaine and countershock, a single 5 milligram per kilogram bolus of bretylium and another countershock may be used. If refractory, administer additional boluses of 0.5 milligram per kilogram boluses of lidocaine with countershocks at 8 to 10 minute intervals to a maximum bolus dosage of 3 milligrams per kilogram (Figure 4).

During early myocardial infarction, ventricular ectopy may not be present, yet a high risk of ventricular fibrillation exists. Thus, lidocaine continues to be recommended with suspected myocardial infarction, on a prophylactic basis, in the absence of ventricular ectopy (43-45).

Procainamide – Procainamide has been included in the management sequence for stable ventricular tachycardia (Figure 2) and acute ventricular ectopy (Figure 3), after repeated boluses of lidocaine have been unsuccesful. The procainamide is given acutely as a drip, running at 20 milligrams per minute. It should be discontinued if the QRS widens by 50% of baseline or after a total of 1000mg of procainamide has been infused (46).

Atropine – Previous algorithms for treatment of asystole limited its dosage to 1 milligram, despite its maximum dosage of 2 milligrams in non-arrested cases (Figure 5). Atropine, 1 milligram, may now be repeated once during sustained asystole, five minutes after the first 1 milligram bolus (47-49) (Figure 4).

Isoproterenol – Isoproterenol has been deleted from the management sequence for asystole and electromechanical dissociation. Specific references to research behind this decision were not given in the latest JAMA paper (3). It is still included in the management of A-V blocks and bradycardia when a pulse is present. This should be used only until a pacemaker becomes available (Figure 5).

Verapamil –Verapamil is the cornerstone of a new algorithm for treatment of sustained paroxysmal supraventricular tachycardia (PSVT) (Figure 1). In the stable patient, it is given after vagal attempts have failed to break the tachycardia. An initial dose of 5 milligrams, intravenously, may be followed, if needed, in 15 to 20 minutes by a repeat dose of 10 milligrams (50). The unstable patient would receive verapamil only if an initial sequence of synchronized countershocks fail. Hypotension following verapamil may be reversed with a 0.5 to 1.0

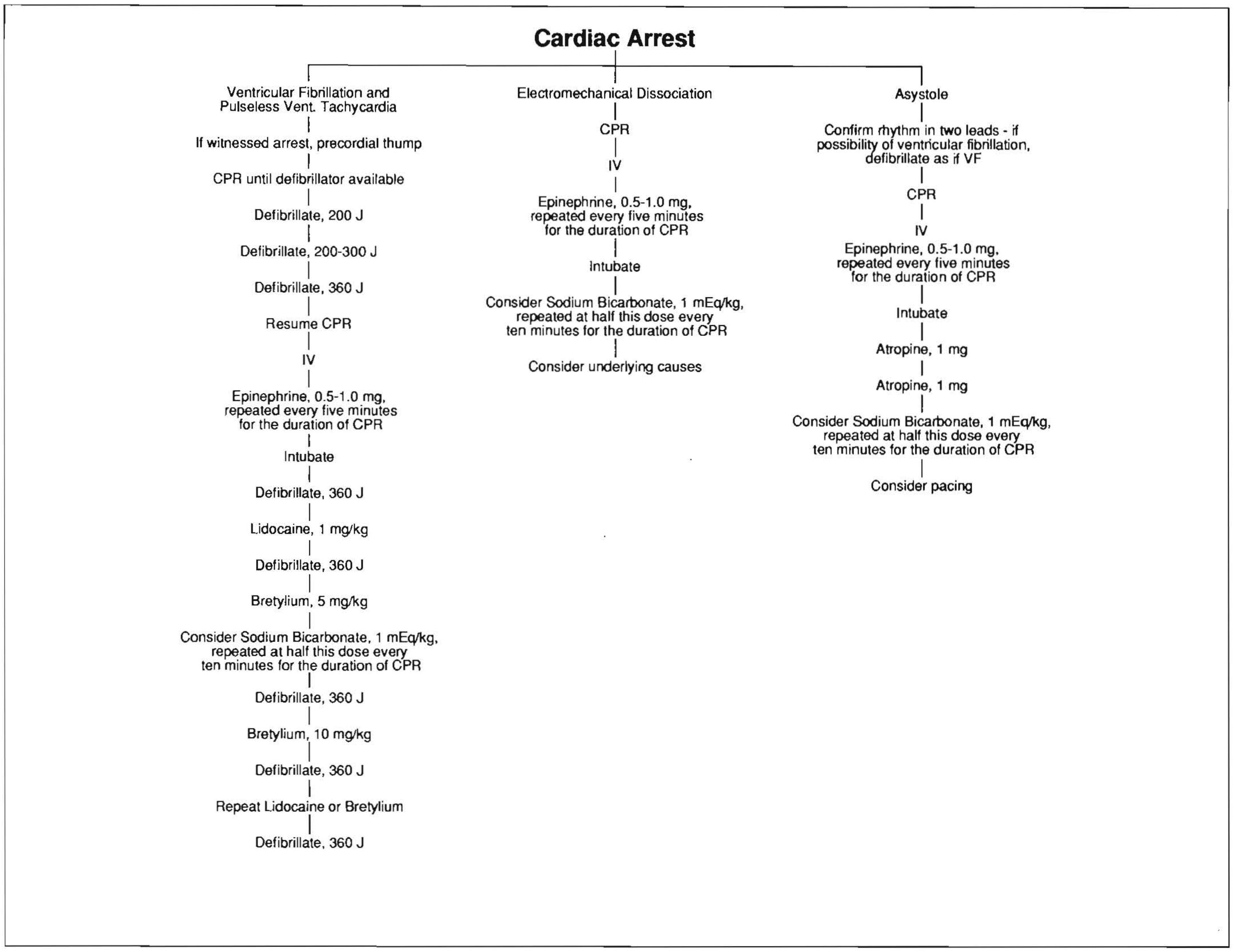

Figure 4 - Cardiac Arrest Algorithms

milligram bolus of calcium chloride.

Dopamine and Dobutamine – These drugs are indicated in the treatment of myocardial infarction with hypotension. However, dobutamine has been portrayed as having preference in first-line treatment, having primarily inotropic effects which will increase the cardiac output. Dopamine, in contrast, has inotropic effects, but also has a more pronounced vasoconstrictive effect which may impair mesenteric and renal blood flow at its higher dosages (51).

Amrinone – This drug is now included in the official ACLS armamentarium, with effects similar to those of dobutamine, yet with a non-adrenergic mechanism (52). Use of this drug is not specifically addressed in any of the clinical management sequences.

Calcium – Calcium has been removed from the routinely recommended management of asystole and electromechanical dissociation. Research studies have failed to show an improvement in survival from asystole or electromechanical dissociation when calcium was utilized (53-55). Calcium has been implicated to cause adverse

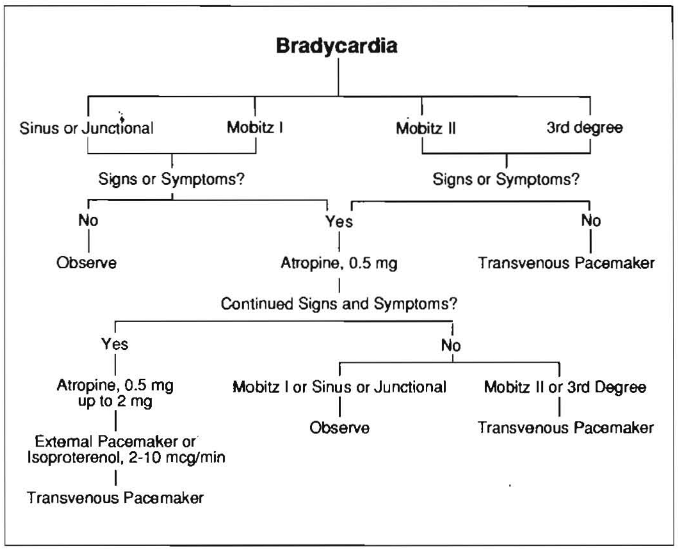

Figure 5 - Bradycardia Algorithm

effects on the brain during and following resuscitation (56, 57). It may be appropriate for use in less common situations with electrolyte imbalances or calcium channel blocker toxicity.

Nitroglycerin – The time honored use of sublingual nitroglycerin remains a part of ACLS. New is its use in I.V. form. It is the preferred agent, over nitroprusside, in treatment of hypertension without congestive failure and in those patients with ischemic heart disease. Nitroglycerin is less likely than nitroprusside to exacerbate ischemia (58). It may be administered intravenously as a constant infusion of 10-500 mcg/min, with a usual effective range of 50-200 mcg/min. Hemodynamic monitoring is required for the safe use of this agent.

Sodium Nitroprusside – This is a new agent for ACLS, indicated in the treatment of heart failure and hypertension. It is similar to I.V. nitroglycerin as a potent vasodilator. It is preferred over I.V. nitroglycerin in cases of hypertension with congestive heart failure (58-60). It is administered at a rate of 10-20 milligrams/min, under careful hemodynamic monitoring.

Sodium Bicarbonate – The role of this drug in resuscitation has been deemphasized. A considerable body of research has shown that sodium bicarbonate may fail to correct acidosis at the cellular level - it may even exacerbate it (61-71). Sodium bicarbonate may produce other adverse complications such as severe hyperosmolality and hypernatremia (72). Endotrachial intubation and effective ventilation seem to provide the best management for acidosis.

Unless there is a strong suspicion of a preexisting metabolic acidosis, more so than that normally expected with an unwitnessed arrest, sodium bicarbonate should not be routinely administered. It should be considered to be a tertiary intervention, behind BLS and the antiarrythmic measures of drugs and countershocks (Figure 4).

COUNTERSHOCKS AND PACING

Defibrillation

In recognition of changes in electrical impedance with repeated shocks (73-88), an initial sequence of three consecutive unsynchronized countershocks should be used in ventricular fibrillation and pulseless ventricular tachycardia. The initial energy level for the adult should be 200 joules (89-99), with the second and third countershock energy levels at 200-300 and 360 joules, respectively (Figure 4).

Pacing

Despite the development of external non-invasive pacing systems, pacing has not demonstrated clinical efficacy during resuscitation (100). However, these same devices do show promise for patients with a pulse in hemodynamically significant bradycadia or blocks that are refractory to atropine (101) (Figure 5).

ADULT TREATMENT ALGORITHMS

Figures 1-5 present treatment algorithms based on those which appear in the 1986 JAMA paper (3).

PEDIATRIC AND NEONATAL ADVANCED CARDIAC LIFE SUPPORT

The advanced life support sequences for treatment of the arrested child are generally the same as those now recommended for the adult. However, atropine is no longer recommended in the acute phase of neonatal resuscitation.

I.V. Access

The difficulty in obtaining intravenous access during resuscitation of children is recognized. Due to more reliable drug delivery to the heart and brain during CPR, central venous access is preferred (102).

The technique of intraosseous (bone marrow) infusion has been acknowledged as an acceptable emergency drug administration route. Intraosseous injections of catecholamines (103) and sodium bicarbonate (104) have demonstrated rapid absorption. In the neonate, the umbilical vein is the preferred route of vascular access.

CONCLUSION

As research continues to provide a better understanding of the pathophysiology of acute cardiovascular diseases, new approaches to clinical management will be developed. The American Heart Association has taken a leadership role in providing much needed interpretations of scientific debate bringing about consensus recommendations for general clinical care. These efforts constitute a major component of emergency and critical care medicine.

REFEREE COMMENTARY

Edward J. Straub, M.D. (Cardiology, Memorial Hospital, Tampa) - I think that a comment should be made regarding ventilation being the corner stone of improving acid-base balance in the cardiac arrest patient. Previous enthusiasm for correcting acidosis with sodium bicarbonate has now subsided in favor of correcting the ventilatory component. When discussing drugs and catecholamines, no mention was made of the use of epinephrine. The new guidelines of advanced life support underscore the importance of the alpha and beta attributes as being salutary and that as a result, epinephrine is the chief catecholamine recommended.

Joe Nelson, D.O. (Emergency Department, Carrollwood Community Hospital, Tampa) - Although regurgitation of stomach contents is a serious problem in BCLS, I question the safety of digital pressure on the cricoid cartilage (Sellick manuver) as a routine BCLS procedure. This manuver was primarily brought about as a tool for "crash" intubation under direct laryngoscopy, although it has been used in pediatric bag-valve-mask resuscitator techniques. Medical professionals not trained in anesthesia and not using this procedure on daily basis may actually do the patient as disservice by 1) closing the airway with too much cricoid pressure, 2) inadvertently impairing a good head-tilt/chin-lift, also closing the airway and 3) misplacement of the fingers on the neck, resulting in impaired carotid blood flow for dangeroud periods of time. More investigation is needed before this manuver is routinely used as a BCLS procedure. Esophageal obturator aiways have never been universally accepted as a first line substitute for endotracheal intubation, although they continue to be a good temporary back-up device, when endotracheal intubation has been unsucessful. The new ACLS guidelines merely reflect an increased emphisis on endotracheal intubation as the only acceptable primary advanced airway adjunct for use during cardiac arrest, specific neck injury or obstructed airway problems not withstanding. Medical anti-shock garment (MASG) use was incorrectly stated in the article. It is inappropriate to utilize the MASG in congestive heart failure. The device is, as you state, beneficial in cases of cardiac arrest with suspected hypovolemia, as a reversible "fluid challenge." Temporary cardiac pacing, either internal or external, should be instituted as early as possible and is preferred over isoproterenol in cases of symtomatic bradycardia refractory to atropine. Isoproterenol can exacerbate myocardial ischemia and cardiac arrythmia.

Mr. Gunderson's response - I agree with Dr. Straub that ventilation should now be considered to be the primary means of acid-base control during resuscitation. This point bears additional emphisis. However, I am concerned about the lack of quantification of ventilation during most resuscitation efforts. While it is easy enough to say that one should provide adequate alveolar ventilation, what does this mean to the paramedic in the field without blood gases or the means to measure delivered tidal volume? The alpha effects of epinephrine are widely regarded in the research literature as being responsible for its beneficial effects during resuscitation. There is controversy about the benefits of its beta attributes. Both these points are made in this latest J.A.M.A. paper. The ACLS textbook based on the 1980 standards made some discussion of the value of pure alpha agonist administration with mixed reviews. Dr. Joeseph Redding has conducted much of the original research along these lines. The new guidelines give additional emphsis to the q5m dosage regimen for epinephrine during CPR. The application of cricoid pressure as a means of both preventing gastric distention during ventilation and for the prevention of regurgitation is presented in the paper for use by "health care professionals in two-rescuer CPR situations; its application is simple but requires an assistant." It would seem the burden is on the medical directors and training staffs to provide an appropriate introduction of this technique that avoids the pitfalls mentioned by Dr. Nelson. I don't percieve this to be that different than many of the other procedures we routinely use in regards to a risk-benefit ratio. I might restrict its use in the field to those who serve in primary EMS roles and withhold training of first responders, etc. until we have some experience with it. The EOA is used by many systems as a primary advanced airway adjunct to the exclusion of endotracheal intubation. While previous standards highlighted the preference of endotracheal intubation, they did not make specific mention that endotracheal intubation should be used in the field in preference to the EOA. I have a problem with having paramedic students learn to manage an airway during their ACLS resuscitation training by first inserting an EOA and later performing endotracheal intubation. The endotracheal tube should be used as a first line advanced airway adjunct after initial ventilation and perhaps a simple oralpharyngeal aiway. That's the message. I don't think we're in disagreement. The use of MAST in cases of heart failure might play a potential role only in those cases where hypovolemia is considered to be a factor contributing to the reduced cardiac output. Typically, these pateints have too much preload. If hypovolemia is suspect on the basis of perhaps of an indicator such as pulmonary artery wedge pressure, MAST might offer a means to give a reverisble fluid challenge. If the hemodynamics deteriorate, it can be withdrawn. That isn't so easy to do with a crystalloid fluid challenge. I wholeheartedly agree with Dr. Nelson's comments about pacing and isoproterenol - they bear further emphisis. I would like to thank Dr. Straub and Dr. Nelson for their remarks.

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Tampa Bay EMS Journal
Volume 1, Number 1
July-September, 1987
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Equipment Review

Lifepak 5 ECG / Voice Recorder

Reviewer: MICHAEL BROWN, REMT-P
Hillsborough County Emergency Medical Services

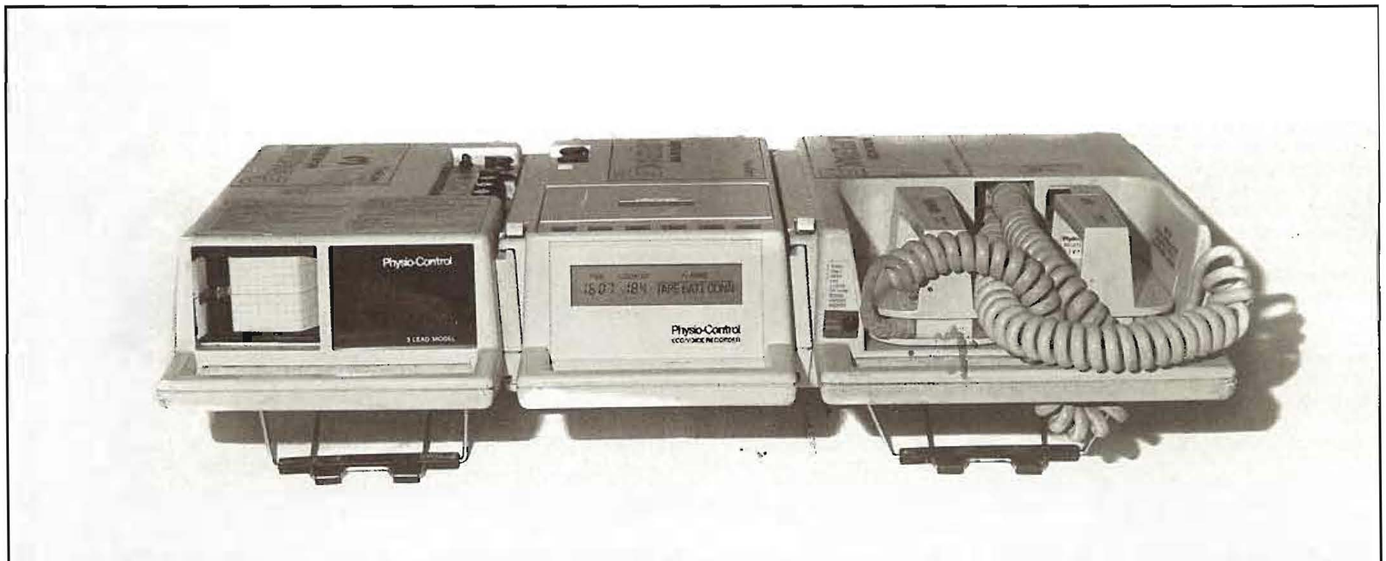
Strict records-keeping during the initial task-intensive minutes in the care of critically ill patients is often a secondary concern. This is particularly true of prehospital care, where there is often insufficient manpower to designate someone to record events during the initiation of care. While approximated records of this crucial period can be rendered, the inherent imprecision and frank loss of data with records made "after-the-fact" severely limit their reliability for retrospective medical review or research purposes. The PhysioControl ECG/Voice Recorder, used with the Lifepak®5, Defibrillator/Monitor, provides an elegant solution to the problem of documentation during acute situations. Properly used, the ECG/Voice Recorder can provide a continuous and precise record, either self contained or by transcription, of patient encounters in the field or hospital.

DESCRIPTION

The Lifepak®5 ECG/Voice Recorder is essentially a portable cassette recorder that simultaneously and continuously records both audio from the scene and the patient's ECG on a standard cassette tape. The unit is designed to fit between the Lifepak 5 monitor and defibrillator modules (Figure 1). This allows monitoring, recording, and defibrillation to be performed with a single, integrated unit. The recorder's ECG channel interfaces automatically with new Lifepak®5 monitors that have two sets of electrical contacts where the modules slide together. ECG interface with older monitors requires the use of a cable from the ECG signal jack (used for telemetry) to the recorder unit. The voices and sounds from the scene are recorded with a condenser microphone located on the top of the recorder chassis. A speaker for playback is located on the bottom of the chassis.

The recorder control panel features membrane switches and a lighted LCD display for the real time clock, tape counter, and visual alarm indicators. The recorder alarm system alerts the operator by audible and visual signals to low battery power, interruption of tape transport, or loss of ECG signal. The recorder module utilizes the standard Lifepak®5 system batteries. An additional 1.5 volt "AA" battery is used to run the real time clock, independently of the rest of the Lifepak®5 battery system.

The ECG/Voice Recorder alone weighs 6.6 lbs. and adds 6.8 inches to the overall width of the defibrillator-monitor system.



D. Kornegay

Figure 1 - Lifepack 5 ECG / Voice Recorder, shown in place between oscilloscope and defibrillator modules.

OPERATION

Operation of the ECG/Voice Recorder is an uncomplicated and almost totally "hands-off" procedure. Unlike periodic recording where the ECG strip writer must be manually switched on and off with times and events written on the strip and/or a separate record, the ECG/Voice Recorder runs continuously. The clinicians simply vocalize the time, their actions and observations as they occur.

The audible and visible alarms assist the operator in assuring that battery power, tape transport, and ECG signal are uninterrupted. Actuation of any of the recorder controls triggers an audible confirmation signal and a red status indicator light, incorporated into each of the control switches. When the ECG/Voice Recorder is used with newer Lifepak®5 monitors, turning on the monitor will automatically activate audio and ECG recording. Replay is accomplished by pressing the "playback" switch three times within five seconds. The tape position counter is reset by pressing the "stop" switch twice. The recorder can be shut off by pressing the eject switch. Alternatively, the unit will shut off automatically if left in the "stop" mode for over ten minutes. When an alarm condition occurs, the audible alert persists only a few seconds, but the visual indicator will continue to be displayed on the LCD panel until the problem is corrected or the unit is shut off. The controls of the ECG/Voice Recorder are clearly marked and operation of the unit is a logical and straightforward process.

DISCUSSION

Medical event recording with the ECG/Voice Recorder has unique advantages and drawbacks. By virtue of it's continuous operation, all ECG events are recorded. When coupled with clear vocalizations of events, observations, and times, very sophisticated records can be made. Further, recording with this precision can be accomplished without diverting rescuers from the primary tasks involved in critical situations.

There are drawbacks to electronic recording. Continuous hard copy records of the ECG can be more rapidly reviewed, but are cumbersome to handle in the field. The time required to review or make transcriptions from the tape equals or exceeds the time period recorded on the tape. Alarm situations, if not expeditiously corrected, will result in loss of data. In practice, this can easily happen when all personnel are actively engaged in activity when an alarm condition occurs. The increase in size and weight of the recorder/monitor/defibrillator package, while significant, may be offset by the advantages gained by electronic recording.

SUGGESTIONS

When considering the ideal characteristics of an electronic data recorder, several features suggest themselves. An auto-reverse on the tape drive would eliminate the temporary loss of data incurred by manually ejecting, flipping and re-inserting the cassette. The need to vocalize real time could be eliminated if real time was electronically recorded onto the tape and displayed during tape playback. The time required to review the tape or make transcriptions could be significantly reduced if there was a method for rapid review of the tape similar to the method for review of Holter monitor tapes. Alternatively, a manually triggered electronic event marker might allow rapid, automatic advance of the tape to significant events. Lastly, audible alarm signals would be more informative if each had its own unique sound, all of which must be distinguishable from the QRS beeper on the ECG monitor.

CONCLUSION

The PhysioControl ECG/Voice Recorder, used with the Lifepak®5 defibrillation/monitor, is an excellent device for acquiring complete and precise records during both hospital and prehospital care. The complete and accurate records obtained with this device makes it one of the best adjuncts currently available for documentation of critical care. These records can facilitate accurate data acquisition for research, quality assurance, and medical review programs.

Tampa Bay EMS Journal
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Volume 1, Number 1
July-September, 1987

Book Review

Problem Solving In Cardiac Arrest
By Ken Grauer, M.D.

There is now a refreshing departure from the ACLS textbooks on medical resuscitation of the arrested patient, *Problem Solving In Cardiac Arrest*. This book addresses nursing, paramedic and physician audiences with an energetic style and unusual clarity that will appeal to novice and veteran clinicians alike.

The reader is faced with a challenge to integrate their past experience with a presentation of new material. A format of progressive complexity discourages "skimming" and premature advancement. This limits the value of the book as a reference, but enhances its effectiveness as an instructional tool. Although it uses a workbook form, this approach is kept to a bearable minimum. A formal workbook, *Interpreting ECGs*, is an excellent complementary publication.

Problem Solving in Cardiac Arrest is basically divided into the following sections – Overview of Resuscitation Algorithms, Humanistic Insight Into Common Misconceptions of Management Techniques, Drug Dosages, Calculations and References Compendium and Problem Solving of Clinical Resuscitation Problems.

There are several areas covered in the book which this reviewer found to be particularly well done. Theses areas include, but are not limited to, tachydysrhythmias, carotid sinus massage, analysis of the Ashman phenomenon and the clinical consequences of its misdiagnosis. An in-depth discussion of differentiation between ventricular ectopy and atrial ectopy with aberrancy was, for this reviewer, the highlight of the dysrhythmia identification section.

In subsequent publication revisions, a greater stress on initial physical evaluation, drug calculation and a segment on the decision to resuscitate may be of benefit to many new providers.

In the final portion of the book, the reader is put in charge of a code team. They are presented with scenarios requiring initiative and stumbling blocks in need of solution. If the reader has followed the text, he or she is made to feel that their knowledge, independent of simple following of an algorithm, can make a difference.

Grauer, K: *Problem Solving In Cardiac Arrest*. C.V. Mosby Company, St. Louis, 1984. 212 pages, softcover, $19.95.

Reviewer: Thomas P. Jeffery, Jr., REMT-P
Hillsborough County EMS

Volume 1, Number 1
April-June, 1987

Career Profile

Raymond McLane, M.D.

By: Patrick Shepler, REMT-P
Clearwater Fire Department

Great strides in medical discoveries are made during every war, and Vietnam was no exception. For the last several years, formal residency training for emergency department physicians has been available. However, there are established emergency medicine physicians whose preparation is difficult to rival even the best of these residencies. Such is the case with Dr. Raymond McLane, Director of the emergency department at Bayfront Medical Center in St. Petersburg and an Associate Medical Director for the Pinellas County EMS system.

McLane's interest in medicine began as a young boy while watching a physician suture his thumb. His inspiration came in high school, listening to a lecture at Notre Dame University by Dr. Tom Dooley, a missionary physician who established hospitals to serve the thousands of Vietnamese refugees fleeing to Laos in the 1950s.

In 1957, McLane attended Notre Dame as an undergraduate, followed by medical school at Indiana University at Indianapolis and internship at Mound Park Hospital in St. Petersburg.

Following internship, he was formally inducted into the U.S. Army. His first assignment was the training of medical corpsmen. McLane was then transferred to Vietnam, where he assumed command of a medical company, composed of ninety medics and nine physicians in a sixty bed facility, fifty miles south of the demilitarized zone. Many of his patients were villagers, treated prior to transfer to a Vietnamese hospital. Their primary mission, however, was treatment of American G.I.'s brought directly from the battlefield by helicopter - the early prototype of today's civilian trauma system goals. His experience in Vietnam gave him the opportunity to become skilled at all the things he altruistically "hopes never to have to do again - mass casualties, triage, blast injuries."

In 1969, McLane returned to the emergency department of Bayfront Medical Center, formerly called Mound Park Hospital, where he had interned. Partly due to Vietnam, changes in emergency medicine had begun. Instead of interns, career physicians staffed the emergency room. A formal training program for a new allied health career, emergency medical technician, was being introduced. Dr. McLane participated in the training of firefighters from St. Petersburg in the new emergency medical technician program. When paramedic level training was later implemented, McLane became the Medical Director for the St. Petersburg Fire Rescue Division, and wrote their original standing orders. In the years following, Dr. McLane has remained responsible for St. Petersburg Fire Rescue as an Associate Medical Director in the Pinellas county EMS system.

McLane is a member of the American College of Emergency Physicians, Pinellas County Medical Society, and the American Medical Association. In 1984, he was the recipient of the President's Award from the Pinellas County Medical Society.

McLane feels that liability is the biggest problem facing EMS now and in the future. "Unless there is a general tort reform, EMS is the next 'deep pocket' malpractice attorneys will go after."

On the positive side, McLane feels that the Basic Trauma Life Support (BTLS) course has also resulted in a more standardized, efficient level of trauma care. Patients are arriving quicker and more packaged than before. McLane's "LBJ Trauma Fellowship" (sic) in 1967 convinced him that the initiatives promoted in BTLS courses must inevitably reduce morbidity and mortality from trauma. He has worked to bring this method of medicine from the battlefields of a country at war onto the streets and into the hospitals of the United States.

Abstracts

Compiled by: Journal Staff

Pulmonary Barotrauma During Cardio-pulmonary Resuscitation

HILLMAN K, ALBIN M

Critical Care Medicine 14: 606-609, 1986

Four case studies of pulmonary injury attributed to artificial ventilation during CPR are presented. Several problems leading to dangerously high peak airway pressures are revealed, including a faulty one-way valve on a ventilation device, vigorous chest compressions, and bag ventilations without airway pressure limiting valves. Discussion is given to the mechanics of barotrauma and the pathophysiology of its consequences. The authors suggest pulmonary barotrauma may be an underestimated complication of resuscitation. Caution is given against simultaneous compression and ventilation as it may cause sufficient peak inspiratory pressure to cause barotrauma. A recommendation is made to require that CPR equipment have airway pressure limiting valves. Clinicians are urged to look for signs of barotrauma during CPR and to consider X-ray after CPR to exclude mediastinal emphysema and pneumothorax.

Glucagon Reversal of Hypotension in a Case of Anaphylactoid Shock

ZALOGA GP, DELACEY W, HOLMBOE E, CHERNOW B

Annals of Internal Medicine 105: 65-66, 1986.

A case report and discussion are given for a 75 year old patient who developed hypotension, diaphoresis, uticaria and complained of itching within minutes following injection of radiocontrast dye for CT scan. CT was to be performed as part of evaluation of vertigo. This patient had a past history of angina, and essential hypertension with prescription medications including atenolol. The patient was given a 1mg. bolus of epinephrine, IV, with minimal improvement in blood pressure. This poor response suggested presence of beta-blockade. Five minutes following the epinephrine, a 1 mg. bolus of glucagon was given. The MAP increased from 55 to 75 mm Hg and later went back down to 60 mm Hg. Another 1 mg. dose of glucagon was given along with Methylprednisolone and Diphenhydramine and initiation of a glucagon drip at 1mg./hr. These interventions restored and maintained normal arterial pressure. Glucagon infusion was gradually discontinued. The patient was observed for 24 hours and discharged. Discussion is made of epinephrine and possible ineffectiveness in patients taking beta-blockers. Glucagon has potent inotropic and chronotropic actions that are only minimally antagonized by beta blockers. The authors conclude glucagon may be effective in arousal of anaphylactic hypotension in patients taking beta-blockers.

Wide Complex Tachycardia: Misdiagnosis and Outcome After Emergent Treatment.

STEWART RB, BARDY GH, GREENE HL

Annals of Internal Medicine 104: 766-771, 1986.

The problem of differential diagnosis between ventricular and supraventricular tachycardia with wide QRS complexes in 12 lead ECG analysis were studied in 46 consecutive episodes of wide QRS complex tachycardia (QRS> .12 sec., rate> 100 beats/min.). The mechanism of tachycardia, ventricular or supraventricular, was later established by programmed stimulation, esophageal leads or other electrophysiologic studies. Of the 46 wide QRS complex tachycardia episodes, 8 were found to be supraventricular and 38 were ventricular tachycardias. Supraventricular tachycardia with aberrant conduction was correctly diagnosed in 8 of 8 episodes (100%). Ventricular tachycardia was correctly diagnosed in 23 of 38 episodes (61%). Ventricular tachycardia was misdiagnosed in 15 of 38 episodes (39%). Ventriculoatrial dissociation was apparent in 11 of the 15 misdiagnosed tracings. Misdiagnosis 13 of 15 cases lead to administration of verapamil, all of which suffered significant clinical deterioration. This investigation lead to conclusions that wide QRS complex tachycardia is often misdiagnosed by 12 lead ECG, that when a tachycardia is of ventricular or uncertain origin, verapamil is potentially dangerous, and that tachycardia of uncertain origin should be treated as ventricular tachycardia until proven otherwise. Request reprints from Gust H. Brady, M.D., Division of Cardiology, Harborview Medical Center, 325 Ninth Avenue, Seattle, WA 98104.

A Method For Evaluating Field Triage Criteria

WEST JG, MURDOCK MA, BALDWIN LC, WRALEN T

Journal of Trauma. 26: 655-659, 1986

A retrospective study was conducted on the Orange County, California, EMS system to determine rates of over and under triage of patients to trauma centers. Overtriage was defined as cases in which the patient was hospitalized for less than 3 days and in which their ISS score was later found to be either less than 10 or 15, depending on which study protocol was used. Undertriage was defined as a ratio of non-CNS MVA deaths in non-trauma center hospitals divided by the total number of non-CNS deaths times 100. The study included all paramedic responses from August 1983 through July 1984. Two different field triage criteria were employed, differing in their sensitivity in detecting potentially serious injuries and in their rates of over and undertriage. Discussion provides insight into how medical control might attempt to balance items included in field triage criteria to obtain acceptable sensitivity of serious injuries needing trauma center care while minimizing the political and economic consequences of overtriage of less severe cases past appropriately capable non-trauma center hospitals. Request reprints from John West, M.D., 1310 W. Stewart Dr., Suite 603, Orange, CA 92668

Preventable Trauma Deaths: Dade County, Florida.

KRIES DJ, GUSTAVO P, AUGENSTEIN P, et al.

Journal of Trauma 26: 649-654, 1986.

All trauma deaths for 1982 in Dade County, Florida were retrospectively examined by a panel of at least four full-time trauma surgeons from the University of Miami/Jackson Memorial Medical Center. Their objective was to evaluate the need for an organized trauma network. There were 1,201 total cases, of which 715 (59.5%) were dead on the scene. The remaining 486 cases included 240 (49.4%) deaths resulting from CNS injury and were excluded from further study. This study concentrated on the 246 (50.6%) non-CNS cases in which death occurred after transport from the scene. The panel determined 52 (21.1%) of the non-CNS deaths were preventable. Failure to perform a procedure or a delay in surgery accounted for 43 (82.7%) of these preventable deaths. The lone Level 1 trauma center in operation in 1982 had a non-CNS preventable mortality rate of 12.1%. The non-CNS preventable mortality rate for the 22 non-trauma centers was 26.4% ($p=<0.01$). The authors conclude that severely injured patients should be triaged directly to trauma centers and that an organized trauma center for Dade County, Florida is justified.

Trauma Center Verification

MAULL KI, SCHWAB CW, MELLENAY SD, et al.

Journal of Trauma 26: 521-524, 1986.

This paper describes a process used in the state of Virginia to verify the performance of trauma centers, 2 years following their formal designation. Designation had concentrated on capabilities whereas verification centers upon performance following designation. This verification process attempted to measure institutional commitment, assess deficiencies and give valid peer review. Verification criteria were based on the 1983 American Colleges of Surgeons Guidelines for Hospital and Prehospital Trauma Resources. The verification was conducted by a multidisciplinary team with surgical, emergency medicine, critical care nursing and hospital administration representation. The verification site visit, after documentation review, required one half day. Site visit activities include a tour, chart reviews and interviews with residents, nurses, emergency medical technicians and anesthesia personnel. Failure of verification results in a re-visit in 6 months to check for correction of deficiencies. Failure at revisitation results in formal notification to the hospital and all concerned agencies within the region. The authors conclude that its verification program has been successful in sustaining the momentum of improvements in trauma care. Request reprints from Kimball Maull, M.D., Dept. of Surgery, University of Tennessee, 1924 Alcoa Highway, Knoxville, TN 37520.

Esophageal Intubation: A Review Of Detection Techniques.

BIRMINGHAM PK, CHENEY FW, WARD RJ

Anesthesia and Analgesia 65: 886-891, 1986

A collective review was conducted of techniques used to detect inadvertent esophageal intubation during efforts to intubate the trachea. This paper reveals that esophageal intubation has been a significant factor in anesthesia related deaths. Each of the many available techniques for confirmation of tube placement are separately discussed, outlining their respective strengths and limitations. Strongly favored in this review were the direct visualization of tube passage through the vocal cords and measurement of carbon dioxide in exhaled gases by an on-line gas analyzer, such as a capnograph or mass spectrometer. The expired gas method works on the basis that CO2 is present in exhaled gases but is not present in escaping esophageal gases. The authors advocate use of on-line CO2 analysis as a technique to be employed routinely, wherever possible.

Acute Cardiac Events Temporarily Related to Cocaine Abuse

ISNER JM, ESTES N, THOMPSON PD, et al

New England Journal of Medicine 315:1438-1443, 1986.

Case reports and discussion are used to explore the intranasal complications of recreational cocaine abuse. Their evidence contradicts commonly held notions among many physicians that cocaine taken by the intranasal route is safe. The authors conclude that 1) Cardiac consequences of abuse can occur with intranasal administration 2) Underlying heart disease is not a prerequisite for cardiac complications of cocaine abuse 3) Seizure activity can occur without cardiac toxicity from cocaine 4) Cardiac consequences are not limited to massive doses. The authors suggest the medical consequences of abuse are of equal importance to the well documented psycho-social consequences. Request reprints from Jeffery Isner, MD, Tufts-New England Medical Center, 171 Harrison Ave, Box 70, Boston, MA 02111.

Ventilation Skills of Emergency Medical Technicians: A Teaching Challenge for Emergency Medicine

CUMMINS RO, AUSTIN D, GRAVES JR, LITWIN PE, PIERCE J

Annals of Emergency Medicine 15: 1187-1192, 1986.

Concern has been expressed in the medical literature regarding the ability of basic emergency medical technicians (EMTs) to adequately ventilate non-intubated cardiac arrest victims with a bag-valve-mask device. A study was made of 64 EMTs, most of which were full-time paid firefighters, during previously scheduled continuing education classes. Their skills in ventilation were assessed with a calibrated recording CPR manikin using a bag-valve-mask in comparison to pocket mask devices while chest compressions were in progress. In a preliminary skills assessment, it took an average of 4.8 ventilation attempts to make the manikin flash its green light, indicating adequacy with an 800ml or greater tidal volume. This is in contrast to an average of only 2.9 attempts required for the pocket mask ($P<.01$). In a two minute skills test, bag-valve-mask ventilation averaged 8.3 adequate ventilations per minute versus 9.9 per minute with the pocket mask ($P<.01$). In an extended ten minute test, there were no significant differences between the two methods. The authors conclude that specific performance objectives of at least 10 ventilations per minute of at least 800ml each be required during chest compressions.. Given the demonstrated ability of EMTs to learn other sophisticated medical skills, the bag-valve-mask should not be removed from the EMT armamentarium. Request reprints from Douglas Austin, King County EMS, Room 508, Smith Tower, 506 2nd Ave., Seattle, WA 98104

Environmental Concentrations of Nitrous Oxide in a Modular Ambulance.

BRISTOW A, GIESECKE AH, THAL E, ATKINS J

Critical Care Medicine 14: 815-816, 1986.

The prehospital administration of 50% nitrous oxide with 50% oxygen has demonstrated analgesic efficacy in a variety of traumatic and medical conditions. However, concern exists over the hazards it may pose to prehospital medical personnel in the confines of an ambulance, in terms of impaired clinical skills, fetal defects (if attendant is pregnant) and various malignancies. The concentrations of nitrous oxide in various locations of a type I ambulance were measured during patient care by an on-line infrared spectrophotometer in the ambulance while stationary at the scene and in transport, with and without operation of the air conditioning (AC) units. The rear patient compartment windows were open in both cases. The maximum time weighted concentration of nitrous oxide recommended by the National Institute for Occupational Safety and Health is 25 parts per million (PPM). On the bench seat beside the patient without AC, the nitrous oxide levels reached 238 and 243 PPM stationary and in transit, respectively. With the AC, the concentrations reached 56 and 52PPM stationary and in transit, respectively. The authors conclude that patient compartment levels of nitrous oxide may exceed maximum recommended levels. They recommend that the AC blower fan be used whenever nitrous oxide is administered and that a scavenger system be developed to prevent high ambient nitrous oxide levels. Request reprints from Dr. AH. Giesecke, Department of Anesthesiology, University of Texas Southwestern Medical School at Dallas, 5201 Harry Hines Boulevard, Dallas TX 75235.

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Volume 1, Number 1
April-June, 1987

Editorial

EMS Continuing Education - By Merit Badge or Comprehensive Review?

For many years, the American Heart Association's Advanced Cardiac Life Support (ACLS) course was the only nationally recognized continuing education curriculum for EMS. The American Heart Association did an outstanding job developing and implementing this program.

Early on in the emergence of EMS, the ACLS program had little competition in continuing education/national certification programs. This is no longer the case. Trauma is now finding the kind of attention that cardiac care had in the 70's. In the 1980's, the American College of Surgeons introduced the Advanced Trauma Life Support (ATLS) course for physicians. A group from the Alabama Chapter of the American College of Emergency Physicians modified that curriculum for paramedics and nurses, calling it the Basic Trauma Life Support (BTLS) course. The National Association of Emergency Medical Technicians in conjunction with the American College of Surgeons later implemented a paramedic trauma course, called the Prehospital Trauma Life Support (PHTLS) course. Prehospital EMS now has ACLS, BTLS and PHTLS. Is that enough? Modified versions of these courses are rumored for pediatrics. Will we see PACLS, PBTLS and PPHTLS?

Cardiac and trauma care account for most of the cases in prehospital EMS, but not all. Will there be similar national standard curricula for other areas? Pediatric versions?

A series of separate national standard continuing education programs has advantages. It allows the academic communities of the various specialties to establish standards of EMS care for their disciplines. Each is responsible for course content, faculty and testing standards. This relieves EMS agencies and individual hospitals of the need to independently develop courses in those fields.

If continuing education became focused upon those specialty courses, subjects outside those courses could easily become neglected. Compare the amount of training and emphasis given to paramedics over the past ten years in trauma training, without a national standard curriculum, to that received in cardiac care with a curriculum during that same time period.

These national standard courses are usually not free. Tuition, books, and possibly travel and per diem can amount to a considerable expense per course. What will the financial burden become with pediatric versions of the courses? What of other national standard specialty courses for EMS in the future? Where will we put all of the wallet cards?

A continuing education program based on a series of discrete specialty courses can lack continuity. Such a scheme is likely to suffer in putting together aspects of trauma, cardiac, psychological and other care that must be integrated in managing real patients who usually have more than one isolated problem.

What will we have to do to maintain complete credentials if the trend of multiple separate national standard courses continues?

Would it be possible to develop a comprehensive national standard continuing education curriculum for EMS? The American College of Emergency Physicians has done so for its physicians with the Comprehensive Review of Emergency Medicine (CREM). Consider taking the best of both approaches - A comprehensive review course for EMS with the specialty groups taking responsibility for developing segments pertaining to their disciplines. The multidisciplinary emergency medicine specialty could develop the later segments which bring all the pieces together into a practical overall approach to prehospital patient care. Final didactic and clinical evaluation should reflect the goal of caring for the whole patient.

Michael R. Gunderson, REMT-P
Editor

Guidelines For Authors

Please submit all materials to be considered for publication to:

Michael R. Gunderson, REMT-P
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Acute Care Foundation, Inc.
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Tampa, FL 33682-0173
(813)932-3659 or 937-8454
Compuserve Electronic Mail - User I.D. # 73067,207

ACADEMIC PURPOSE AND REVIEW

The *Tampa Bay EMS Journal* is an academic publication serving the emergency medical community. It is published by the Acute Care Foundation, a non-profit organization dedicated to the academic development of EMS.

Manuscripts submitted to the *Journal* should concern the prehospital or inhospital care of emergencies or related technical, logistical, educational or administrative issues. Acceptance for publication will be based on significance, validity and originality. These assessments will be made by the editorial staff and advisory board. After acceptance for publication, selected manuscripts will be subjected to additional referee review by persons who may or may not be members of the editorial advisory board. These referees will be asked to submit commentary that will appear immediately following the article. The author(s) will be given an opportunity to respond in print to referee comments and provide closing remarks. The article, referee commentary and author(s) response will all appear together as a single paper.

A major goal of the *Journal* is to stimulate academic activity in EMS. Persons who have not previously submitted work for publication in a medical journal are strongly encouraged to do so. The *Journal* staff will attempt to provide any assistance it can to new authors (see "Assistance in Preparation of Manuscripts").

COVER LETTER

All manuscripts should be accompanied by a cover letter. The cover letter should specify if this or a similar manuscript has been published, accepted or is under consideration by another publication. The cover letter should make mention of any facilities that are available to the authors for transmitting the article to the *Journal* via modem or on a personal computer diskette (5 1/4 or 3 1/2). Specify word processor software and type of computer system. Availability of an electronic version of the article may expedite the review and publication process. The cover letter should also address issues of institutional review board involvement, human or animal, if the article presents original research. Authors of articles accepted for publication will be asked to transfer copyright to the Acute Care Foundation, Inc.

MANUSCRIPT FORMAT

The manuscript should be submitted in triplicate (one original and two copies). Use plain white paper, 8.5 by 11 inches, with 1 inch margins on all sides. Lines should be double spaced, excluding the bibliography. The bibliography should be in the form used by Index Medicus. The names of journals not listed in the Index Medicus should not be abbreviated Use the following as examples:

Journal citation - Harrison EE, Amey BD: Use of Calcium in Prehospital Cardiac Arrest. Ann Emerg Med 1984;13:820

Chapter citation - Yendt ER: Disorders of Calcium, Phosphorous and Magnesium Metabolism, in Maxwell MH, Kleeman CR(eds): Clinical Disorders of Fluid andElectrolyte Metabolism, ed 2. New York, McGraw-Hill, pp 401-504

ILLUSTRATIONS

All photographs or other artwork should be black and white, with professional photographic quality or drawing only. Freehand and typed lettering is not acceptable. Artwork of published articles cannot be returned. Authors should retain their own set of copies of all materials submitted. The *Journal* requires three copies of all artwork. Each item should have a label affixed to the back which states the names of authors, top of figure and the number by which it is referred to in the article. Do not write directly on the artwork or photograph. If the artwork was obtained from another source, fully acknowledge the source and include permission for use from the copyright holder, excluding materials in the public domain. Legends or captions for artwork should be on a separate sheet of paper with numbers corresponding to the appropriate illustration. Identify all arrows and labels in the legend in addition to any explanations that may appear in the article itself.

ASSISTANCE IN PREPARATION OF MANUSCRIPTS

The *Journal* strongly encourages submission of articles and correspondence from persons who have not previously submitted materials for publication. The *Journal* staff will provide whatever assistance it can to new authors in planning and preparation of manuscripts and artwork. Novice authors are strongly encouraged to make preliminary written contact with the editorial staff. The staff may be able to assist authors in development of outlines, finding sources of information, sources of grant support, sources of artwork, collaborating authors and preliminary critiques. The *Journal* wants to be as accessible as possible to the emergency medical community yet maintain high standards of academic credibility and excellence. Please do not hesitate to call upon us. The Journal is a collaborative endeavor drawing upon prehospital, inhospital, academic and administrative resources. The administrators of EMS agencies and hospitals are strongly encouraged to make secretarial support available to personnel wishing to prepare manuscripts for publication in return for the academic exposure to their agency and the educational value to the authors.

National Paramedic Certification Program

presented by the

Acute Care Foundation

in cooperation with the

National Association of Emergency Medical Technicians

The Acute Care Foundation is proud to announce the first component of its professional certification program - Paramedic Certification from the National Registry of Emergency Medical Technicians. Attainment of certification as a Nationally Registered Paramedic is an important step in development of the professional credentials that are becomming required in a growing number of states and major EMS systems. National certification as a paramedic offers a much larger scope of nationwide employment opportunites and demonstrates an individual committment to professionalism.

The faculty and staff for each examination session are chosen from EMS agencies and institutions throughout the area. All our faculty must meet criteria established by the National Registry and additional qualifications required by the Acute Care Foundation. Extraordinary care is taken to assure all candidates of fair, uniform and unbiased evaluations from the highest caliber of faculty and staff. An officially designated representative from the National Registry with an examination coordinator and aide from the Acute Care Foundation will be on-site to enforce these standards and assist candidates throughout the examination process.

Each one day examination session consists of a written and practical evaluation. The comprehensive written exam covers the entire USDOT paramedic cirriculum. The practical exam will be conducted in five stations:

- Trauma Assessment / Management
- Static Cardiology / Medical Emergencies
- Cardiac Arrest Skills
- Cardiopulmonary Resuscitation
- Fracture Immobilization Skills
- EOA / IV Therapy

Registration for each session is limited to the first 30 candidates who satisfy the following registration requirements, verified with appropriate documentation:

- Current State paramedic certification
- Completion of a State approved USDOT paramedic course within two years of the examination date. If a candidate completed their paramedic course more than two years prior to the examination date, the candidate must have completed 48 hours of ALS level continuing education in the two years preceding the examination date. The continuing education must be verified by the candidates' EMS agency or medical director.

Candidates should submit a Registry application form and complete documentation to support their eligibility to the Acute Care Foundation. Upon receipt of a sufficient number of applications, a specific date for the next examination will be set with approximately six weeks notification to all applicants on file. Applicants wishing to attend that examination must then submit a check or money order for $90.00 to cover all tuitions and fees, including refreshments and lunch, at least four weeks prior to the examination date.

For additional information and applications, contact the Acute Care Foundation, P.O. Box 280173, Tampa, Florida, 33682-0173. (813)932-3659. The Acute Care Foundation is a non-profit organization chartered to provide academic services to the prehospital, emergency department and critical care community.

ACUTE CARE FOUNDATION

Professional Membership Program

The Acute Care Foundation is a non-profit charitable organization that has been chartered to provide academic resources to the prehospital, emergency department and critical care community.

Professional membership in the Acute Care Foundation provides an opportunity to participate in education and research programs on an interagency and interhospital basis. These programs are developed and conducted by Acute Care Foundation members operating through its committees in Community Education, Emergency Medical Television®, Professional Publications, Seminars and Symposia, Professional Certifications, and Post-Graduate Paramedic Education. These programs will be a prototype for implementation of similar efforts.

Professional membership entitles you to tuition discounts for Foundation sponsored education functions, a personal subscription to the Tampa Bay EMS Journal, and a credential on your CV or resume' that demonstrates your committment to the improvement of our emergency and critical care systems.

Professional membership is limited to licensed health care professionals. Dues are only $30 annually. Consider the benefits that the Foundation's interagency and interhospital collaboration in regional training and research programs can offer. This is an opportunity for you to interact with your collegues on an interagency basis, enhance your professional experiences and credentials, and make a meaningful contribution to your EMS system. Please complete the Professional Membership application and help make a difference. Consider it an investment in our EMS community.

Application for Professional Membership in the ACUTE CARE FOUNDATION

Name ____________________

Business Mailing Address ____________________

City, State, Zip ____________________

Business Telephone (   ) ____________ (   ) ____________

Home Mailing Address ____________________

City, State, Zip ____________________

Home Telephone (optional) (   ) ____________________

Primary Medical Employer, Affiliated Agency or Institution ____________________

____

Professional Licensures (attach proof of licensure)

	State of Licensure	License #
EMT	________	________
Paramedic	________	________
Physician	________	________
Specialty	________	
RN	________	________
Other	________	________

Committee Appointment Interests

Select committees for which you would like to be considered for appointment to by the Board of Trustees. Please include a copy of your CV or resume' with this application. A cover letter is desireable but optional. Mark desired appointments with a number in order pf preference, such that a "1" is placed next to the committee of primary interest.

Community Education Committee
Emergency Medical Television® Committee
Professional Publications Committee
Post-Graduate Paramedic Education Committee
Professional Certifications Committee
Seminar and Symposia Committe

Please forward this application with appropriate documentation and a check or money order for $30 to: Acute Care Foundation, P.O. Box 280173, Tampa, FL 33682-0173 Please call (813)932-3659 for any additional information. Thank you for your participation and support.

ACUTE CARE FOUNDATION

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Corporate Membership Program

The Acute Care Foundation is a non-profit, charitable and tax exempt organization that has been chartered to provide a means to promote academic activities in emergency medical service (EMS) systems. These programs are also designed to promote collaboration between the prehospital, emergency department and critical care sectors in joint research and academic programs to the benefit the EMS system as a whole. There are extraordinary human and material resources available between the various agencies and institutions that compose EMS systems within a region. The Acute Care Foundation was formed by EMS professionals to use these regional resources collectively for professional education, research and planning towards superior system design and exemplary quality in patient care.

The goals of the Acute Care Foundation are complementary to those of public and private organizations and businesses involved in providing or supporting emergency health care - the Acute Care Foundation needs their participation and financial support.

Corporate membership in the Foundation is available to corporations and other types of businesses having an interest in promoting quality within the emergency medical services industry. Corporate membership offers businesses and excellent opportunity to put something back into their communities and improve relationships with the clinicians, agencies, institutions and patients that Foundation programs ultimately benefit. Corporate membership also facilitates direct participation through appointment of a designated representative in Foundation committees.

The generosity and commitment to excellence of corporate members in the Foundation will be prominently and formally recognized in various ways, to include a full page acknowledgement in each issue of the new Tampa Bay EMS Journal that presents a dignified listing of all current corporate and organizational members.

Corporate membership is offered in levels corresponding to the size of an annual contribution pledge. The prestigious Primary Corporate Membership is available for an annual contribution of $5000 or more. Major Corporate Membership is available for annual contribution pledges of $2500 or more and Participating Corporate Membership is extended for annual pledges of $1000 or more.

Overall, the advantages of corporate membership in the Acute Care Foundation should consider the a firm's professional relations program, marketing efforts and the tax advantages in charitable contributions. The Acute Care Foundation is an organization worthy of your firm's participation and financial support. It is an effective and influential group that your firm will be proud to be associated with. Consider your contribution to be an investment in our EMS community. Please contact the Foundation for any further information or to arrange for an appointment to your convenience.

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Advertising Rates and Policies

The Tampa Bay EMS Journal is published quarterly by the Acute Care Foundation, Inc. - a non-profit, charitable and tax exempt organization. The Tampa Bay EMS Journal is targeted for a professional audience in prehospital, emergency department and critical care medicine. Its editorial policies are designed to address the medical, logistical and administrative issues which influence emergency pateint care.

Each issue is distributed primarily through subscription free circulation agreements in the five county Tampa Bay region covering all of its prehospital EMS substations, training officers, administrators, medical directors, EMS councils and EMS training centers, all emergency rooms, critical care units and inservice training departments, along with their respective head nurses, and medical directors. Additional subscription free distribution will include emergency and critical care professional organizations, State of Florida EMS Office, selected public officials and major media in this primary service area of Hillsborough, Manatee, Pasco, Pinellas and Polk counties.

Each issue will have a distribution of 1000 copies. Pass along readership is projected at 5 to 1, due to the nature of the distribution agreements. Our aim is to provide 100% EMS market penetration and prioritize delivery of individual copies to parties responsible for policy and purchasing decisions. This provides an ideal marketing vehicle for regional sales representatives and concentrated test marketing to a defined regional audience.

The majority of funding for the Tampa Bay EMS Journal is from advertising. Additional funding, via the Acute Care Foundation, is sought through grants, contributions, and professional, organizational and corporate memberships. Paid subscriptions to individuals are available with professional memebership in the Acute Care Foundation.

Advertising materials must be submitted camera ready, with color separations if applicable. Standard rates assume black and white materials. Color charges are added to the standard black and white charges. Discounts of 2.5% are available for a repeated ad in a consecutive issue with advance payment. Additional cumulative 2.5% discounts are offered to a maximum of 10% with repeated ads in four consectutive issues with advance payment. All advertising materials should be submitted eight weeks prior to each quarterly publication date on the 1st of January, April, July and October. The Tampa Bay EMS Journal reserves the right to refuse any advertising materials.

The following advertising rates are effective May 15, 1987:

Inside Front or Inside Rear Cover	$1000
Full page	$300
Half page	$150
Quarter page	$75
Eighth page	$40

For any additional information or an appointment, please contact Patrick Shepler, Advertising Director, Tampa Bay EMS Journal, P.O. Box 280173, Tampa, Florida 33682-0173, (813)932-3659.

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