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Emergency Intraosseous Fluid and Drug Administration

MICHAEL R. GUNDERSON, REMT-P,¹ and MICHAEL BROWN, REMT-P,²

Establishing a route for fluid and drug administration is an important task in managing patients requiring advanced life support. The inability to secure an IV during an emergency can seriously delay what might be life saving therapy. This problem can be particularly acute in the field, where clinical privileges for venous access usually do not include central routes, due to the higher potential for serious complications and limited EMS agency access to appropriate clinical training in central venous procedures.

Other fluid and medication routes are available, but have significant limitations. The endotracheal route requires that the patient be intubated and will only accommodate a limited number of commonly accepted drugs, to include naloxone, atropine, diazepam, epinephrine and lidocaine. In general, endotracheal drugs should be lipid soluble and relatively non-irritating (1). The intracardiac route will accept most any drug, but the patient must be in arrest. The intracardiac injection has a high potential for complications and must interrupt chest compressions (2). Sublingual, subcutaneous, transdermal, intramuscular and rectal administration routes are only viable when perfusion to those tissues are sufficient to promptly absorb medications and carry them to the central vascular circuit.

There is an evolving rediscovery in the emergency and critical care community of a previously developed medication route via the rich venous network of the bone marrow, commonly called the intraosseous route.

HISTORICAL DEVELOPMENT

The idea of intraosseous infusion dates back to 1922 in the works of Drinker (3) and Doan (4), describing the circulation of blood through marrow. Drinker suggested the marrow as a potential route for blood transfusion. Diagnostic puncture of the sternum to obtain marrow samples was described by Arinkin (5) and cited by Josefson as the original basis for his use of sternal injection of liver extracts to treat anemia (6). Josefson speculated about the bone marrow as a route for administration of other drugs. He also discovered a serious complication potential with inadvertent sternal perforation and undetected pleural infusion. He suggested use of the tibia as another marrow infusion site.

Dr. Leandro Tocantins of Philadelphia played a major role in bringing this new technique to the attention of the medical community. In 1936, the seeds for Tocantins' later work were planted while he was performing bone marrow transplant experiments in which he noticed significant loss of fluids injected in rabbit femurs. Local tissue examination did not reveal any signs of infiltration. Absorption by the vascular system was implicated (7).

Research by Benda and colleagues, published in 1937 (8,9) and 1940 (10), demonstrated rapid migration of bacteria and other substances into the venous circulation and lungs following sternal injection. In 1940, Henning (11) experimented with sternal injections of radiopaque fluids, dextrose and blood in persons with circulatory collapse and concluded that it would be a good route for transfusions to battlefield casualties.

The team of Tocantins and O'Neil collaborated in several widely cited studies of intraosseous infusion (12-21). Their work included an experiment where mercury was infused into the humeri and tibias of an infant, showing systemic distribution by full body fluoroscopy (7). Congo red dye infused into the tibia was found in the heart 10 seconds after injection (12). Seizures secondary to hypoglycemia were successfully treated with a tibial infusion of glucose (12). Tocantins refined the procedure with development of special equipment (13-14) and published on use of the intraosseous technique in pediatrics (15-16).

Following a surge of publications in the 1940's and 1950's (22-56), intraosseous infusion fell from common use with the introduction of improved intravenous methods, including the indwelling plastic IV catheter.

Contemporary interest in the intraosseous route reappeared in 1977 with a publication by Valdez recounting his "discovery" and 15 cases of intraosseous fluid and medication delivery (57). In 1979, Shoor published on an animal study where flow rates with pressure infusion and the general pharmacokinetics of intraosseous therapy were described (58). In the 1980's, the volume of publication on the topic of intraosseous infusion is growing (59-81).

The clinical anatomy of the tibia will be described, as it is the most common site for intraosseous infusion.

CLINICAL ANATOMY

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The tibia is a superficial long bone which contains a significant marrow cavity, even at birth. The marrow of the tibia, as well as other bones, is contained in its intramedullary space. Its spongy marrow tissue contains a dense network of blood filled sinuses. These venous sinuses interconnect to drain out of the bone through emissary veins. These in turn drain into the popliteal veins (Figure 1).

The spongy semi-rigid marrow tissue surrounding the sinuses do not collapse with hypotension, as do peripheral veins. Thus, they are consistently accessible in the critically ill or injured patient.

The long bones, such as the tibia, femur, and humerus, have a delicate layer of tissue at the distal and proximal ends called the epiphyseal plates (Figure 1). These structures are responsible for longitudinal bone growth. Therefore, introduction of any needle into these long bones must be particularly cautious to avoid the epiphyseal plates and thereby avoid disruption of normal bone growth. On the tibia, the proximal epiphyseal plate is located just proximal to the tibial tuberosity. Proximal introduction of an intraosseous needle is therefore angled slightly caudad, to minimize risk of epiphyseal contact. On the distal tibia around the malleolus, the needle is directed slightly cephalad to avoid the distal epiphyseal plate. These same principals hold true for the femur and humerus. The mid-shaft of long bones are usually not utilized because the neck of the bone near either end is wider and has a larger marrow cavity, making success more likely. Further, the mid-shaft has more yellow marrow, in contrast to the red marrow at the ends. The yellow marrow has a higher theoretical potential for fat embolism, although Tocantins reports that the work of Harris and Bolle indicates this risk to be minimal (7).

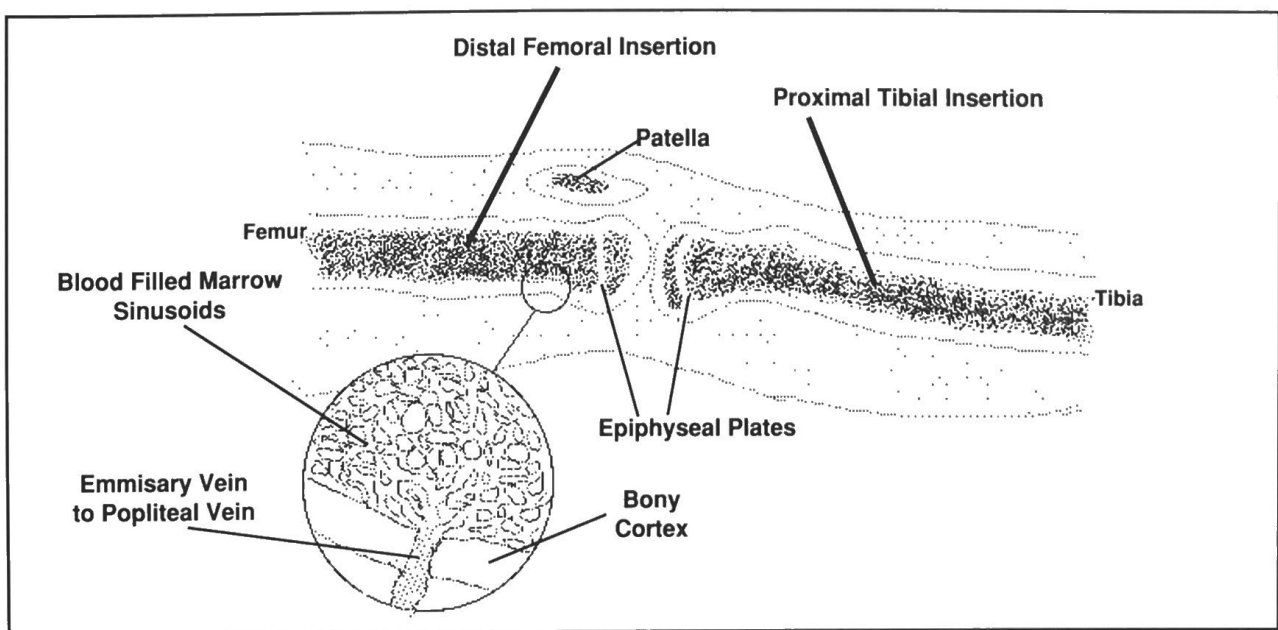
INDICATIONS

The intraosseous route should be considered as a second line route for drug therapy in an emergency situation. Although the procedure might also be used in less urgent situations, the contemporary short and long term complication rates and marrow reactions to many modern drugs are not well documented. Rapid emergency fluid replacement in the adult may not be practical by the intraosseous route. The maximum flow rates by gravity and by pressure infusion (300 mm Hg) are approximately 10 milliliters per minute and 40 milliliters per minute, respectively (58). These rates are probably inadequate for serious hemorrhage in the adult, but may still be useful in children.

The literature sites osteopetrosis (a condition of excessive bone calcification which causes spontaneous fractures - also called Albers-Schonberg disease) and osteogenesis imperfecta (defective bone matrix with propensity for fracture) as general contraindications (64,66). Fresh or recently fractured bones must not be used to avoid subcutaneous extravasation (66).

EQUIPMENT

The procedure will require skin antiseptic, tape, IV fluid and administration set, and a suitable needle. The needle used for intraosseous access should be stiff, large bore (at least 18 gauge) and include a stylette. Needles without stylettes may be used if other more suitable needles are not available. To clear bone particles that might occlude the lumen of a plain needle, a smaller and longer needle may be inserted into it through the hub.



M. Gunderson

Figure 1 - Clinical Anatomy for Tibial and Femoral Approaches to Intraosseous Infusion

Bone marrow aspiration needles are commonly used for the intraosseous procedure, including the Osgood and Rosenthal varieties. The modified Illinois aspiration needle (Figure 2) is currently in use by the Fairfax, Maryland EMS agency (66,67). That needle seems to be very well suited for the procedure. It has a collar which can be adjusted after insertion to prevent the needle from going too deep and thereby reducing the risk of perforating the bone. It is also a disposable item. The Rosenthal and Osgood needles are non-disposable with a cost of approximately thirty-five dollars each.

SITE SELECTION

There are many potential sites. In general, any superficially accessible bone with significant marrow cavities may be utilized. Particular caution should be used in children and adolescents to avoid disruption of the epiphyseal area of long bones, so normal bone growth will not be disturbed (65). The literature most frequently mentions the sternum, tibia, femur and humerus as suitable targets.

The sternum provides an adequate marrow cavity only after three years of age. There is a potential for causing dangerous accumulation of fluids in the chest if there is leakage from either the primary site, previous sternal sites where access was unsuccessful, or from inadvertent sternal perforation (56).

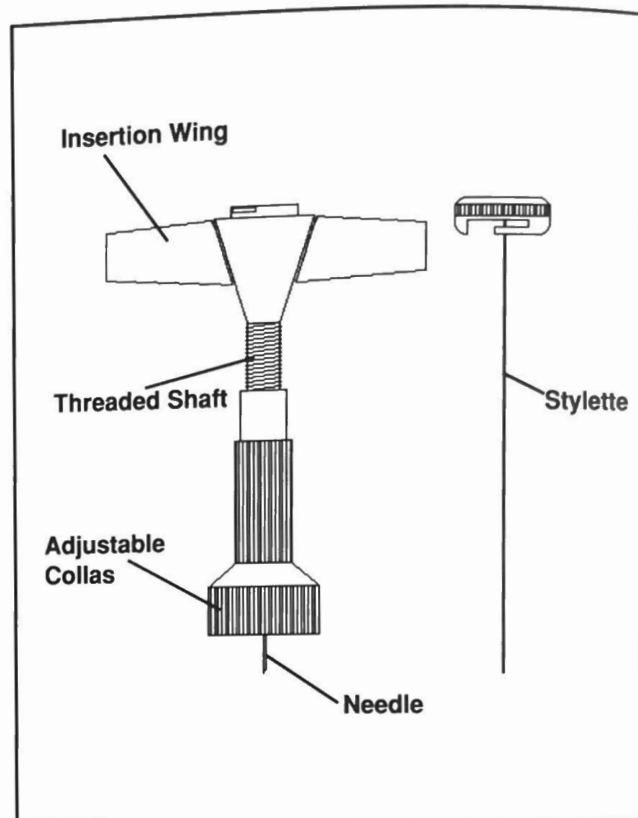
The proximal tibia appears to have a consensus of favor in the literature we have reviewed. It is suitable for patients of most any age and has a minimum potential for complication, so long as the epiphyseal area is avoided in younger patients. The point of needle insertion should avoid areas with cellulitis and infections. The complications of osteomyelitis and iatrogenic infection are much higher when the intraosseous line is utilized for more than 24 hours (64).

Should the procedure be used in a trauma patient requiring the use of the MAST garment, a femoral or proximal tibial site would interfere with MAST placement. In such cases, the malleolus or humerus may be preferable. Infusion from the malleolus to flow up through the tibia within the MAST garment should theoretically not be a factor limiting infusion flow rates. However, the effect of the MAST on flow out of the tibia via emissary veins and into the popliteal veins has not been studied.

PROCEDURE

The principles of the technique are similar for all sites, but this discussion will use the proximal tibial site to describe specifics.

The best detailed description we found for tibial access was offered by Friery and Weiner (66). Using a modified Illinois aspiration needle, the needle is inserted at a point even with or slightly inferior to the tibial tuberosity. The point of



M. Gunderson

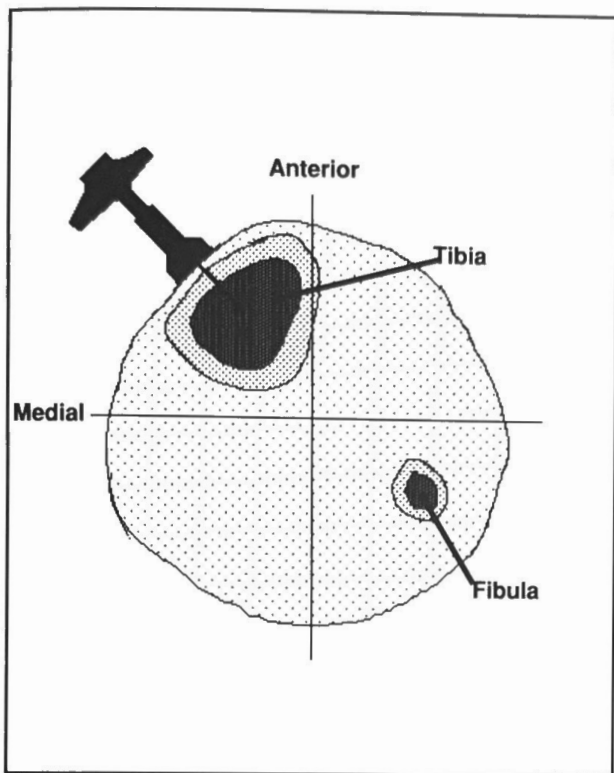
Figure 2 - Modified Illinois Aspiration Needle

insertion should be slightly medial on the leg to give a better exposure of the marrow cavity (Figure 3). External rotation of the leg will provide proper positioning.

If the patient is awake, local anesthesia may be provided by a wheal of lidocaine infiltrated along the intended insertion pathway, to include the periosteum where bone penetration will be made.

The needle is inserted in two steps. The first is through the soft tissues to bring the point of the needle against the bone. The second step uses a firm twisting motion to penetrate through the bony cortex and into the marrow cavity, as indicated by a break in resistance as the needle tip "pops" into place. The stylette is removed and replaced with a syringe containing 5 cc of intravenous fluid. Blood and marrow may be freely aspirated if the tip of the needle is properly inserted into the marrow. However, aspiration is reported to be very uncomfortable in the awake patient, so limit aspiration to 1 ml in confirming placement. The 5 ml of IV fluid can be injected to help clear the lumen and tip of bone fragments. The IV fluid administration set should be attached and opened as soon as possible to prevent clotting. If this is delayed, reinsert the stylette.

The modified Illinois needle features a circular collar that may be screwed down around the outside of the needle until firmly against the skin to prevent inadvertently deeper penetration. Tape may then secure the entire assembly in place.



M. Gunderson

Figure 3 - Angle of Insertion for Proximal Tibial Approach

DISCUSSION

There are several issues that appear to be unresolved with this procedure.

Use of intraosseous infusion for adult trauma victims may offer a faster and more consistently accessible route for fluid replacement, for both crystalloids and blood. However, the pressure infusion studies of Shoor in an animal model found flow rates of only 41 ml per minute (2460 ml per hour) with a 300 mm Hg infusion pressure level (59). This would probably be inadequate for an adult with a serious hemorrhage. Studies have not been identified which have examined the potentials of multiple simultaneous pressurized intraosseous infusions, in the same or multiple bones.

The contemporary literature does not provide a study of long term complications of the procedure, particularly in children. Further, the short and long term effects on the bone and marrow of many drugs now used in emergency and critical care have not been identified.

In the prehospital arena, we are not able to locate publications which document complication rates, attempts and time required for placement, or responses to medications given via the intraosseous route. Complication rates, attempts, and times may vary with the site and equipment utilized.

SUMMARY

The intraosseous route for fluid and medication delivery is a practical alternative in an emergency setting when other routes of peripheral IV access are not available. It may be particularly useful in the field, where central venous procedures are not widely utilized. While pediatric cases are most often cited as candidates for the procedure due to their inherent problems in venous access, the method appears to be equally practical and efficacious in adults. Further study is needed to clarify the optimal equipment and sites for the procedure. Contemporary statistics on overall complication rates and local tissue reactions to current emergency and critical care medications are lacking.

REFEREE COMMENTARY

Harold F. Sherman, M.D. (*Trauma/Critical Care Fellow, Tampa General Hospital; Clinical Associate in Surgery, University of South Florida; Tampa, FL*) - The technique discussed here is interesting and probably some applications in unique settings. Yet it seems largely to be a method currently looking for its niche.

In an era of increasing emphasis on a 'scoop and run' field approach to trauma, especially in an urban setting, another time-consuming technique to infuse inadequate amounts of fluid seems superfluous. Regarding the use of this technique in the pre-hospital treatment of acute medical illnesses, the authors state quite clearly that studies of drug absorption by and drug effects on marrow are yet to be done. Within these constraints, it is difficult to accept the statement that this technique is "...practical and efficacious.."

While there may be unique and specific situations within the prehospital setting, including the emergency department, where this procedure could or should be used on a controlled trial basis, I can imagine little immediate role in the pre-hospital setting for intraosseous infusion. I look forward to more clinical trials exploring the areas of drug absorption, fluid flow rates, complication rates, and long term effects.

Richard Weibley, M.D. (*Assistant Professor of Pediatric Critical Care, University of South Florida; Tampa, FL*) - Gunderson and Brown have written a comprehensive and valuable review article on intraosseous fluid and drug administration. They clearly outline its conceptualization, disappearance with improved intravascular techniques of the 1950's and subsequent reemergence two decades later. This is primarily because of the needed emergence of prehospital care and the ability to treat patient who previously died.

Gunderson and Brown also nicely describe the anatomical sites, necessary equipment and acceptable techniques. Hopefully, after reading the article, those who have not utilized this modality will reconsider when the indication arises.

Finally, Gunderson and Brown discuss the need for research documenting both the efficacy and long-term complications.

As with so many techniques in medicine, the intraosseous route appears to be a rediscovery of the wheel - what goes around comes around.

Thomas J. Abrunzo, M.S., M.D., F.A.A.P., F.A.C.E.P. (*Chief, Section of Pediatric Emergency Medicine, All Children's Hospital / Bayfront Medical Center; St. Petersburg, FL*) - There's "good news" and "bad news" regarding emergency intraosseous infusion, particularly for the pediatric patient. The paper by Gunderson and Brown outlines the situation well.

The "good news" is that this technique is time-tested, requires simple, inexpensive equipment, is easy to learn and is potentially life-saving.

The "bad news" is the unknown; there is not data to document safety and efficacy versus other venous access techniques in specific emergency situations. As is the case so often in pediatrics, there is not a simple approach or response to the variety of pediatric arrest or pre-arrest conditions. One must be quick to individualize therapy. Similarly, one needs to evaluate intraosseous infusion in such common but pathophysiologically disparate situations as: multiple trauma with skeletal fractures, respiratory failure, isolated head injuries, status epilepticus, septicemia/bacteremia.

A multitude of questions emerge: Are fat and/or marrow embolism a significant problem with high pressure, high volume infusion versus low pressure, low volume medication infusion? Does septicemia significantly increase the occurrence of osteomyelitis? Should the risk of osteomyelitis constitute a relative contraindication? With lower extremity and pelvic fracture, are the humeral and sternal sites as easily used? Are the complications significantly increased? Are volume and medication uptake rates the same in full cardiopulmonary arrest as compared to some of the above pre-arrest conditions?

Should emergency intraosseous infusion be advocated for the prehospital setting in the absence of data on comparative efficacy and complications? Present data suggest that the cost-benefit comparison, as best as we can tell, is skewed heavily on the benefit side. I therefore strongly believe that the technique should be studied and used by prehospital caregivers, with the following, specific admonitions:

- The provider should develop and rehearse a time-directed protocol based on patient severity.
- A plastic-jacketed steel needle with adjustable shaft should be used to minimize difficulty and complications of insertion.
- The infusion site should be switched to a more conventional site as soon as status allows.
- The provider should facilitate collection of data regarding protocol implementation, acute results and long term follow-up.

Mr. Gunderson's reply - All of the reviewers have appropriately reemphasized that there are still many unknowns concerning the intraosseous route, particularly on long term effects to the marrow. I believe Dr. Abrunzo puts these concerns in proper perspective in terms of a cost - benefit comparison. The possible risks of epiphyseal, marrow and fat emboli complications must be balanced against the possible benefits that early fluid and drug administration can provide in pre-arrest and arrest conditions. The evidence indicates that these possible risks are indeed genuine. However, these studies also support use of the intraosseous technique, despite these risks, due to a relatively low incidence of such complications.

From a conservative perspective, the intraosseous route might be implemented in the emergency department and prehospital environments under well defined research protocols. Those protocols should be designed to minimize the potential for complications, as Dr. Abrunzo suggests, with an optimal needle and a switch to a

conventional IV as soon as possible. Rigorous data collection should be mandatory. The fluids and drugs used in these early applications might be limited to those of a less innocuous nature, such as crystalloids, blood and some specific drugs for which there is experience reported in the literature. It's hard to turn one's back on the considerable experience and favorable cases that have been published. The potentials of this route for the patient in extremis with vascular collapse should encourage us to conduct these studies and see if those potentials can be realized. I thank the reviewers for their critique. Their comments will be helpful to those who will pursue research in this area.

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The Relationship Between Oxygen Administration and Airway Resistance: Clinical Considerations

RICHARD KONRAD, BA, REMT-P

The goal of oxygen therapy is the maintenance of adequate arterial oxygen tension by insuring the necessary concentration of oxygen in the inspired gas. When sufficient oxygen is present, metabolism of glucose provides adequate energy for all body processes—including active transport, protein synthesis, and muscle contraction. The level of energy production when sufficient oxygen is not available is markedly different. In anaerobic metabolism energy production drops nearly twenty-fold. This remarkable and disastrous decrease in energy available for all body processes is a hallmark of any situation in which hypoxemia or poor tissue perfusion occurs (1).

The relationship of oxygen availability and energy production for muscle contraction has a direct bearing on the provision of supplemental oxygen therapy. Many patients who receive supplemental oxygen are already hypoxic or in states of widespread inadequate perfusion. Any increased work for these patients represents a demand for energy which they are in poor condition to meet. In the normal course of treatment, the activities of these patients are restricted and demands on their limited energy reserves are limited (they are not allowed to move about actively or to become hypothermic and shiver). A review of anatomy and physiology as they affect oxygen therapy reveals that the way in which oxygen is provided may have a deleterious effect on the level of work required of the patient.

While the available energy for work is dependent on the level of oxygen available to the tissues, the work required for breathing depends on three factors: the compliance of the lungs (their propensity to collapse or recoil from the chest wall) along with the resistance of the chest wall to expansion; the resistance to expansion of the lung tissue itself; and, the resistance to the flow of gas through the airways. Any situation which decreases the compliance of the lungs (making them resist expansion), increases the viscosity of the lung tissue, or increases the resistance to airflow through the airways will increase the work of breathing. Some conditions requiring provision of supplemental oxygen increase the patient's work of breathing by causing hypoventilation which stiffens the lungs or interstitial pulmonary edema which increases tissue viscosity. Some disease states like asthma or chronic obstructive lung disease increase the work

of overcoming airway resistance. The level of work necessary to overcome airway resistance is also affected by supplemental oxygen therapy.

The resistance to the flow of gas through a tube is determined largely by the cross-sectional area of the lumen of the tube according to Poiseuille's law which is demonstrated in the following equation:

$$R(\text{resistance}) = 8 \times \text{viscosity} \times \text{length} / \pi \times r(\text{radius})^4$$

Thus, resistance varies directly with viscosity of the gas and length of the tube, but inversely by an exponent of four with changes in the radius of the cross-sectional area of the lumen of the tube. Small decreases in this radius produce large changes in the resistance to the flow of gas and hence the work required to move gas through the tube. The cross-sectional area of the entire length of the tube lumen need not be decreased in area in order for resistance to be increased; any cross-sectional plane which has a narrowed area will produce this effect. Research by Mertz and his associates has demonstrated that airway resistance is altered in exercise induced hypercapnia by increased cross-sectional area of the nasal passage lumen in such a way that resistance decreases with an attendant increase in gas flow rates. This decreased resistance, which also decreases the work necessary to move a volume of gas, is consistent with increasing ventilation and lowering CO₂ levels (2). All oxygen administration devices applied to a patient's face alter the cross-sectional area of the patient's airway at its origin. Only two sources of inspired gas exist for the patient wearing an oxygen administration appliance: the gas within the mask (or supplied through the cannula prongs) and gas outside the mask (or available to the cannula wearer in the atmosphere). The gas reservoir within the mask is replenished at a constant rate by the flow meter. While the inspiratory work to overcome compliance and tissue resistance produce a nearly linear development of the relationship of lung volume to intrapleural pressure, the work necessary to overcome airway resistance produces a markedly uneven development of this relationship. In other words, the work of overcoming airway resistance is much higher in the middle of inspiration and less at the beginning and end. Flow meters cannot alter rate of gas flow to match these physiological changes. The only way for a mask wearer to inspire the gas outside the mask is to draw it through openings in the mask which usually consist of small exhalation holes arranged in a tight circle and breaks in the seal of the mask at

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the patient's face. Whether the patient needs to inspire gas through these small openings depends on his or her minute respiratory volume as compared with the oxygen volume supplied by the flow meter.

An average adult male has a tidal volume (V_T) of approximately 500 ml and a respiratory rate between twelve and twenty (the limitations of the useful statistical concept of the average patient are recognized). In an adult possessing normal lungs and no disease or injury with the previously stated V_T of 500 ml and a respiratory rate of fifteen breaths per minute, a volume of 7.5 liters (500 ml x 15) of gas is breathed each minute. This figure represents the minute respiratory volume (V_M). The V_M is adjusted moment to moment in order that metabolic demands for oxygen and removal of waste products be met. Any time a patient is not able to achieve the necessary V_M , the work of respiration is increased in order to achieve the necessary volume.

Guyton points out that, if sufficient energy exists, the V_T may approach vital capacity (V_C), about 4600 ml in adult males (3). In rapid breathing, however, the V_T rarely exceeds half the vital capacity. Imagine that in response to early pulmonary edema (interstitial pulmonary edema), which increases the work of overcoming lung tissue resistance, a patient's breathing rate increases to 30 breaths per minute, accompanied by a modest increase in V_T to 750 ml. The subject's minute volume is now 22.5 liters per minute. This elevation in V_T is occasioned by increased expenditure of energy in order to perform the increased work.

If a simple face mask is placed on the above considered patient and the flow meter is set to provide 10 liters of medically pure oxygen each minute, slightly less than half the patient's minute volume is met by the reservoir of gas within the mask. This patient must draw the remainder of his minute volume, 12.5 liters, through the small holes in the mask. The cross-sectional area of the patient's airway has suddenly been markedly reduced at its origin, with an attendant rise in resistance of gas flow through the airway and increase in the work of breathing. The non-linear development of work to overcome airway resistance discussed above means that even in circumstances where the patient's minute volume is met by the reservoir of gas within the mask, at times during the inspiratory cycle the work of inspiration may increase. The discomfort of which patients receiving oxygen therapy by mask complain has often been laid at the foot of the mind, a psychological aversion or claustrophobic reaction. A physiological basis for the patient's wish to remove an obstructing object from his or her airway is just as possible in some circumstances.

These facts are not set out in order to condemn the simple face mask or to suggest that it is inappropriate in all circumstances. Each oxygen administration device must be used with an appreciation for the ways in which it affects the mechanics of breathing. The venturi mask, which mixes room air with oxygen at specific concentrations, produces a

high total gas flow with fixed fractional concentration of inspired oxygen by percentage (F_iO_2). In the Hudson Multi-Vent mask, oxygen concentrations range from low (23-30%) to moderate (35-50%). Note that the simple face mask provides only 60% oxygen concentrations under ideal conditions. The construction of the venturi mask differs from that of the simple face mask in one other important regard. The exhalation port of the venturi mask is one large hole, rather than a collection of very small ones. A patient with a very high minute volume is served well by such a mask because of the high total gas flow into the mask and the relatively low resistance to the flow of gas through the large exhalation port. Considering that the F_iO_2 possible with each mask is relatively close, the least obstructive of the devices might be a wise choice in patients with high minute volume. The consideration of the relationship between the patient's minute volume and the oxygen administration device leads to the question of whether a method exists to determine the patient's minute volume at the time of treatment. Aside from spirometry, no easy method of accurately determining the minute volume exists. The minute volume may, however, be indirectly estimated with a flow meter and an oxygen mask with a reservoir bag. Either a partial rebreathing mask or a total non-rebreathing mask provide high F_iO_2 . If the reservoir bag is filled prior to placing the mask on the patient, the effect of the patient's breathing on the volume of oxygen in the bag may be observed. If, at the peak of the patient's inhalation, the bag remains at least one-third filled, the minute volume being delivered through the inlet tubing of the mask should be sufficient to meet the patient's minute volume demand. If, on the other hand, the patient's inhalation collapses the reservoir bag completely, the flow meter can be adjusted to supply increased minute volume. In any event, although claustrophobia as an etiology for the discomfort patients feel with oxygen masks is entirely plausible, an understanding of the mechanics and work of breathing as affected by oxygen masks reveals that a physiologic etiology is equally plausible. Oxygen administration devices vary widely in the degree to which they obstruct the airway and increase the work of breathing. Devices such as the face tent offer no increase in airway resistance, but are incapable of supplying high F_iO_2 . The nasal cannula is another low-to-moderate concentration device which has little effect on airway resistance, since the mouth remains open to the atmosphere. Almost all high concentration devices have the potential to increase airway resistance and the work of breathing. Remember, however, that the bag-valve-mask system with oxygen reservoir can act as a non-rebreathing mask for oxygen inhalation therapy. Because of the larger volume of the reservoir and the relatively large diameter of its tubes, this device has less effect on airway resistance than smaller oxygen masks. Finally, patients are well served when the least obstructive oxygen administration device which provides an adequate level of therapy is chosen.

REFEREE COMMENTARY

Brian Cobb, MD (*Department of Anesthesiology, University of South Florida*) - This review of oxygen administration in the spontaneously breathing patient should assist in the selection of techniques and provide an understanding of their ramifications. It is worthwhile to point out that when a spontaneously breathing patient is intubated, using a tube shortened to the minimum length combined with the largest possible diameter will decrease the work of breathing.

In addition, It might be remembered that an inadequate mask seal, e.g. when using the bag-valve-mask unit on a spontaneously breathing patient, can result in entrainment of room air with subsequent reduction in F_{iO_2}

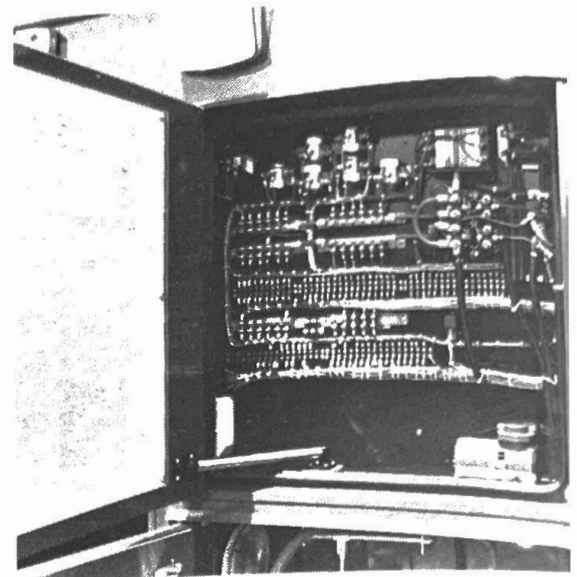
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PROTOCOL ROUNDTABLE

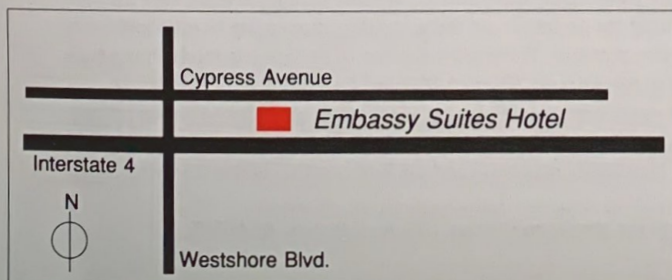
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PROTOCOL ROUNDTABLE

Acute Congestive Heart Failure

The following article contains an edited transcript taken from the Protocol Roundtable Symposium sponsored by the Acute Care Foundation in Tampa, Florida on October 21, 1987.

The Protocol Roundtable is a session in which paramedics, physicians, nurses and other acute care clinicians review a specific emergency disorder. This entails discussion of the pathophysiology, clinical recognition, therapeutic objectives, review of current EMS agency protocols, and a computerized literature search. The session culminates in a "roundtable" group discussion to develop a model protocol.

Recognizing that congestive heart failure may arise from a wide variety of problems, the session approached the issue with the general idea of myocardial infarction as the etiology for the purpose of these proceedings.

A lecture on the pathophysiology and therapeutic objectives was delivered by Michael Brown, REMT-P, from the Hillsborough County EMS system. The clinical recognition of congestive heart failure was presented by Michael Gunderson, REMT-P of the Palm Harbor Fire Department in the Pinellas County EMS system. Patrick Shepler, REMT-P, of the Clearwater fire Department, Pinellas County EMS, delivered a presentation of current protocols from other EMS agencies. The literature review was conducted by the Acute Care Foundation via the CompuServe® information network using the PaperChase® interface to the National Library of Medicine's Medline® medical literature data base. The search strategy and results are shown in Table 1.

The model protocol development effort was moderated by Mr. Gunderson. The dialog below was immediately preceded by a separation of items in the current protocols and then categorized under the appropriate therapeutic objective(s) (Tables 2-5).

- Gunderson: We have identified the specific therapeutic objectives. Let's now take what we have on the screen (composite of Figures 2-5) here and try to translate it into a specific protocol. Of all of these items under oxidation and ventilation (Figure 2), which of them do we want to put in the protocol? Are there any we want to exclude? They all seem to be pretty uniformly accepted. PEEP (positive end-expiratory pressure) is something that we haven't seen in a lot of them and the aminophylline is one that we haven't seen in a lot of protocols. *(Michael Gunderson, REMT-P)*
- Nelson: We're talking about basic treatment right now? *(Joe Nelson, DO, Carrollwood Community Hospital)*
- Gunderson: Let's consider it in a specific format. Let's consider part one to be standing orders, or actions which might be authorized prior to physician contact. Part two orders could be ones where physician contact is required and we could even consider a part three for use when physician contact is unavailable, for whatever reason. Where those lines are drawn I think again might be a variable with the system and this least common denominator factor, if we can come to some sort of consensus about this imaginary average paramedic with his 2.5 kids, etcetera.
- King: Is that our purpose? *(Michael King, REMT, Acute Care Foundation)*
- Gunderson: In determining a typical protocol?
- King: I thought we were shooting for an optimal protocol?
- Gunderson: That's a good point. If we make a supposition that we have top of the line paramedics let the system gear itself to this level.
- King: I think that's what we need to do. I think we need to set goals for people to try to achieve.
- Segal: I agree with what Dr. Nelson said earlier - Let's make the standards high and bring our paramedics in the system up to them.
- Todoroff: I agree, I think it's in the interest of patient care rather than catering to the lowest common denominator of provider. *(Molla Todoroff, REMT-P, Medic One Ambulance Service, Pinellas County EMS)*
- Shepler: I think it sounds good and altruistic. Make sure all your paramedics are screened carefully enough to make sure they are as sophisticated as the protocols, rather than just putting the protocols out there and then attempting to retrospectively bring the paramedics up. This protocol is an aggressive protocol. There are a number of therapeutic modalities which could confuse your average paramedic and jeopardize patient care. *(Patrick Shepler, REMT-P)*
- Gunderson: What we might have is a situation where we'll recommend this protocol as an ideal protocol, suited to a well trained, tightly controlled system. If your individual system isn't all that healthy, this might be the goal you shoot for. You might initiate some components of this until your training, continuing education and medical control catches up with it. That would have to be decided by the individual system.
- Brown: You can vary the border between standing orders and the physician contact. *(Michael Brown, REMT-P)*

- Todoroff: It might be a good idea for us not even to try to define where the borders are. Define an ideal protocol and leave it up to the individual providers to feel where they need the borders.
- Brown: I think we should still suggest the same because when it comes to number three, when physician contact is unavailable, obviously that's a divergence.
- Todoroff: I disagree here. You're setting up an ideal protocol and it would be up to medical control for each individual entity to say you can do this only with medical control, you can do this when you're not able to get medical control. We don't need to worry about it because the protocol's the same.
- Segal: If we're talking about the optimal protocol, the idea is that people would be trained in order to do this. Physician contact really should not be a factor. The control is needed until the paramedics are brought to a level of the top paramedic or the top system that we're aiming at.
- Nelson: Although our goal should not be to eliminate medical control.
- Segal: No, not at all.
- King: We're just trying to optimize medical control.
- Todoroff: Let's optimize the protocol and let everyone worry about their own medical control.
- King: If you have a physician who is what I consider to be a good physician or person that takes a lot of interest in the system, and is not doing it for other reasons, then obviously it's going to follow that your paramedics are going to be fairly sophisticated because he is not going to sign off on any that aren't. I'm sure Joe (Dr. Nelson), as a physician, would not sign off on people if he is not confident in them.
- Gunderson: We've kind of filtered this as far as excluding things that we wouldn't have in an optimal protocol. So really, we're going to include everything on this sheet (Figures 2-5). It's just a question of what the sequence should be.
- Nelson: Would you all agree with me on this, that everything on here could be or should be included in an optimal system, an optimal protocol, all of these components are valid components that are needed?
- Gunderson: Now the other point of confusion might come in where we have multiple agents that do the same thing, and selecting which one we want to use at any point in time. Let's take the case of a real sick patient in extremis. He's intubated, he's got PEEP, he's having assisted ventilation, he's sitting bolt upright, we have aminophylline running in, he's got lasix in, he's got nitrates in, he's got MS, he's got dopamine or dobutamine.
- Nelson: He wouldn't have both dopamine and dobutamine - He would have one or the other.
- Brown: Probably not simultaneous dopamine or dobutamine and aminophylline.
- Nelson: Probably not.
- Gunderson: So that's where we're going to need to make some choices.
- Brown: Something else we haven't addressed is the presence or absence of cardiac asthma overlying the congestive failure.
- King: Is that definitive?
- Nelson: Well that's where you have to be differential. If in cardiogenic shock, then you go with dopamine. I don't think that's really indicated without cardiogenic shock. If they are in cardiac asthma, then maybe you should go with a beta agent for reactive airway.
- Gunderson: Clarify if you would what you're specifically talking about: cardiac asthma?
- Brown: I'm talking about bronchospasm evidenced by wheezing in the presence of congestive failure.
- King: Is it quite obvious and definitive? Is it something that you're going to be able to define, diagnose?
- Nelson: The wheezing, I think that's the commonly perceived cardiac asthma. You can have some bronchospasm components there, but most often wheezing in congestive failure, commonly thought of as cardiac asthma, is simply edema of the bronchial walls. Again, treating the pulmonary edema symptoms will relieve the wheezing ideally. There may be some additional things you'd want to do.
- Brown: Mic (Gunderson) and I noted that we have very different clinical experiences as far as cardiac asthma. I have felt it to be relatively rare. In most of the patients I've come across that were in pulmonary edema, they did not have accompanying wheezing and yet Mic detected it a number of times. Would anybody else care to comment?
- Shepler: I have very little experience with it, but the experience I've had has always been coincidental to temperature change. The patient, he's loaded up with MS, nitro, and lasix, on high flow O2, and we're on our way out the door and to the ambulance. So often during colder months and with the temperature change, they get in the ambulance and have a wheezing component that they didn't have originally. At the hospital, the doctor chews us out and then gives some type of Bronchosal or something like that. The wheezes resolve themselves only to reveal rales once again. And, no apologies necessary. Now we're out the door. My question - is the bronchospasm perhaps also being precipitated by cold or temperature change?
- Nelson: It can be.
- Shepler: The only time I've ever seen it, it's always occurred in that setting.
- Nelson: Especially in the case of preexistent COPD. But I think we're kind of clouding the picture. For the purpose of this discussion, let's stick with straightforward pulmonary edema and congestive failure.
- Todoroff: You said that you felt that dopamine and dobutamine weren't indicated unless the patient was in cardiogenic shock. Or maybe for the purpose of this protocol we should go ahead and assume the patient isn't in cardiogenic shock. If they are, we will refer them to the cardiogenic shock protocol and take dopamine and dobutamine out of the protocol.

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B) ACUTE	85591	I) EMERGENCIES	10462
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E) *ON A&D	2000	P) SHOCK	16300
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I) 1983...87	1421878	T) *ON A&S	8
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EMERG MED CLIN NORTH AM 1983 Aug; 1(2):313-43			

Figure 1 - Literature Search - The medical literature was searched by computer. These searches were in pulmonary edema and congestive heart failure. Bold items dictated selection for printing.

- Gunderson: And if they did crash and they are in cardiogenic shock, we use two protocols.
Todoroff: What I'm saying is, we didn't put lidocaine in the protocol because we didn't feel it was for the purpose of treating pulmonary edema.
- Nelson: Does anybody disagree with that? Mike, how do you feel about it?
Brown: There are two different aspects to this. They could have simple pump failure without having dilatation sufficient enough to push them off the end of the Starling curve, which would be absolutely cardiogenic shock, pump failure, just from loss of muscle mass of the heart. Whereas, if they have the left ventricular dilatation to an extreme degree, that is another mechanism of the hypotension. And let's not forget the fact where we have a person who is extremely reactive to their own sympathetic hormones, where they have very strong central pulses, but simply have nearly imperceptible peripheral pulses. So we have three different things here to deal with. If you're going to automatically bump it into a protocol for cardiogenic shock, obviously many of the aspects are going to be the same. A lot of cardiogenic shock patients should be intubated. Most of them have, to a greater or lesser degree, congestive failure, superimposed on pump failure. There are a lot of aspects that can be grouped together. The Killip classification goes exactly the same way, they don't differentiate between serious failure in cardiogenic shock so much as simply put them in different categories - Killip I vs. Killip II. So you could almost, if necessary, combine protocols. Cardiogenic or CHF/cardiogenic shock protocol and simply extend it to the degree of seriousness of the patient.
- Gunderson: What you might end up doing is saying you wouldn't use these two agents (dobutamine and dopamine) unless the blood pressure dropped below a certain threshold.
Brown: Which was precisely my point.
King: At that point, would you have to make a definitive diagnosis? Would you feel comfortable?
Todoroff: You'd have to say the same thing about all the antidysrhythmics then.
Nelson: Don't you think we could put the dobutamine and dopamine under a cardiogenic shock protocol to be used for any cardiogenic shock?
Todoroff: With or without pulmonary edema?
Brown: It all depends on how you want to slice it up, really.
Todoroff: We use antidysrhythmics on patients that are in pulmonary edema, but they're not to treat pulmonary edema. So, we don't put them in pulmonary edema protocol.
- Gunderson: We could have someone in cardiogenic shock who does not have pulmonary edema.
Shepler: To put the two protocols together, combine them, is kind of implying that we have one patient that we watch go through the whole constellation of high output failure into low output failure and a terminal situation, which is not going to be

Therapeutic Objective: **Increase Oxygenation / Ventilation**

- Patient Positioning
- High Flow O₂
- Positive Pressure Ventilation
- Intubation, if any of the following:
 - Decreased level of consciousness
 - Decreased ventilation (RR>36 or <10)
 - Hypotensive
- PEEP (intubated cases)
 - 5-10 cm H₂O
- Aminophylline
 - 250 mg in 50 ml over 30 min
(if not on oral theophylline products)

Figure 2 - Therapeutic Objective: Oxygenation / Ventilation - The above items are modalities which are of clinical benefit by improving oxygenation and ventilation.

Therapeutic Objective:

Decrease Preload

- Patient Positioning - Upright
- Positive Pressure Ventilation
- Intubation, if any of the following:
 - Decreased level of consciousness
 - Decreased ventilation (RR>36 or <10)
 - Hypotensive
- PEEP (intubated cases)
 - 5-10 cm H₂O
- Lasix
 - 40 mg (80 mg if on oral lasix); repeat at double dose, if needed
- Nitrates
 - 2.5 mg isordil or 0.4 mg nitroglycerin
- Morphine
 - 2.5 mg increments @ 5 min intervals until diastolic 70-100 or systolic 120-160

Figure 3 - Therapeutic Objective: Decrease Preload - The above items are modalities which are of clinical benefit by decreasing the cardiac preload (venous return).

- the clinical picture that we typically see.
- Brown: Here's an interesting question for our physician here; Cardiogenic shock in the absence of pulmonary edema - Is that chiefly related to right ventricular infarction? It's now realized that right ventricular infarction is a lot more common than was once believed. If it's related to left sided pump failure, you should also get concomitant pulmonary edema.
- Nelson: Yes, you should. Usually in that case you'll have pulmonary edema, at least as a terminal event.
- Gunderson: Well the other thing that could happen is that they could get hypotensive quickly enough to where their vascular pressures go down so quickly that it doesn't force any fluid out of the alveoli.
- Brown: At least not to a clinically recognizable extent on bedside exam.
- Nelson: I would have to refer that question to a cardiologist.
- Brown: So it would be useful to separate cardiogenic shock from congestive failure?
- Nelson: I think it would, yes.
- Brown: However, if we're going to still talk about pulmonary edema, we could include that at the tail end or the bottom end of the congestive failure protocol. There's no reason that we can't put one therapy in more than one situational protocol.
- Nelson: Absolutely.
- Gunderson: Okay, let's look at some sequencing then. Of all these things, what would be one of the first things we'd want to do?
- Audience: Oxygenation, positioning, high flow O₂
- Nelson: Cardiac monitoring, establishing an IV.
- Gunderson: There's various ways we can handle that. The way we handled it in the Pinellas protocol, we had this supportive care protocol, which had the ABC exam, getting the history, etcetera. We could keep it specific to pulmonary edema. We could forgo specification of that (supportive care) and somehow reflect it in the documentation. Now, we have him upright, on high flow O₂...
- Brown: And/or ventilatory support.
- Gunderson: Depending on how bad they are.
- Nelson: Ventilatory support/positive pressure ventilation.
- Gunderson: These on an as needed basis.
- Nelson: If we're talking about protocols, do we recommend trying positive pressure ventilation before intubation, or do we intubate first and then recommend positive pressure with PEEP, if available?
- Brown: If they're in full respiratory collapse, obviously we can't wait to start the positive pressure ventilation.
- Todoroff: Can't you recommend positive pressure ventilation with or without intubation?
- Nelson: Yes.

- Todoroff: Yes.
- Gunderson: Recommend all three of them and if you don't have one, you don't have one.
- Nelson: Again, we're shooting for the ideal protocol.
- Gunderson: Yes, because we're saying we should get a peep valve.
- Nelson: So, ideally they're either intubated with positive pressure ventilation and peep or their not intubated with positive pressure ventilation.
- Brown: With intubation to follow.
- Nelson: Yes, with intubation to follow.
- Gunderson: Basically, we're saying that all patients, regardless of really how bad they are, should receive positive pressure ventilation as a modality. Patients should receive peep as a modality, but only if they have decreased consciousness, decreased ventilatory exchange and if they're hypotensive. Only then would we include intubation with the positive pressure ventilation.
- Nelson: Yes, and with decreased ventilation you could also put a respiratory rate in there.
- Gunderson: Thirty-six?
- Brown: I've often told people to consider CHF patients as candidates for intubation. Given a heart rate greater than 120, and that the respiration is greater than 36 and that any other factors would tip them over that line. For instance, hypotension, respiratory failure, decreased state of consciousness or central cyanosis.
- Nelson: Actually, you can put respiratory rate greater than 36 or less than 10. The less than 10 we've already gone through.
- Brown: Yes, it occurs to me we've left central cyanosis completely off that list of indicators.
- Nelson: I think central cyanosis is not a valid criterion. It is a good clinical sign. If you see it it's great, but I sure don't think anybody should get hung up on looking for cyanosis.
- Brown: What about in the presence of vital signs that are obviously poor.
- Nelson: If they meet the criteria on vital signs, you don't need to see a cyanosis.
- Brown: That's true.
- Gunderson: So the intubation of the area in the bracket, the intubation would only be implemented with these three parameters (Figure 2). Okay, do we want to start the aminophylline right away? How about the nitrates?
- Brown: Did we mention IV and ECG monitoring in there as part of our baseline?
- Gunderson: That's our general supportive care protocol.
- Brown: I would like to bring up one aspect here, and that is establishing an IV versus establishing a heparin lock. From some

Therapeutic Objective: **Decrease Afterload**

- Nitrates
 - 2.5 mg isordil or 0.4 mg nitroglycerin
- Morphine
 - 2.5 mg increments @ 5 min intervals until diastolic 70-100 or systolic 120-160- **Patient Positioning**
- High Flow O2 (by decreased sympathetic tone)
- Aminophylline
 - 250 mg in 50 ml over 30 min
(if not on oral theophylline products)

Figure 4 - Therapeutic Objective: Decrease Afterload - The above items are modalities which are of clinical benefit by increasing the afterload (aortic diastolic pressure).

- of the nursing journals, I've even come to understand that having it flushed with heparin isn't strictly necessary, at least in the initial management. You can flush with sodium chloride in small amounts.
- Gunderson: We're running it t.k.o. - We're not talking about a lot of fluid anyway.
- Brown: That may very well be true.
- Nelson: You're recommending a heparin lock?
- Brown: First of all, remember we're going to have to deal with humans in a critical care situation where you're liable to be without adequate manpower or without adequate space or adequate equipment. We don't have IV poles, we don't have a number of things there. In an ergonomic sense it might be more beneficial. It would certainly prevent a runaway IV from occurring if you had a heparin lock instead.
- Shepler: It's called a reseal.
- Nelson: I don't know, I'm afraid that it's...
- Gunderson: What don't you like about it?
- Nelson: I don't like the idea of not being able to flush something through readily. I don't know, I guess a heparin lock would work.
- King: It's not widely accommodated.
- Brown: I'm just talking about here, something that could be considered.
- Nelson: Sure.
- Brown: It's a technology that hasn't found its way, at least generally into prehospital care.
- Nelson: Although I've noticed as an ER physician, I see more attendings using it after the patient is admitted. Usually what happens, if I start a D5W t.k.o., they switch it to a heparin lock when they admit the patient.
- Brown: If we have a patient that's this sick, I've seen a great many of them arrest right at the point of passive exercise, in moving them from bed to stretcher or whatever. I tend to initiate at least a certain amount of treatment including pharmacologic therapy before I move them, trying to get them out of such acute distress. It (heparin lock) certainly eliminates a lot of the plumbing problems.
- Nelson: It does make a cleaner patient. If they do arrest, I'd rather have a line running.
- Brown: Then it's relatively easy to establish the line with a heparin lock in place.
- Nelson: I think that's just an extra move.
- King: Can we defer that to a literature search or something?
- Gunderson: Yes, that can be something debated at a later time, I'll just let it suffice that venous access should be obtained.
- Brown: We could put IV access/D5W.
- Nelson: Sure. We can put heparin lock if you want. I'm not that dead set on it.
- Brown: I was just throwing it out because it's something that could be advantageous. In an ergonomic sense, it would prevent the human error of accidentally having a runaway IV.
- Gunderson: Okay, so we've got this patient packaged with venous access of one sort or another, and he's on a monitor. We've done a history and physical. He's sitting upright, he's on high flow oxygen and he's receiving positive pressure ventilation. He might even be intubated. What would be our next intervention?
- Brown: Before even the IV we could certainly administer sublingual aids.
- Gunderson: Okay.
- Nelson: You can, but I don't recommend it. What happens if this patient does what we were talking about, bradys down or even goes into asystole, you don't have a line established.
- Shepler: One thing I've always been taught is that people that take nitro on a regular basis tend to tolerate it well, and it just makes

Therapeutic Objective: **Increase Left Ventricular Ejection Fraction**

- Aminophylline
 - 250 mg in 50 ml over 30 min
(if not on oral theophylline products)

Figure 5 - Therapeutic Objective: Increase Left Ventricular Ejection Fraction - The above item is a modality which is of clinical benefit by increasing the left ventricular ejection fraction.

Model Protocol: Congestive Heart Failure

- 1 - General Supportive Care**
 - ECG monitoring
 - Venous access (IV D5W, t.k.o. or reseat)
 - history, physical assessment
- 2 - Patient Positioning - Upright**
- 3 - High Flow O₂**
- 4 - Positive Pressure Ventilation**
- 5 - Intubation, if any of the following:**
 - Decreased level of consciousness
 - Decreased ventilation (RR>36 or <10)
 - Hypotensive
- 6 - PEEP (intubated cases)**
 - 5-10 cm H₂O
- 7 - Nitrates**
 - 2.5 mg isordil or 0.4 mg nitroglycerin
- 8 - Morphine**
 - 2.5 mg increments @ 5 min intervals until diastolic 70-100
or systolic 120-160
- 9 - Aminophylline**

Figure 6 - Model Protocol - The above protocol is a composite of the review and discussion of the session.

- good sense. But people that don't take nitro, if you give them a nitro, they'll hit the bricks sometimes.
- Nelson: Yes, I personally never give nitro in my ER without having an IV established. I know a lot of places do, I know many physicians give it in their office. I will not do it. I've had too many patients have problems.
- Brown: Not as a matter of routine, but this is also going to be a patient population due to their age and due to their sympathetic tone, where establishing rapid IV access might be in fact difficult to do. So once again, we get back to the modular as opposed to the linear protocol concept.
- Nelson: I'll say this, if there's any delay in establishing the IV, go ahead and give one sublingual nitro or a nitro spray while you're trying to obtain venous access.
- Gunderson: I think that sequencing we can leave to the discretion of the paramedic. If he's having a hard time he can go ahead and maybe come back a little later.
- Nelson: To summarize nitrates, I agree that nitrates should be right up there at the top of the list.
- Gunderson: What next, lasix? MS?
- Nelson: The only reason morphine is not above the nitro is nitro just takes a second to pop it in. If it's going to cause any delay at all, you want to go with MS first.

- Gunderson: Okay, then do we want to also go with lasix? Should the patient who's receiving nitrates and MS also receive lasix?
- Nelson: Yes, they should probably receive all three at some point. Nitrates, then your MS and then your first dose of lasix after your first dose of MS is on board. We're talking someone who is in extremis now. The harder you can hit them with multiple therapy, the better they're going to do.
- Gunderson: The only thing that really leaves is the aminophylline.
- Nelson: Probably the right place for it.
- Gunderson: Okay, so we would see that after the lasix, assuming that they're unresponsive. Because something that seems to be implicit is, if, say by the time we get them intubated and just give them some nitrates things have pretty well resolved, we'll stop at that point.
- Todoroff: You can include that little blurb that says how you're assuming that one step is unsuccessful before proceeding to the next - like they say in ACLS.
- Gunderson: Right. Well, it looks like we have somewhat of an optimal protocol (Figure 6).
- Nelson: It's interesting that our protocols are similar in what we do but certainly in almost reverse order from all the standing protocols out now.
- Gunderson: What's going to be really interesting is getting some feedback from some of the medical directors. The thing that I hope to have different at the next protocol roundtable, this being kind of a shake out session admittedly, is having the cardiologist sitting right there, and we can shoot some bullets at him and we can shoot some bullets at some ER docs and medical directors and maybe a pulmonary intensive care guy.
- Nelson: I'd like to see a pulmonologist and a cardiologist.
- Gunderson: So, three months hence we'll be having another similar session. After this meeting, I'd appreciate it if everyone would stick around a little while because I would like to get some feedback from you on how this session went and the sort of things we might be able to do on the next one that might meet your needs better, in your capacity as an inservice educator, as a clinician, or whatever other hat you might wear. If there are no other questions we'll go ahead and conclude the session and thanks alot.

Comments on the protocol and discussion are encouraged. Please forward your comments for publication to the correspondence editor. The next Protocol Roundtable symposium will be on Airway Control and Ventilation. It will be held March 2, 1988, in Tampa. Contact the Acute Care Foundation at (813) 988-0115 or consult the announcement elsewhere in this issue for additional information.

Book Review

Electrocardiography: The Monitoring Lead

By Richard Wiederhold

Richard Wiederhold's "Electrocardiography - The Monitoring Lead" seems to fulfill its prefaced intentions. It does provide the student/clinician with the means to interpret and recognize major dysrhythmias without the "cluttering" of the reader's mind by the more complex aspects of cardiology.

The necessary topics of cardiac physiology and EKG interpretation are addressed in an orderly, logical, yet simple manner. Major objectives and terminology are outlined at the beginning of each chapter and these are reinforced by summary and a self study guide at chapter's end. Also helpful in the emphasis of "key" points is the red boldface print of important "axioms" or facts placed in the page margin. These offer a visual separation and emphasis from the text.

Nearly one half of the book is dedicated to practice EKG rhythms, most of which are clear and readily interpreted given that the reader has understood the text. For a greater challenge there are 42 "advanced" rhythm strips provided.

Also included are tear-out flash cards with EKG tracing, interpretation and differential criteria of twenty one major dysrhythmias.

The Wiederhold Algorithm also deserves recognition as a useful tool with which the student/clinician may learn to rapidly identify underlying dysrhythmias. The algorithm structure is based upon five "primary criteria" of rhythm analysis. This structure enforces a methodical approach to interpretation.

Overall, Mr. Wiederhold's book appears to be a comprehensive, thorough text and a very useful resource for the student clinician.

Wiederhold R: *Electrocardiography: The Monitoring Lead*. 319 pages, soft cover. Harcourt, Brace, Jovanovich/Academic Press, Orlando, 1988.

Reviewer: Charles Anderson, REMT-P
Palm Harbor Fire Department
Pinellas County EMS

Abstracts

Compiled By: Journal Staff

Spinal Immobilization and the Logrolling Maneuver

MCGUIRE RA, NEVILLE S, GREENE BA, WATTS C

Journal of Trauma 27:525-531, 1987

The safety of the log-rolling maneuver, backboard, and scoop stretcher were evaluated in three studies. A normal volunteer was examined with AP radiographs when supine and in 90 degree log roll. A significant scoliotic sag is apparent during the log roll. A cadaver was studied after surgical destabilization of the L1-L2 joint in the spine. AP and lateral radiographs during a 45 degree log roll show a 21 mm AP displacement, 5 mm laterally and a 30 degree rotation of the joint. Following application of the backboard, AP displacement was corrected but lateral and rotary deformity remained. On the scoop, AP displacement was reduced to 6 mm, lateral displacement to 6 mm and the rotary displacement was corrected. A 38 y.o. male with recent, and previously known, T12-L1 fracture was log rolled during angiography for suspected aortic aneurism. AP and lateral films reveal 7 mm lateral displacement, without AP or rotational distortion. The authors concluded that the backboard and scoop offer adequate thoracolumbar spine immobilization, but the log roll maneuver could lead to extreme spinal motion and neurological compromise. No definitive solutions are offered, nor are any guidelines for prehospital care of spinal injuries presented. These issues must be addressed by further research.

Request reprints from: Robert McGuire, MD, Department of orthopedics, Naval Hospital, Portsmouth, VA, 23708.

A Radiographic Comparison of Prehospital Cervical Immobilization Methods

GRAZIANO AF, SCHEIDEL EA, CLINE JR, BAER LJ

Annals of Emergency Medicine 16:1127-1131, 1988

The efficacy of the California Stif-Neck Collar (CSC), Kendrick Extrication Device (KED) and the Extrication Plus-One (XP-One) were compared to standard short board technique (SBT) for cervical immobilization in 45 normal volunteers. Radiographic studies were performed sagittally and frontally. Direct protractor measurement was made for axial rotation. The KED, XP-One, and SBT studies did not incorporate cervical collars in conjunction with those devices. In lateral bending, SBT was superior to CSC and KED, but was comparable to the XP-One. For axial rotation and flexion, there were no significant differences. In extension, the SBT and KED were comparable but the CSC and XP-One were significantly less effective. In a total range of motion in mean degrees of movement (flexion-extension between occiput and C7), the KED and XP-One

were comparable to the SBT, but the CSC did not perform as well. The authors conclude that the SBT is the technique against which other methods should be compared. The KED and XP-One were found efficacious and superior to the CSC alone.

Request reprints from: Anthony Graziano, MD, 5042 Seven Mile, NE, Belmont, MI 49306

The Effect of Axial Traction During Orotracheal Intubation of the Trauma Victim with an Unstable Cervical Spine

BIVINS HG, FORD S, BEZMALINOVIC Z, et al
Annals of Emergency Medicine 17: 25-29, 1988

Axial traction with and without orotracheal intubation of 17 blunt trauma arrest victims were studied radiographically immediately following unsuccessful resuscitation. Four of these cases had unstable cervical injuries, including C6-7 fracture/dislocation, hangman's fracture, and two atlanto-occipital dislocations (AOD). Axial traction with intubation produced a mean distraction at the fracture site of 7.75mm. In axial traction alone, 4mm produced subluxation at the C6-7 injury was produced along with the distraction. The authors conclude that intubation of trauma victims prior to complete radiographic comparison should be via the endotracheal method. If nasotracheal intubation is contraindicated, cricothyrotomy should be utilized.

Request reprints from: Herbert Bivins, MD, Department of Emergency Medicine, Valley Medical Center, 445 S. Cedar Ave., Fresno, CA. 93702

Thrombolytic Treatment in Acute Myocardial Infarction

ACAR J, VAHANIAN A, MICHEL P, ET AL
Seminars in Thrombosis and Hemostasis 13:186-200, 1987

A thorough review article (including 129 citations) is presented on the pathophysiologic rationale and historical development of thrombolytic treatment of acute myocardial infarction (AMI). The pathophysiologic role of thrombosis in AMI has been controversial. Angiography performed within 6 hours of the onset of symptoms shows an 80% incidence of thrombosis, compared to approximately 55% 6-12 hours after post-infarct. A natural thrombolysis is suggested. However, thrombosis is not the only etiology of AMI. Disorders such as ulceration of atherosclerotic plaques, spasm and platelet activation are also cited as alternative or contributing events. However, thrombosis appears to be the most common and therefore the impetus for a considerable world-wide thrombolytic research effort over the past two decades. Animal studies with controlled coronary occlusion show that reperfusion after 40 minutes allows salvage of 60-70% of the effected myocardium. After 3 hours, salvage drops to 10% and is negligible after 6 hours. This highlights the importance of early thrombolytic intervention as has been confirmed with a number of studies using various agents. The authors provide insight on the following agents: streptokinase, urokinase, ACYL enzyme, prourokinase, and tissue plasminogen activator (t-PA) as well as percutaneous coronary angioplasty. The paper concludes that thrombolysis is effective if given early and is

particularly useful in younger patients with critical infarction. These agents cannot be administered in all patients, as some contraindications must always be observed. Residual stenosis often remains after thrombolysis and coronary angioplasty may be useful here, but logistics limit widespread availability on an acute basis. Heparinization should follow thrombolysis, but dosage remains controversial. They close with the reminder that thrombolysis is not the only treatment of AMI.

Request reprints from: Jean Acar, MD, Department of Cardiology, Hospital Tenon, 4 rue de la Chine, 75020 Paris, France

Metabolic Acidosis After Acute

Ibuprofen Overdosage

LINDEN CH, TOWNSEND PL

Journal of Pediatrics 6:922-925, 1987

Two case reports of metabolic acidosis following ibuprofen overdosage are presented. The first case is a 2 y.o. male with a 666 mg/kg ingestion. The second is a 15 month old male who sustained a 560 mg/kg ingestion. Both had pH values of 7.27 and bicarbonate was 19 mEq/l and 14 mEq/l, respectively. Anions gaps of 15 mEq/l and

16 mEq/l were found, respectively. Discussion in the paper outlines that peak serum concentrations occur 1-2 hours after single therapeutic ingestion, the pharmacokinetics are not cumulative, and delayed absorption does not occur with overdosage. Ibuprofen is highly bound to proteins and has a very limited volume of distribution (0.18 l/kg). Hepatic metabolism is the primary means for elimination with a half-life of 0.9 to 2.5 hours. Minimal toxic exposure is indicated as 100 mg/kg, but many individuals do not exhibit and signs or symptoms at these levels. The metabolic acidosis eluded to earlier was relatively mild and of brief duration in both patients. Treatment should be directed towards limiting absorption and providing standard supportive care. GI decontamination and 4 hours of observation are recommended for all intentional and pediatric cases with ingestion of at least 100 mg/kg. In cases of over 400 mg/kg, gastric lavage, activated charcoal, or both may be preferable to induce emesis in cases of coma or seizure. Alkaline diuresis and multiple dose charcoal are of very limited value.

Request reprints from: Christopher Linden MD, Department of Medicine, University of Massachusetts Medical Center, Worcester, MA 01605

Equipment Review

Tube-Stat Lighted Tip Endotracheal Tube Stylette

Reviewer: Patrick Shepler, REMT-P

Clearwater Fire Department; Pinellas County EMS

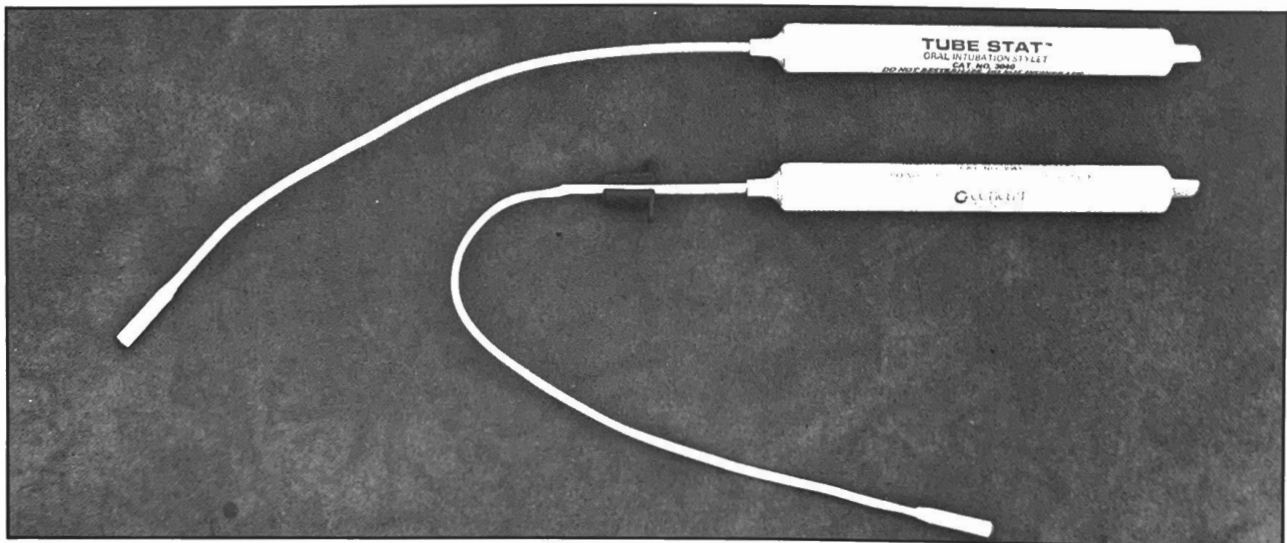
Tube-Stat® (*Concept, Inc., Clearwater, FL*) is a reusable lighted tip stylette which provides transillumination of the trachea for confirming correct placement during the initial procedure of endotracheal intubation and as a periodic recheck. It also provides supplemental lighting during direct laryngoscopy.

The device consists of a light securely affixed to the distal tip of a malleable stylette. The stylette is permanently attached to a battery source about the size of a common penlight (Figure 1). The currently available model is only 25 cm. long, requiring one to trim the proximal end of an endotracheal tube to get sufficient depth into the tube. A new longer model should be available in a few months that will eliminate the need for tube trimming. An adjustable collar will be added to the stylette that will allow control of stylette depth in the tube. A flaccid stylette is also available, which may be used for transillumination during blind nasotracheal intubation.

Transillumination of the trachea is reported to be a reliable indicator of correct tube placement (1). The author has had considerable experience with the device and finds that the bright mid-line transillumination from the trachea is easily distinguished from the dull, diffuse light which appears on the side of the neck with esophageal placement. Bright ambient sunlight during outdoor intubation can obliterate the transillumination. However, these conditions also make it difficult to see in the pharynx using standard laryngoscope illumination with one's pupils constricted by the sunlight. Here, the lighted stylette provides very helpful supplemental lighting in the pharynx to visualize landmarks during outdoor intubation. The extra light is often helpful indoors as well. Blind orotracheal intubation is also facilitated by the stylette.

The flaccid stylette is now in use by the Bayflite helicopter program of the Bayfront Medical Center in St. Petersburg, Florida. They find it helpful in performing in-flight blind nasotracheal intubations and in re-confirming tube placement enroute. The high levels of noise in the helicopter make lung auscultation an unreliable means by which to confirm tube placement. This is also applicable to high ambient noise environments on the ground.

The Tube-Stat® is a useful tool in the prehospital environment for performing standard and various alternative methods of endotracheal intubation. Transillumination is another utilization of the five senses to determine and re-determine proper endotracheal tube placement.



Marshal Dickson

Figure 1 - Tube-Stat® Lighted Tip Endotracheal Tube Stylette - This newer model features an adjustable collar to vary depth of placement inside tube. It is no longer necessary to cut the endotracheal tube prior to use.

Calendar

CALENDAR EDITOR: Steve Gross, REMT-P

The Journal wishes to promote continuing education and other academic events open to the EMS and critical care community by offering free listings of such events in this section. Please submit the name of the event, sponsoring organization, date, time registration information, tuition and a contact person address and a phone number for additional information. Calendar items must be received no later than 45 days prior to our quarterly publication dates on the first of January, April, July and October to appear in the next issue. Send items to the Calendar Editor, Tampa Bay EMS Journal, P. O. Box 280173, Tampa, Florida 33682 or call 813/988-0115.

January 20-22, 1988

Medico-Legal Aspects of Sexual Battery Crimes

Orlando, Florida - HJ Plaza Inn, 603 Lee Road. Tuition \$150. For additional information, contact Robert L. Milke, Valencia Community College, P. O. Box 3028, Orlando, Florida 32802. 305/299-5000, ext. 3265.

January 29-30, 1988

Southwest Florida Regional Medical Center Trauma Symposium

Sanibel Island, Florida - Sundial Beach and Tennis Resort. For further information, contact Peggy Bateman, 813/939-8550.

February 11-14, 1988

Disaster '88 Management Seminar

Orlando, Florida - Hyatt Orlando. Sponsored by the Florida Chapter ACEP. For further information, contact Craig Story, Polk Community College, 999 Ave. H NE, Winter Haven, Florida 33880, 813/297-1000.

February 18-20, 1988

2nd Annual Conference on Childhood Resuscitation and Stabilization

Tampa, Florida - Hyatt Regency-Tampa (Downtown). Sponsored by the Center for Emergency Medical Education, Inc., Tampa Emergency Associates for Medicine and the University of South Florida College of Medicine. For further information, call 813/251-6911.

February 18, 1988

IV Therapy Theory and Skills

St. Petersburg, Florida - Days Inn and Marina (formerly Sheraton), 6800 34th St. S. Tuition \$95. For further information, contact Susan Villaesevs, Carondelet Management Institute, P. O. Box 12069, Tuscon, Arizona 85732, 602/721-3838.

February 26-28, 1988

BTLS Instructor Course

Tampa, Florida - University of South Florida Campus. Tuition \$154. For additional information, contact Florida Chapter ACEP, 5824 S. Semoran Blvd., Orlando, Florida 32822, 305/281-7396.

March 2, 1988

Protocol Roundtable: Airway Management

Tampa, Florida - Embassy Suites Hotel, 4400 W. Cypress Ave. Tuition \$30 for ACF members, \$40 non-members. For additional information, contact the Acute Care Foundation, P. O. Box 280173, Tampa, FL 33682, 813/988-0115.

March 4, 1988

1988 Florida Conference on Poison Information

Tampa, Florida - Lincoln Hotel, 4860 W. Kennedy Blvd. Tuition \$45. For further information, contact Judy Sommers, Continuing Education Coordinator, College of Public Health, University of South Florida, 13301 Bruce B. Downs Blvd., Tampa, Florida 33612, 813/974-3623.

March 5-6, 1988

Terrorism and Trauma '88

Tampa, Florida - St. Joseph's Hospital, 3001 W. Buffalo Ave. Tuition \$90. For further information, contact Kellie S. Fitzpatrick, St. Joseph's Health Care Center, Community Relations Dept., P. O. Box 4227, Tampa, Florida 33677, 813/870-4340.

March 27, 1988

EMS Street Survival Seminar

Ft. Lauderdale, Florida. Tuition \$49. For further information, contact Calibre Press, Inc. (800) 323-0037.

April 5, 1988

Trauma Care: Thoracic and Abdominal Injuries

Orlando, Florida - Best Western Catalina Inn, 3401 L.B. McLeod Rd. Tuition \$94. For additional information, contact Health and Education Council, Inc., 7201 Rossville Blvd., Baltimore, MD 21237, (301)686-3610.

April 15, 1988

Controversies in Critical Care Pharmacology

Jacksonville, Florida - Park Suite Hotel, 9300 Baymeadows Rd. Tuition \$94. For additional information, contact Health and Education Council, Inc., 7201 Rossville Blvd., Baltimore, MD 21237, (301)686-3610.

April 20, 1988

The Acutely Ill Child

Orlando, Florida - Best Western Catalina Inn, 3401 L.B. McLeod Rd. Tuition \$94. For additional information, contact Health and Education Council, Inc., 7201 Rossville Blvd., Baltimore, MD 21237, (301)686-3610.

April 23-24, 1988

Advanced Cardiac Life Support (AHA)

Polk Community College, 999 Ave. H, NE, Winter Haven, Florida 33880. For further information, contact Craig Story, 813/297-1000.

May 6-7, 1988

1st Annual National Symposium on Prehospital Medical Control and Continuing Education

Embassy Suites Hotel, 4400 W. Cypress Ave., Tampa, Florida. Tuition \$100 for ACF members, \$120 non-members. For additional information, contact the Acute Care Foundation, P. O. Box 280173, Tampa, FL 33682, 813/988-0115.

May 7, 1988

Advanced Cardiac Life Support Provider Course

USF College of Medicine Medical Center Cafeteria - 7:30 a. m., For further information, contact The Center for Emergency Medical Education, Inc., Harbourside Medical Tower, 4 Columbia Dr., Suite 810, Tampa, Florida 33606, 813/251-6911.

May 9, 1988

***Arterial and Mixed Venous Blood Gases:
Advanced Concepts***

Orlando, Florida - Best Western Catalina Inn, 3401 L.B. McLeod Rd. Tuition \$94. For additional information, contact Health and Education Council, Inc., 7201 Rossville Blvd., Baltimore, MD 21237, (301)686-3610.

June 10, 1988

Crisis Intervention Skills for Emergency Department Personnel

Orlando, Florida - Best Western Catalina Inn, 3401 L.B. McLeod Rd. Tuition \$94. For additional information, contact Health and Education Council, Inc., 7201 Rossville Blvd., Baltimore, MD 21237, (301)686-3610.

July 23-24, 1988

Basic Trauma Life Support

Polk Community College, 999 Ave. H, NE, Winter Haven, Florida 33880. For further information, contact Craig Story, 813/297-1000.

The Tampa Bay EMS Journal is a unique publication, devoted to acute care medicine in the prehospital, emergency department and critical care environments. As a formally refereed medical journal, it provides a forum for the presentation and debate of issues related to patient care, research, administration, education and logistics associated with emergency medical services.

The Journal is published quarterly by the Acute Care Foundation, a non-profit organization of members of the EMS community. Your subscription support of efforts such as this will help the Foundation to catalyze the high quality research and education programs showcased in the Journal. Consider it an investment in your EMS community.

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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.

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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)988-0115 for any additional information. Thank you for your participation and support.

Correspondence

The Correspondence section is designed to accommodate feedback and debate regarding papers previously published in the Journal. It is also an appropriate place for other smaller items of interest that may not require the space of a separate article. The Correspondence section can be one of the most interesting parts of a professional publication, where published debate can fully explore all sides of an issue. We invite critique and comment. Direct all correspondence to be considered for publication to the Correspondence Editor, Tampa Bay EMS Journal, P.O. Box 280173 Tampa, Florida 33682 (813) 988-0115.

Editorial

In Search Of Jedi: Revision of Journal Distribution Policies

The Acute Care Foundation and the *Tampa Bay EMS Journal* were created to help catalyze a trend towards the academic sophistication of EMS, particularly in the prehospital sector. The founders took their best estimates of how these efforts should initially be organized and promoted to meet these academic goals.

The *Journal* has been very successful. It is being very well received by the medical directors, administrators, and educators in the prehospital community. Among field personnel, there has been a predicted reception. The *Journal*, in contrast to other prehospital EMS publications, is not attempting to appeal to the broadest possible field audience in the fiftieth percentile. They already have several trade publications, which are serving an excellent purpose. These publications are slowly increasing the quality of their content with better manuscripts and more critical perspectives on the industry. The target audience for the *Journal* are those in the ninetieth percentile. We want to provide a professional organization to catalyze original EMS research, and a forum in which the prehospital and in-hospital communities can explore and debate their common issues in acute care medicine. Hence, the people in the field we have gotten the strongest response from are the very dedicated and motivated, who are always looking for those all too rare pathways for professional growth. Unfortunately, these extremely talented individuals are usually left latent by the agencies, unchallenged in their substations. However, they are the future leadership of our industry. They will become the educators, administrators and innovators.

Our editorial board, membership and our advertisers know that the magazine is being read by the decision makers, present and future. We were told us ahead of time, but we had to test for it, that substation distribution would not be a practical mechanism to reach that latent audience. The experience gained from the first few issues allow us to refine our distribution policies to reach a greater share of the current and future vanguard. The following policies will commence with the April-June, 1988 issue.

Pass along circulation and participation from the critical care community has been extremely limited. We will focus standard distribution in our primary encatchment area in the Tampa Bay area to the nurse managers of the CCU and SICU areas. In the emergency department, we will expand our distribution to include the medical director, nurse managers and all emergency department physicians. Our staff faces a substantial task in defining this audience for our distribution list. We will attempt to do so through the ED physician groups and nurse managers. Elsewhere in the hospital, we will distribute copies to the education department, medical library and the director of marketing/public relations.

The prehospital distribution will also be expanded. In our primary distribution area, we will distribute to all EMS medical directors, educators, administrators, supervisors and advisory council members. On a statewide secondary distribution, we will distribute to the medical director, training officer and administrator for all ALS providers.

The latent talent in the substation will present a challenge. How do we know who is in that ninetieth percentile or aspires to be? You know who they are. They are the people you work with who you would want to care for your family in an emergency. They go to seminars. They have instructor credentials. They are the ones you learn the most from when you work with them. If the shoe fits, we want to send you the *Journal*. We want your help in building the academic programs of the Foundation and in the process, establish your own experience, credentials, and reputation to facilitate your professional growth and self-actualization. Let us know who else fits the profile or has potential to. This type of direct and personal networking is essential for us to reach our intended audience in the substations and among the staffs of the emergency departments and critical care units.

There is a subscription form in this issue of the *Journal* that I would appreciate you taking the time to fill out and send to us if you would like to continue to receive this publication. Also, tell us who else we should send it to. Your feedback would be appreciated. What can we do to better serve your educational and academic needs? If you appreciate what we have done and would like to help us continue to do so, please show it by purchasing a subscription. Should you want to participate in the Foundation, with the *Journal*, the television network, seminars and symposia, post-graduate education programs on either activities, join the Foundation. Membership includes a subscription. You may remain skeptical. We are willing to speculate on you and anybody who you refer to us. We will include you on our distribution list for the next issue, without charge. In return, seriously consider your career potentials and the opportunities you have available for professional growth. Look at what you have for post-graduate level education materials, original prehospital research and high quality symposia. You can then decide if we deserve your support and participation with a subscription or a membership in the Foundation. We are an investment in your EMS community. Your participation will help insure a healthy dividend.

Michael R. Gunderson, REMT-P
Editor



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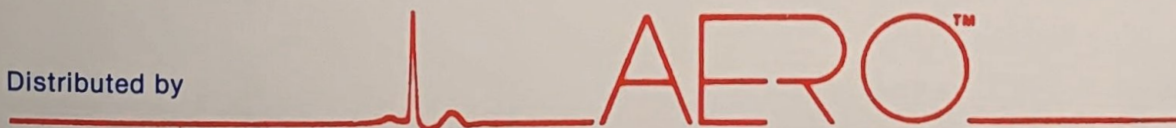
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ERRATA

In the January-March, 1988 issue, the proceedings for the Protocol Roundtable on Acute Congestive Heart Failure (*Protocol Roundtable: Acute Congestive Heart Failure. Tampa Bay EMS Journal 1(3):62-70, 1988*) contains errors in Figure 6 on page 69, listing items for the model protocol. Most significantly, the section on furosemide and the dosage for aminophylline were omitted. The corrected contents for the figure, with underlined changes, are shown below.

1. General Supportive Care
 - ECG monitoring
 - Venous access (IV D5W, t.k.o. or reseat)
 - History, physical assessment
2. Patient Positioning - Upright
3. High Flow O₂
4. Positive Pressure Ventilation
5. Intubation, if any of the following:
 - Decreased level of consciousness
 - Decreased ventilation (RR>36 or <10)
 - Hypotensive
6. PEEP (intubated cases)
 - 5-10 cm. H₂O
7. Nitrates
 - 2.5 mg. isosorbide or 0.4 mg. nitroglycerin, sublingual
8. Morphine
 - 2.5 mg. increments @ 5 min. intervals until diastolic 70-100 or systolic 120-160
9. Furosemide
 - 40 mg. (80 mg. if on oral furosemide); Repeat at double dose, if needed
10. Aminophylline
 - 250 mg. in 50 ml. over 30 min.
(if not on oral theophylline products)

Errata normally appear in a subsequent issue of the Journal. However, the significance of these errors prompted our decision to publish this special errata. We apologize for the omission and any confusion it may have caused. Despite the very limited time and resources under which we produce the Journal, we make no excuses for such errors - We take our responsibility for the content of this publication very seriously. We appreciate the support of our readers and assure that as we grow, we will continue to improve the composition and academic quality of its contents.

The Editorial Staff