Tampa Bay EMS Journal
Copyright © 1988 by the Acute Care Foundation
Volume 1, Number 3
January - March, 1988

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 their 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 the 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 Bronchosol 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.

LIST	REFERENCES
A) PULMONARY EDEMA	6590
B) ACUTE	85591
C) *ON A&B	1000
D) REVIEW	193080
E) *ON C&D	60
F) EMERGENCY MEDICAL SERV	4206
G) *ON C&F	**2**
H) *ON A&F	**10**
I) EMERGENCIES	10462
J) EMERGENCY SERVICES, PS	436
K) *SUM IJ	10898
L) 1980...88	2224859
M) *ON E&L	**22**

(Non-English language publications excluded off-line)

Wood LD Prewitt RM
Cardiovascular management in acute hypoxemic respiratory failure.
AM J CARDIOL 1981 Apr; 47(4):963-72

OBrodovich HM Mellins RB
Bronchopulmonary dysplasia. Unresolved neonatal acute lung injury.
AM REV RESPIR DIS 1985 Sep; 132(3):694-709

Redding RA
Modern concepts about drowning.
CAN MED ASSOC J 1974 May 4; 110(9):1057

Shapiro BA Cane RD Harrison RA
Positive end-expiratory pressure in acute lung injury.
CHEST 1983 Mar; 83(3):558-63

Renner IG Savage WT 3d Pantoja JL Renner VJ
Death due to acute pancreatitis. A retrospective analysis of 405 autopsy cases.
DIG DIS SCI 1985 Oct; 30(10):1005-18

Nowakowski JF
Acute alveolar edema.
EMERG MED CLIN NORTH AM 1983 Aug; 1(2):313-43

Ettien JT Webster PD 3d
The management of acute pancreatitis.
ADV INTERN MED 1980; 25:169-98

Stathers GM
Near drowning: principles of emergency care.
AUST FAM PHYSICIAN 1981 Dec; 10(12):943-6

Guntupalli KK
Acute pulmonary edema.
CARDIOL CLIN 1984 May; 2(2):183-200

Rude RE
Acute myocardial infarction and its complications.
CARDIOL CLIN 1984 May; 2(2):163-71

Depman ST Wendel CH
Congestive heart failure and pulmonary edema.
PRIMARY CARE 1986 Mar; 13(1):71-5

LIST	REFERENCES
A) HEART FAILURE, CONGEST	16911
B) ACUTE	85591
C) *ON A&B	2000
D) REVIEW	193080
E) *ON A&D	2000
F) *ON C&D	100
G) 1980...87	2224835
H) *ON E&G	2000
I) 1983...87	1421878
J) *ON B&E&I	14
K) ENGLISH	3657165
L) HUMAN	3294441
M) *ON J&K&L	**6**
N) EDEMA, CARDIAC	156
O) MYOCARDIAL INFARCTION	42803
P) SHOCK	16300
Q) SHOCK, CARDIOGENIC	2494
R) *SUM OPQ	61597
S) EMERGENCY MEDICAL SERV	4206
T) *ON A&S	**8**
U) *ON F&S	0
V) *ON F&I	**14**

Rosenthal ME Oseran DS Gang E Peter T
Sudden cardiac death following acute myocardial infarction.
AM HEART J 1985 Apr; 109(4):865-76

Goodman LR Putman CE
Diagnostic imaging in acute cardiopulmonary disease.
CLIN CHEST MED 1984 Jun; 5(2):247-64

Nowakowski JF
Acute alveolar edema.
EMERG MED CLIN NORTH AM 1983 Aug; 1(2):313-43

Jaffe AS
Complications of acute myocardial infarction.
CARDIOL CLIN 1984 Feb; 2(1):79-94

Swedberg K
Use of nitrates in acute and chronic congestive heart failure.
DRUGS 1987; 33 Suppl 4:147-9

Blakeley C Tinker J
Vasodilators in acute circulatory failure.

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 O_2**
- **Positive Pressure Ventilation**
- **Intubation, if any of the following:**
  - **Decreased level of conciousness**
  - **Decreased ventilation (RR>36 or <10)**
  - **Hypotensive**
- **PEEP (intubated cases)**
  - **5-10 cm H_2O**
- **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 conciousness
  - Decreased ventilation (RR>36 or <10)
  - Hypotensive
- PEEP (intubated cases)
  - 5-10 cm H20
- 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 O_2

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 O_2...

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 reseal)
- 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 conciousness
- Decreased ventilation (RR>36 or <10)
- Hypotensive

6 - **PEEP (intubated cases)**
- 5-10 cm H_20

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.