PANEL DISCUSSION

Guest Editor: Howard B. Sprague, M.D.

Prevention and Management of Cardiovascular Emergencies

The Panel was comprised of the following members: Howard B. Sprague, Boston, Mass., Moderator; James Metcalfe, Boston, Mass.; Benjamin M. Gasul, Chicago, Ill.; Louis A. Soloff, Philadelphia, Pa.; Maurice Sokolow, San Francisco, Calif.; and Stewart G. Wolf, Jr., Oklahoma City, Okla.

The Panel Discussion on prevention and management of cardiovascular emergencies was conducted at the Thirtieth Scientific Session of the American Heart Association held at the Sherman Hotel, Chicago, Illinois, on Friday afternoon, October 25, 1957.

Moderator Sprague: Those of you who were here this morning are familiar with the forms and faces of our panelists this afternoon. I am moderator or chairman, and Mr. Dewey Barrett recently said that the position of a chairman is that of a minor official at the bull fight who opened and closed the gates to let the bull in and out. Well, at least like the Portuguese bull fighter we will let the bull live rather than kill him at the end.

I have great many questions here. I tried to be selective at lunch time. We are going to start with a question to Dr. Metcalfe. "Do you consider toxemia or pre-eclampsia as a cardiovascular emergency? What is the current management of the acute hypertensive episodes accompanying these conditions?"

Dr. James Metcalfe: I do consider pre-eclampsia as a cardiovascular emergency, at least historically. In the series collected before 1950, about 10 per cent of the maternal mortality in pre-eclampsia was due to pulmonary edema, and a great amount of theoretical work was done without much experimental work trying to explain whether or not this was a myocardial toxemia, as was suggested by Dexter and White among others, or whether this could all be explained by the effect of hypertension.

Fortunately from the standpoint of the population and unfortunately from my own experience, pre-eclampsia has disappeared from our population. I have never seen pulmonary edema in a patient of the Boston Lying-In Hospital or its affiliated service. I have seen pulmonary edema in patients sent to the hospital who have had an atrocious sodium intake. I believe that the decline in toxemia of pregnancy is due to a realization of the role of sodium in its production and to prophylaxis by sodium restriction.

Once pulmonary edema occurs in these circumstances, I assume it would be managed as pulmonary edema in general with more attention to diuresis because these people have a demonstrable large water content although not a large blood volume.

As for the acute hypertensive emergency itself, I would gather that, as in other forms of acute hypertension and hypertensive crises, any of the high potency agents will get you over the acute phase; diuresis and eventually delivery will eradicate the disease.

Moderator Sprague: Dr. Gasul, at what age do you discontinue streptococcus prophylaxis in children with rheumatic fever? At what age would you not start the streptococcus prophylaxis program when rheumatic disease is first diagnosed in adults? You may
have to ask someone else to answer the second question.

Dr. Benjamin M. Gasul: In children we do not discontinue prophylaxis at all. We continue to give it to them until we send them to an adult cardiologist. That we used to do at 12 years. Then we extended it to 14. Now we take care of them up to 15 years of age.

Moderator Sprague: Do you think you would be unhappy if the adult cardiologist did not keep up with the prophylactic regimen?

Dr. Gasul: This is one time when it is good to be a pediatrician. I do not know.

Moderator Sprague: I would like to ask the whole panel this question: Do you continue prophylactic therapy for rheumatic patients indefinitely?

Dr. Maurice Sokolow: Frankly, I do not personally. I think up to the age of 30, if a patient has had rheumatic fever during the preceding 5 years, say, we would continue prophylactic therapy. If we saw patients after the age of 30, particularly after the age of 35, who had not had acute rheumatic fever for a number of years, we would probably not start prophylaxis because of the potential hazard from the continued use of the drug. We would, however, see to it that the patient was observed closely, had blood cultures, and was watched for respiratory infections, and if he did not develop streptococcal infection, we would not give it. It is a calculated risk. It is now known that if you use prophylaxis you will prevent streptococcal infection. But acute rheumatic fever is so rare after the age of 30, we would probably not use it.

Moderator Sprague: Dr. Metcalfe, do the patients at the Lying-In Hospital have prophylactic therapy?

Dr. Metcalfe: Yes. I am not a pediatrician and I can only take them another 20 years. Childbearing ends at about 45. So I do not have to make the decision, but I would say unqualifiedly I would put the patient on penicillin and tell the patient to keep on it until something better comes along.

There is one point I would make in this connection: people in the childbearing age have children and children are the best targets in the world for streptococci and everything else. I think that any patient who has ever had rheumatic fever should go back to penicillin when her children get into school.

Moderator Sprague: Would you take the patient up at age 45?

Dr. Louis A. Soloff: I agree with Dr. Sokolow's statement with one exception. I let the patient go ahead and use penicillin at any age. In other words, the publicity is so great at the moment and we have no evidence that it will not be good. So I think that if the patient wishes to take penicillin by mouth daily, whether he is 40, 50, or 60, he may certainly do so.

Moderator Sprague: Do you disagree with this, Dr. Wolf?

Dr. Wolf: We are in a quandary about this, not knowing what is the best thing to do. Very often the patient will settle the thing himself by just discontinuing the taking of penicillin, and if he has not had rheumatic infection for a good many years one is likely not to see it. We are inclined to put special emphasis on the literal interpretation of the meaning of the doctor as a teacher and to give the patient careful instructions about having throat cultures made at times when they do have upper respiratory infections, particularly sore throats. I think that is one of the most important things to get across to the individual.

Moderator Sprague: Dr. Sokolow, I will ask you some short questions. After treatment with quinidine for acute paroxysmal atrial fibrillation and sinus rhythm is established, should quinidine be continued indefinitely?

Dr. Sokolow: That, of course, depends on what caused the atrial fibrillation in the first place. If it were an acute episode, possibly related to infection or acute emotional stress, we would not. We would wait to see whether or not the fibrillation was perpetual. If it occurred in a patient with rheumatic heart disease who has a large left atrium and who may be expected to have recurrent attacks,
we would put him on continuous prophylaxis. If the patient, regardless of the cause, had recurrent attacks of acute fibrillation, we would then perhaps first try digitalis for prevention, and if that did not work we would put him on permanent quinidine therapy.

Moderator Sprague: Professor Sokolow, for how long would you have a patient on quinidine?

Dr. Sokolow: Certainly for 5 years; possibly longer.

Moderator Sprague: If no quinidine blood levels are available, what total over-all dosage of quinidine orally gives a toxic level?

Dr. Sokolow: That varies with individuals, but a safe moderate dose that probably should not be exceeded unless one can do frequent electrocardiograms and observe the patient very closely is probably 3 Gm. a day, and for the person who uses quinidine infrequently probably less than that.

Moderator Sprague: I have had patients on much longer than 5 years, but then I am a little older than you are. I have had them on 20 years.

May quinidine be used safely in treating arrhythmias due to digitalis?

Dr. Sokolow: Yes, as a matter of fact, that was the common form of therapy prior to the recent emphasis on the importance of potassium in digitalis toxicity. Moderate doses of quinidine are quite effective in treating arrhythmias due to digitalis toxemia.

Moderator Sprague: If a patient gets procaine amide, then you give one of the others to try to stop it?

Dr. Sokolow: I would say I think the arrhythmia due to digitalis might be considered to be an irritable arrhythmia whereas an arrhythmia due to procaine amide might be considered to be a depressive one. I think if the patient had atrial tachycardia from either procaine amide or quinidine, I would be very hesitant to use the other drug.

Moderator Sprague: Dr. Soloff, how long should one delay mitral commissurotomy in a patient who has mitral stenosis, atrial fibrillation and, recent peripheral embolization, granting indication for commissurotomy exists. Should anticoagulants be used during or after commissurotomy? The peripheral emboli were major factors.

Dr. Soloff: We would proceed with mitral valvotomy while the patient was in the hospital recovering from atrial fibrillation. About anticoagulant therapy, we would proceed with anticoagulant therapy after operation. We have as yet not had the courage to give it during operation as some of our Swedish colleagues have, so that it might be worth trying in this country. As far as I know, no one has as yet, but we would proceed immediately after operation.

Moderator Sprague: If you had a patient who had multiple emboli would you put the patient on anticoagulants for some weeks or months prior to mitral valve surgery, with the idea that you might then be past the period when thromboembolism was likely?

Dr. Soloff: This raises a very important problem that needs a great deal of discussion. It would be my opinion that the patient who has had multiple emboli—I was thinking in terms of this before—very probably has acute carditis, and is not at that particular time a candidate for mitral valvotomy. We would carry the patient through and make absolutely certain that we were not dealing with acute rheumatic carditis before we would proceed with a valvotomy. However, if he had had a simple stroke, a simple cerebral embolus, we would not hesitate to go ahead with a mitral valvotomy during his stay in the hospital.

Moderator Sprague: One other question. What are the indications for prophylactic digitalization, that is, prior to surgery?

Dr. Soloff: This morning I stated that if an old person has short breath and we are uncertain of the cause of the shortness of breath, it is safe to assume that there might be some underlying heart failure and, therefore, I would proceed with complete digitalization. If in any patient who has heart disease and faints, we are uncertain on the basis of clinical grounds whether there may or may...
not be some incipient heart failure and whether the stress of surgery may be too great, I would proceed with digitalization.

I think that the objection to digitalis before operation is based mostly on work done many years ago in which it was suggested that the cardiac output was decreased if the patient who had a normal heart was digitalized. But the patient who has had heart disease, whether he has had heart failure or not, if subjected to a stressful situation, such as an operation, is not a normal patient and does not have a normal heart. The possibilities of doing harm by digitalizing the patient I think are so slim that at least we have never been able to recognize harm from it. So I would be more on the active side of digitalizing any patient when there was a possibility that either some heart failure was present or the stress of the operation would be unusually great.

Moderator Sprague: Dr. Wolf, is the bradycardia often seen in acute myocardial infarction a vagal reaction to fear?

Dr. Stewart G. Wolf, Jr.: I think it would be awfully difficult to answer that question. I think it is important to realize that this is a way in which bradycardia can be brought about. The cardiovascular structures are capable of responding to a variety of stimuli, among which are stimuli that emanate from the interpretive areas of the brain.

Moderator Sprague: Can you explain atrial fibrillation that occurs in sleep, in patients in whom no organic disease exists?

Dr. Wolf: Yes, I think it is very important to realize that the connection is in the interpretive areas of the brain that make a significant experience an effective stimulus to put into operation some bodily mechanisms that do not require consciousness to operate. They do not require consciousness of motion and neither do they require an awareness of a conflict situation; therefore periods of sleep are just as good for the production of these bodily changes in responsive circumstances as waking periods. As a matter of fact, they may be somewhat better, and certainly in studies that have been done of patients with peptic ulcers, the hypersecretion of the stomach is often much more vigorous during the night hours than it is during the day.

Moderator Sprague: What about dreams?

Dr. Wolf: Dreams would be another way of showing how the individual responds to meaningful events when he is not actually aware of what is going on.

Moderator Sprague: Dr. Sokolow, in atrial fibrillation before conversion of the embolism? How long after beginning the anticoagulant do you attempt conversion?

Dr. Sokolow: If the patient has no history of peripheral embolism, as a rule we do not give anticoagulants prior to therapy. We have not run into very much difficulty with this technic. If the patient has had a recent embolus, we use anticoagulant therapy first, on the assumption that the recent embolus indicates a recent thrombus which may discharge particles of remnants.

Moderator Sprague: How often have you had this happen in your experience?

Dr. Sokolow: It has happened to me quite rarely. I think we have seen certainly not more than perhaps 6 cases of systemic emboli following conversion in approximately 200 conversion attempts. So that in our own experience it is an instance of approximately one half of 1 per cent of systemic emboli.

Moderator Sprague: I mean that this business of arrhythmias can be a real emergency. Would you say that you now attempt to convert patients with more serious cardiac disease than you used to? In other words, are you less fearful in converting sick patients than you used to be?

Dr. Sokolow: I have considerable respect for quinidine and I think that before I would attempt to convert a patient, I would try to get the patient in the best possible condition and to arrive at a judgment that sinus rhythm would be better for him. I would say that we have a better awareness of the patient who is less likely to be converted. Particularly in the individual with rheumatic heart disease, with mitral insufficiency, who has a
large heart, who is in chronic failure, who has had atrial fibrillation for more than a year, we know that the batting average is not more than 40 per cent even though quinidine is given to toxic levels.

On the other hand, if an individual has had coronary heart disease or hypertensive heart disease our success with conversion is sufficiently high, over 80 per cent in patients whose fibrillation is more than a year—better than 90 per cent if the fibrillation is less than 1 year—that we would attempt conversion in most patients. So that we try to distinguish those patients in whom the likelihood of conversion is good and those patients in whom the likelihood of conversion is poor.

MODERATOR SPRAGUE: Following valve surgery do you always try to convert them?

DR. SOKOLOW: I would say practically always, if the surgeon has been able technically to perform a good mitral valvotomy and the patient is still fibrillating, and certainly every patient who had sinus rhythm prior to mitral valvotomy we try to convert.

MODERATOR SPRAGUE: Is procaine amide as effective when given diluted as when it is undiluted?

DR. SOKOLOW: It is probably less effective when diluted but it is safer. We have had patients who failed to convert when the drug was given very slowly, diluted, say, 25 mg. a minute, who succeeded in having the rhythm converted when the injection was sped up. So that one has to weigh the increased toxicity on giving rapid injections against the decrease in likelihood of success if it is given too slowly.

MODERATOR SPRAGUE: Dr. Metcalfe, what is your experience concerning pregnancies following apparently successful treatment of subacute bacterial endocarditis?

DR. METCALFE: Well, it is not extensive but it is successful. It is uniformly successful. Just off the top of my head, as they say, I would guess that perhaps there have been 10 or 15 of these people who have gone through one or more pregnancies. Usually they have an aortic valve disease or mitral insufficiency, and our experience and success with these valve lesions is considerably better than it is with mitral stenosis. So that I would think that the fact of having subacute bacterial endocarditis would not alter my judgment. What would alter my judgment would be the valve lesions and the functional state of the heart's operation as a result of rheumatic heart disease.

MODERATOR SPRAGUE: I am a little surprised at the good success with aortic valve disease following subacute aortic endocarditis. I would like to know about the panels' experience with this. Are you getting into all sorts of trouble with those who had an ulcerative process in the aortic valves that were "cured" and then developed rapid congestive failure?

DR. SOKOLOW: Of course, I have had the same experience that you are referring to; that is, the patient is cured of bacterial endocarditis and then within the succeeding year develops ventricular failure from increasing aortic insufficiency; but that does not always happen.

MODERATOR SPRAGUE: Does any other member of the panel want to comment on that?

DR. GASUL: Our surgeon began operating on these about 21/2 years ago. I believe that so far he has had about 18 patients. I believe that about 12 of them are alive, but that does not mean that they do not have any evidences of aortic stenosis. Until now he has used the closed method in performing the operation. Starting this coming week he is going to use open cardiotomy for aortic stenosis.

I hesitate to advise an operation on a child with an aortic stenosis unless he is practically in heart failure. We have a patient right now whose diastolic pressure in the left ventricle goes up to 50 and he is going to be operated upon, but unless open cardiotomy is done, I hesitate very much to advise an operation on a child with a congenital aortic stenosis. Most of the cases that we have had, following a successful operation, have evidences of aortic regurgitation.

We have not followed them up long enough—as far as I know, nobody else has followed
them up long enough because the operation is not older than 4 or 5 years—to know how dangerous this aortic regurgitation is. We know of patients who developed pulmonary regurgitation following pulmonary valvulotomy. They got along very well, but there the pressure differential is very different from the development of aortic regurgitation after an operation for aortic stenosis.

MODERATOR SPRAGUE: Dr. Soloff, have you any comment on this?

DR. SOLOFF: No, we do not treat children that way.

MODERATOR SPRAGUE: I might say that a real indication of emergency in aortic stenosis in children in our experience has been the appearance of the inversion of the T wave in the lateral precordial leads. I think that the Children’s Hospital experience shows that when that occurs as evidence of left ventricular difficulty, these children are likely to die suddenly.

DR. GASUL: May I disagree with you on that? I think we have quite a number of children who have definite electrocardiographic evidences of left ventricular hypertrophy with inverted T waves, who are acting just like any other normal children, who with left heart catheterization do not necessarily show a large gradient between the left ventricle and the aorta, and we do not advise an operation on these patients.

MODERATOR SPRAGUE: This is a question concerning another real emergency, addressed both Dr. Sokolow and Dr. Soloff. How often have you seen bilateral adrenal hemorrhage alone during anticoagulant therapy?

DR. SOLOFF: I have never seen it as a sole cardiologic finding.

DR. SOKOLOWS: Nor have I.

MODERATOR SPRAGUE: Dr. Sokolow, in giving oxygen to emphysema patients would it be wise to give carbon dioxide with it?

DR. SOKOLOWS: Carbon dioxide does not stimulate the respiratory center in patients with emphysema and carbon dioxide retention because apparently the respiratory center no longer responds to increased pressure of carbon dioxide in the blood, but only responds peripherally as a result of oxygen lack. Therefore, in patients with emphysema and carbon dioxide retention giving carbon dioxide does not increase ventilation.

MODERATOR SPRAGUE: Would you tell us what drugs are contraindicated in ventricular tachycardia and fibrillation?

DR. SOKOLOWS: Well, taking the last part first, I do not know which drugs are indicated in ventricular fibrillation. There are no drugs that affect it either one way or the other except for perhaps electric shock to produce cardiac standstill and then to use a different type of cardiac shock to get the heart beating again.

In ventricular tachycardia, it has usually been stated that digitalis is contraindicated because it may produce cardiac irritability and result in ventricular fibrillation. Actually the data to support this statement are pretty scanty, and most of us who have had experience in dealing with patients with ventricular tachycardia, that failed to respond to all other measures and have had cardiac failure, have found that actually digitalis reverses it to the sinus rhythm. So I am not certain in my own mind that digitalis is contraindicated in ventricular tachycardia in spite of the statements to the contrary.

MODERATOR SPRAGUE: Does anyone else on the panel want to comment on this?

DR. SOLOFF: I would like to emphasize what Dr. Sokolow has said. I think it is a very important point because, as Dr. Gasul said this morning, it is some times very difficult to tell the difference between a supraventricular tachycardia with intraventricular block and ventricular tachycardia, and therefore it is important for us to know that the patient has not received digitalis. Digitalis is not necessarily contraindicated under certain circumstances. I would go along with Dr. Sokolow, and I have used drugs I believe successfully in this situation.

MODERATOR SPRAGUE: Yes, I think that is a very important point.

I am going to ask Dr. Wolf to tell us in more detail about serum lipid changes with stress.
Dr. Wolf: This is not strictly relevant, I think, to the question of emergency, and so I will be very brief. Information at the present time on this subject is fragmentary. A paper is going to be on the program early in the week that I am sure will bring the story further along. But at the moment I think the evidence is good enough to say that the mechanisms that regulate the level of lipids in the blood are just other bodily mechanisms that are connected with and capable of responding to impulses reaching them from the interpretive areas of the brain.

In our own experience when elevations of the serum cholesterol have occurred in association with stressful circumstances in the lives of individuals who have had well documented coronary occlusions, the lipid protein patterns have indicated that the cholesterol increase is in the beta area rather than in the alpha area. These data are still preliminary, however, and I think it would be a mistake to try to draw too many conclusions from them.

Moderator Sprague: Dr. Sokolow, do you agree that a hypotensive reaction due to nitroglycerin is a rare event, considering that many patients take and tolerate up to 40 tablets daily? I have one now taking 150 tablets a day.

Dr. Sokolow: How one defines rare and infrequent I do not know. In the past year I have seen 2 or 3 patients whose pressure fell considerably. The one that I referred to this morning was a patient who would get nauseated and green and his cardiac pain would increase every time he took nitroglycerin; and we observed that his blood pressure fell to a systolic of 70 even in the sitting position after only 1/200 gr. I quoted Russek, who has particularly studied this problem and found that approximately 10 per cent of the patients have adverse reaction. My own experience has been that.

Dr. Soloff: I think it is important, since we have a drug as good as nitroglycerin, that we should not fear to use it unless we are absolutely certain that it has a bad effect. It is a very rare person who will develop symptoms due to hypotension when he is taking nitroglycerin, but it is very easy to recognize that patient and to be sure that he is not standing but is sitting down at the time. So I personally think that patients who have angina pectoris should be surveyed and told to take it even though there is some evidence that it may lower blood pressure in the standing position.

Moderator Sprague: I am going to agree with both of these gentlemen. However, I did write a paper once on nitroglycerin collapse because I was impressed with the rather frightening and true emergency situation of a patient who is really sensitive to nitroglycerin the first time it is tried. I know of a young man just starting in practice in the last year, and about the first patient he had in his office was a person with angina to whom he gave nitroglycerin in the office. The patient collapsed and proceeded to have such a low blood pressure that it undoubtedly promoted a cardiac infarct at that time.

On the other hand, we have treated patients in acute stages of myocardial infarction, one a colleague of ours with heart block, with repeated doses of nitroglycerin with apparent beneficial effect. So that again people differ.

I think I agree with you, Dr. Soloff, that one of our great troubles is in convincing the patient to use enough prevention because of the knowledge that nitroglycerin is related to dynamite and the idea that it is going to lose its effectiveness, and that a patient who takes this powerful medicine is about done for anyway. I really try to sell them on nitroglycerin more than I do to take it away from them.

Dr. Metcalfe, here is a question about embolic complications associated with rheumatic heart disease occurring occasionally in pregnancy. How would you treat these and what management would you use to prevent recurrence?

Dr. Metcalfe: I gather that this question is specifically concerned with anticoagulation, and fortunately our experience with this as a complication of pregnancy is very small.
However, on a couple of occasions we have used heparin and Demerol after embolic episodes during pregnancy.

There is some animal work that gives a very bad picture of the use of Demerol because of its effect on the fetus. This was used in rather large doses, and the only reason for not saying the same thing about the anticoagulant qualities of the other drugs is that not enough experimental work has been done on them. This is clearly a toxic drug, and if you give enough of it you will harm the fetus.

In the 2 instances that I referred to, which constitute the sum total of my experience, we brought the patients to the hospital 10 days before their expected day of confinement, reversed the effect of Demerol with KIO4 oxide and crossed our fingers until 24 hours after delivery when we reinstated it. I am not sure that this is good but it worked.

MODERATOR SPRAGUE: Dr. Wolf, is it possible that many unstable electrocardiograms may be explained on the basis of emotional stress?

Dr. Wolf: I think it is important to realize 2 or 3 things here. The first is that there are certain changes in the electrocardiogram that can be brought about under circumstances of emotional stress. There are a good many that are not. The T-wave changes are particularly those that may occur under these circumstances.

Again I would like to emphasize that this simply means that the heart is capable of reacting to impulses emanating from the interpretive areas of the brain, so that the contribution of the significance of the event to one's clinical and laboratory findings must be taken into account.

MODERATOR SPRAGUE: I think that many of you know the experiments that Dr. Graybiel performed during World War II in coming silently up behind a healthy young man and firing off a gun behind his ear, when he was connected with the electrocardiograph. The patients developed intraventricular block, T-wave inversions, and so on, and certainly this must have come from the soul somehow or other.

DR. WOLF: It is attributed to the direct effect of sound.

MODERATOR SPRAGUE: There were no bullets.

Dr. Sokolow, have you noted hypotension or tachycardia with the use of Demerol before and after operation?

DR. SOLOFF: Yes, I think this does occur, particularly in older people, in 2 situations: in some older people who have had preoperative sedation that might be more than they needed, and in patients with mitral stenosis preoperatively for mitral valvulotomy. In the latter instance, for example, because of diminished cardiac output the peripheral arterial vasodilation is compensatory, and when that is removed by a drug such as Demerol, the blood pressure may fall precipitously and be the primary reason these patients may develop ventricular fibrillation, some times after the Demerol and some times slightly later after induction of the Pentothal.

In older people who have had barbiturates and Demerol, the pressure may fall, and on more than one occasion we have seen such patients and had the surgery delayed or even stopped because of the hypotension that resulted. It is for this reason that many anesthesiologists prefer not to give Demerol preoperatively by the usual subcutaneous method, but to give it very slowly in very small amounts intravenously with a very small needle so as to titrate the amount that the patient needs. Furthermore, they prefer not to give hypnotic drugs prior to the use of Demerol unless the patient requires it.

MODERATOR SPRAGUE: Dr. Soloff, will you comment on the antidiuretic effects of sedatives.

Dr. Soloff: I think that is a very important subject, particularly when we are dealing with men in the upper age group. Demerol, codeine, and morphine very commonly produce symptoms that can mimic or perhaps actually precipitate prostatic obstruction. Whenever we get a patient who apparently has developed prostatism, we always ask whether he has had any one of the other
sedatives, which in a lesser degree can actually suppress the flow of urine.

The prostatic type of manifestation may also occur in my experience after antispasmodic drugs, such as Banthine.

So I think that is a most important problem, and when we are dealing with very intractable heart failure I always go into the problem of this type of sedative as to the possible cause for suppression of urination.

**Moderator Sprague:** I am glad to have you emphasize that because it can certainly create an emergency or an intractable situation, if you do not understand that this therapy is responsible.

There is another question here for you, Dr. Soloff. It has been said that the effective and toxic doses of digitalis may change after surgery. What criteria do you use in sinus rhythm in questionable cases to decide whether the level of digitalization is proper, too low, or too high?

**Dr. Soloff:** That is a very difficult question to answer because we, unfortunately, do not have any criteria to judge digitalization in the patient with normal sinus rhythm except for the critical condition of the patient. If the patient's heart beat is inordinately fast for his temperature after operation, one should, of course, be on the alert for complications. Pulmonary embolism, for instance, may increase the heart rate. If we do not find any acute reason for the rise in the heart rate, then I would be inclined to use a short acting digitalis preparation very slowly. If that is done, one must very carefully watch the patient several times a day for possible overdigitalization. There are 2 things one would look for. For instance, in the conscious patient any impairment of appetite, a patient whose appetite is diminished for any nonapparent reason. The second is the appearance of premature beats. Either of these situations would make me feel that my patient has had too much digitalis and that I have increased it above the average maintenance dose that the patient needs. But it is a trial-and-error method and means very careful observation of the patient several times each day.

**Moderator Sprague:** Dr. Gasul, someone thinks that in the treatment of rheumatic fever you stress the use of steroids contrary to the American Heart report, as to the value of salicylates versus steroids. Will you clarify that?

**Dr. Gasul:** I believe that it is generally accepted now-a-days that that experiment was deficient in some ways; that is, not a sufficient amount of steroid was given, no definite distinction was made between acute carditis and chronic carditis, and it was not given for a long enough period of time. Since the termination of that experiment a number of papers have appeared in the literature emphasizing the fact that if one excludes children with chronic rheumatic carditis—I believe that almost all of us will agree that if a child comes in with a chronic carditis, the steroids are not going to change that situation—if you limit yourself to acute carditis, if you start the treatment early enough, if you give sufficient amounts of steroids for a long enough time, particularly in the severely ill patients, I believe the generally accepted opinion now-a-days—and this includes our experience, too—is that the steroids are superior to the salicylates.

**Moderator Sprague:** Do I hear any dissenters among the panel? I do not dissent.

At the first Cornell conference on therapy, published in 1946, you remember that Dr. Harry Gold examined the bags of seven doctors to see what drugs they carried for emergency use. I thought that 10 years later it might be of value to see what these drugs are and I would ask this panel, if they were now packing their saddle bags and getting on their horses to go out and treat the public, without the great advantages of our teaching hospitals, what they would put into them or if they would change these at all.

Thirty drugs were selected by Dr. Gold from 7 bags, and 12 of these were for cardiac emergencies. I will read the names of the drugs and then I will ask the panel what they would add or subtract from this group: Syrup of ipecac, morphia, digitaline nati-velle tablets, onabain, mechoyl, atropine sulfate, quinidine tablets, quinine dihydrochloride for intravenous use, mercurhydrin, epine-
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was fact, most controlled which would personality but fast intravenous would is important, penicillin. That is, safe, nice, safe treatment of the dangers of the other therapies. The ipecac, as you know, is returned quantitatively. We have a short time to ask the panel to add any drugs to the bag.

Dr. Sokolow, have you any pet drugs that you would like to add to this list that I read?

Dr. Sokolow: Well, I would change a few with the same basic principles. I think that I would use one of the rapidly acting glycosides, such as digoxin, in lieu of the digitalis, because it is faster acting, and possibly eliminate ouabain. Instead of using quinine dihydrochloride I would substitute quinine gluconate in its place. Also Mecholyl is used so infrequently I think that it could be eliminated.

Dr. Metcalfe: I am surprised that the antibiotics are missing as a group. I suppose that if I had to carry one, I would carry penicillin.

Dr. Soloff: I just don’t own a bag, but I would mention that the 3 drugs that are important, as I see them, are morphine, a very fast intravenous digitalis preparation and I would agree with Dr. Sokolow that digoxin is probably as good as any, and also penicillin which Dr. Metcalfe suggested. I think that most of the other cardiac emergencies can be controlled by means of morphine alone.

Moderator Sprague: Dr. Wolf, would you add anything to the bag?

Dr. Wolf: I think the doctor’s placebo personality has to go along in the emergency, but I would agree with the other changes that have been made.

Moderator Sprague: Dr. Gasul, would you add anything?

Dr. Gasul: Did you mention one of the most important ones—aspirin?

Moderator Sprague: No. As a matter of fact, it was in the bag, but I didn’t think it was put in as an emergency drug.

Dr. Gasul: I would take aspirin and digitalis.

Moderator Sprague: Dr. Metcalfe, what is your objection to applying the generally accepted criteria for selection of cases for mitral commissurotomy during the first and second trimester and avoid their slipping into class IV?

Dr. Metcalfe: You know this is sort of like “Have you stopped beating your wife.” This is a matter of judgment and of opinion, but our experience with mitral valvotomy in pregnancy is as follows: We have had 3 deaths in 300 pregnancies in women with rheumatic heart disease, without doing a single mitral valvotomy. We have had 4 deaths in 14 women who were subjected to mitral valvotomy during pregnancy. Now there is no question that the 14 worst ones might have been picked. All I can tell you is that we did not pick any of them. We have never recommended this procedure during pregnancy. Now part of this is probably a Boston obstinacy and part of it is a conviction that a pregnant woman can get through pregnancy with good medical management. Somebody has to take that stand and I am prepared to defend it.

Another discouraging thing is that we have had 4 maternal deaths in the state of Massachusetts of women who have had successful mitral valvotomy according to the fellows who advised them and supervised them afterwards. They died in the next pregnancy. So that I do not regard mitral valvotomy as an answer to mitral stenosis in that or succeeding pregnancies. I guess that is all I want to say.

Dr. Soloff: I think that that question should be answered in the way that Dr. Metcalfe did but with an addition. First of all the assumption is being made that mitral valvotomy carries a higher risk in pregnancy than it does in a nonpregnant state. At least in our experience that is not true. In other words, it does not make any difference whether the patient is pregnant or not, the morbidity and the mortality experience is about the same. The answer is, as Dr. Metcalfe put it, however, mainly that our patients do not die during pregnancy with mitral stenosis. We have not had the experience that Dr. Metcalfe
has had, but we have had no deaths in our obstetrical ward ever since Dr. Robert Wilson took over as professor of obstetrics about 9 or 10 years ago. So we know no reason for going ahead with mitral valvotomy during pregnancy.

The other important thing is that when you do get a death in pregnancy, if you should have one with rheumatic heart disease, it is important to ask yourself at what stage of pregnancy did it occur and why. Dr. Metcalfe showed this morning that the extra load of pregnancy occurs somewhere around the eighth month. If you are to attribute the death to mitral stenosis itself and not pregnancy, one should expect death at that time, but patients with rheumatic heart disease and mitral stenosis do not die at that time. If there is any death at all that I have been able to find in the recent literature, it is mostly in the immediate postoperative period. In other words, about 12 to 14 hours after the delivery. So I think that there is perhaps, with extremely rare exception, no indication for mitral valvotomy during pregnancy.

Dr. Metcalfe: I want to make this point. The success of the pregnancy after mitral valvotomy, either during the pregnancy or the year before, does not mean that the mitral valvotomy was indicated or successful.

Moderator Sprague: The final question as we end this discussion once again goes to Dr. Wolf. Every time a prominent national or local person dies of a heart attack, many anxious heart patients visit the office. Is it not important that their electrocardiograms are normal even with anxiety?

Dr. Wolf: Yes, I think it is important, but I think that we should be alert to the possibility that the changes we might detect at this time might be transitory changes associated with anxiety.


The authors present the results of studies in man of early aortic atherosclerotic lesions. Structures with the general form of capillaries were demonstrated in the superficial layers of the intima by use of the alkaline phosphatase staining technique for endothelium. In 1 instance, red blood cells were found within a structure that showed enzyme activity. These vascular structures were interpreted as representing an extremely early feature of the disease. The foam-cell accumulations, the fibrous-tissue proliferation, and the deposition of hemosiderin, which are all found in the earliest atherosclerotic lesions, may be explained by recurrent hemorrhages from the rupture of “high pressure” intimal capillaries.
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