PANEL DISCUSSION

What's New in Arrhythmias?

The Panel was comprised of the following members: HERRMAN L. BLUMGART, Boston, Mass., Moderator; W. Proctor Harvey, Washington, D.C.; R. Bruce Logue, Atlanta, Georgia; Maurice Sokolow, San Francisco, Calif.; and E. Grey Dimond, Kansas City, Kans. The Panel Discussion was conducted at the Thirty-first Scientific Sessions of the American Heart Association held at San Francisco, Calif., on Friday, October 24, 1959.

MODERATOR HERRMAN L. BLUMGART: I turned the title of this panel discussion into a question which I asked each of the panelists. Their replies formed a list of 27 subjects. Some were about new agents or new methods of treatment, others had to do with new types of evidence concerning old problems. The first subject will be on "Multiple Sounds in Ventricular Tachycardia—A New Auscultatory Aid in the Bedside Diagnosis," by Dr. Harvey.

DR. W. PROCTOR HARVEY: I am sure that if most of us looked back on cases of ventricular tachycardia that we have observed, we would remember that they often sounded different from other types of rapid heart action. The reason for this difference is the frequent occurrence of multiple low-frequency sounds, sometimes 5 or 6 in a single cardiac cycle. The mechanism of these multiple sounds appears to be related to wide splitting of the first and second heart sounds plus gallop sounds (atrial and ventricular). A patient having these multiple sounds with ventricular tachycardia is shown in figure 1. After reversion to normal sinus rhythm with quinidine these sounds were absent. On listening specifically for these multiple sounds other cases were found, and on reviewing other cases that we had previously recorded, similar findings concerning the presence of these sounds were found. Figure 2 shows 5 examples of ventricular tachycardia, all illustrating these multiple sounds. The auscultatory clues suggesting ventricular tachycardia previously emphasized are a changing intensity of the first heart sound, slightly irregular ventricular rate, and lack of slowing of the ventricular rate with carotid sinus stimulation. In addition to these important findings, which can be observed on auscultation at the bedside, the hearing of multiple low-frequency sounds can aid in even more rapid suspicion of the diagnosis of ventricular tachycardia. Figure 3 shows 13 examples of tachycardia other than ventricular tachycardia, in which these multiple sounds are not present.

As to the mechanism of these multiple sounds, further light can be shed on the problem by the observation of splitting of heart sounds with right or left bundle-branch block in the presence of congestive failure. In bundle-branch block the second heart sound is generally widely split, and frequently the first heart sound also. If the patient has congestive failure a ventricular diastolic gallop is usually present, and if first-degree heart block (or P-R interval on the longer side) or hypertension is present, atrial sounds may be heard. Figure 4 represents a patient with left bundle-branch block and cardiac decompensation, illustrating the wide splitting of the first and second heart sounds plus both atrial and ventricular diastolic gallops.
WHAT'S NEW IN ARRHYTHMIAS?

Fig. 1. Male, age 58, several weeks after acute myocardial infarction with ventricular tachycardia (upper tracing). Had multiple sounds in addition to changing intensity of first sound (S1) which were not present immediately after reversion (lower tracing).

Our hospital staff has been on the alert for this additional clue to the presence of ventricular tachycardia, and as illustrated by the patient in figure 5, may lead to more prompt recognition and treatment. This 52-year-old man, having pressure in the precordial area of several hours' duration, arrived at the hospital pale, anxious, and with no obtainable blood pressure. Auscultation of the heart revealed a rapid rate of 188. An immediate clue was the presence of multiple low-frequency sounds heard along the lower left sternal border and apex, in addition to a changing intensity of the first heart sound. Slight irregularity of the ventricular rate was present, and carotid sinus pressure produced no slowing. On the basis of these findings procaine amide was immediately ordered to be held in readiness until the electrocardiogram confirmed the diagnosis of ventricular tachycardia. Reversion to normal rhythm occurred after 450 mg. of intravenous procaine amide.

Not every patient with ventricular tachycardia will have these multiple sounds. They are most likely to be present in patients with ventricular tachycardia of longer duration, with wide spread of the QRS complexes, and having congestive failure.

Fig. 2. Tracings of 5 patients, each having ventricular tachycardia. Note multiple low-frequency sounds and changing intensity of first sound (S1). Lower tracing taken from tape recording.

DR. BLUMGART: Are there any further comments by any of the other panelists?

DR. R. BRUCE LOGUE: I'd like to ask Dr. Harvey if he has ever seen multiple sounds with atrial flutter?

DR. BLUMGART: May I also add to that and make it one package—paroxysmal atrial tachycardia with block?

DR. HARVEY: Yes, we have heard atrial sounds with both atrial flutter and paroxysmal atrial tachycardia with block. These would naturally come up as part of the differential diagnosis, but we utilize the other clinical features; carotid sinus pressure will slow the paroxysmal atrial tachycardia with block, or flutter, and have no effect on ventricular tachycardia.

DR. BLUMGART: The second subject is one that has played an important part in medical literature for some decades. Dr. Sokolow has consented to deal with it, that is to say, 'Present Status of Conversion of Chronic Atrial Fibrillation to Sinus Rhythm.'
Dr. Maurice Sokolow: One of the most important things that is new about chronic atrial fibrillation is the appreciation of the fact that atrial fibrillation is physiologically undesirable, even though we have seen many elderly individuals who developed atrial fibrillation and who tolerated the arrhythmia reasonably well. Some recent work has shown that with the development of atrial fibrillation, particularly when associated with cardiac failure, the cardiac output fell considerably; when sinus rhythm was restored the cardiac output increased about 30 per cent. Contraction of the atria are important for ventricular filling and for normal cardiac output, particularly in patients who have failure, and therefore individuals who have chronic fibrillation may have progressive enlargement of the heart and may develop chronic cardiac failure.

The influence of atrial fibrillation on the incidence of systemic emboli is well known. There are considerable data in the literature on the point, and perhaps we need not elaborate on it. Patients with atrial fibrillation may be quite uncomfortable, even though they are not in failure, because they may have a rise in ventricular rate that may reach 150 to 160 for the first few seconds or minutes after exercise; this rise in ventricular rate with exercise may occur even though the patient has been digitalized to the point of toxicity. Dr. Blumgart, I think, in the twenties, was one of the first to show this, and recent work has confirmed it. Even though patients have ventricular rates at
rest that are in the range of say 70 to 80, and one would think they were perfectly satisfactorily controlled, their ventricular rate rises considerably with effort. The increased ventricular rate after effort may persist longer in patients who are fibrillating than in patients with sinus rhythm. These patients complained of palpitations, and they are uncomfortable. Because of these 3 difficulties (decreased cardiac output, emboli, and palpitations) we believe that patients with atrial fibrillation are better off if the rhythm can be converted to normal.

If this is true, do we attempt to convert all patients who have chronic atrial fibrillation? The answer is no; primarily because of the fact that conversion of atrial fibrillation with quinidine, which is the only potent drug that can be used for this purpose, is not without hazard. The 2 major hazards are, first, the likelihood of ventricular arrhythmias, rarely, ventricular fibrillation; and secondly, the fact that in a very small percentage of patients, perhaps 0.5 per cent, a systemic embolus may be released following conversion. We have a healthy respect for quinidine, and although we believe that patients are better off with sinus rhythm, we are not unmindful of the fact that whenever we attempt conversion we are accepting a certain risk. Elderly patients, then, who have had chronic atrial fibrillation for some years, who are tolerating the arrhythmia well, and who have no complaints, are usually maintained on digitalis; we usually do not attempt to convert them with quinidine.

The group of individuals whom we attempt to convert can be listed in the following: First, those individuals with fibrillation of short duration, less than 6 months, because of the fact that this group is relatively easily converted and the complications are few; individuals who continue to fibrillate following adequate treatment for thyrotoxicosis usually convert easily, and therefore we attempt conversion in these patients; patients who have had a technically successful mitral valvulotomy and still fibrillate, or who previously had sinus rhythm and then fibrillate postoperatively, usually are given quinidine after a period of 2 weeks or so when everything has stabilized; patients whose ventricular rate is uncontrolled while they are fibrillating, despite full doses of digitalis. These patients may have low-grade, chronic failure, or they may have severe palpitations, and in these patients following conversion to sinus rhythm the ventricular rate may be slow and the patients considerably improved. Lastly, we attempt conversion in patients who have cardiac failure which is not improved despite full treatment of the usual variety. Sometimes, converting to sinus rhythm will be the critical procedure that will relieve the symptoms and signs of cardiac failure.

One should be aware of the patients in whom the most favorable results can be anticipated. In our experience, patients whose fibrillation is less than a year's duration, and particularly less than 6 months' duration, those who have no cardiac failure or considerable cardiac enlargement, and those who do not have mitral insufficiency, usually have the greatest possibility of success, and roughly 85 per cent of these patients will convert with quinidine.

It is important to emphasize that before patients are given quinidine for conversion they should be hospitalized, cardiac failure should be treated, they should be digitalized.
so that the ventricular rate is slow, and any complication, infection, or nutritional disturbance should be managed first. Quinidine may then be given in moderate doses with close clinical, electrocardiographic, and serum quinidine concentration observations. The dose may be gradually increased, depending upon the tolerance of the patient. We believe that if a patient does not convert following 3 Gm. of quinidine a day, or with a serum concentration of 8 mg. per ml., then the entire situation should be reviewed and a new decision made. One should not use larger doses unless he has had considerable experience with the drug, or unless the clinical condition is sufficiently urgent to warrant a more heroic attempt.

With the careful use of quinidine, and in some instances the combination of quinidine with procaine amide, the majority of patients with chronic atrial fibrillation, who do not have severe failure or fibrillation of long standing, can be converted to sinus rhythm.

I would like to emphasize that we believe the use of quinidine should be likened to an elective surgical procedure, and that the drug should be given only after considerable thought and awareness of its toxicity. It is a very valuable drug, and one must compare the potential therapeutic benefits with the possible risks in deciding whether or not to use it.

One additional point—when moderate doses of quinidine are used, and the patient does not convert, the addition of procaine amide, which by itself is not very effective in producing sinus rhythm, may be successful.

Dr. Blumgart: Would any of the panel like to ask any questions or make any additional comments on this?

Dr. Logue: Dr. Sokolow, at what level of QRS change will you abandon the administration of quinidine?

Dr. Sokolow: We believe that moderate widening of the QRS complex that occurs with quinidine is not a toxic manifestation, but is the result of the therapeutic effect of the drug. We usually continue quinidine until the QRS interval exceeds 50 per cent of control value, although we use particular caution when widening exceeds 25 per cent. We arbitrarily stop quinidine at 50 per cent QRS widening, because of the possibility that when QRS widens further that cardiac standstill may occur. In actual practice, we have never seen this occur. We have seen the QRS interval, for example, widen to as long as 0.2 second without the patient’s being aware of any particular disturbance; but it has disturbed us.

Dr. Logue: I should like to ask Dr. Sokolow, also, do you treat those with bundle-branch block?
WHAT'S NEW IN ARRHYTHMIAS?

DR. SOKOLOW: We are very hesitant to use quinidine in patients with bundle-branch block, and our own experience on this is quite limited. Dr. Mervin Goldman of the Veterans Hospital in Oakland, however, has had very good results using quinidine in bundle-branch block and his incidence of toxicity was no greater than that of those who used it in the absence of bundle-branch block. It is possible that the old adage regarding bundle-branch block as a contraindication is unjustified and perhaps should be studied further. I would be considerably more cautious in using quinidine in the presence of bundle-branch block, but if the clinical indication was clear, the block would not deter me.

DR. HARVEY: I would like to mention a couple of "tricks" that have helped us in alleviating the incidence of the toxic gastrointestinal side effects from quinidine. The administration of the drug after meals has helped reduce nausea, and if diarrhea is a problem, Amphojel, probably because of its constipating effects, has been useful (15 to 20 ml. between meals).

DR. BLUMGART: We will go on to the next subject, which is a very important one. I think that the problems we face in this day and age of powerful drugs, which seriously affect electrolytes and the heart itself, are great. As a matter of fact the first paper this afternoon mentioned some of the arrhythmias that occur with chlorothiazide. It is appropriate then that Dr. Dimond will discuss the arrhythmias that occur with various medications such as veratrum, cortisone, hydralazine, reserpine, and so forth.

DR. E. GREY DIMOND: I would like to comment on the subject of mitral valvulotomy and arrhythmias. Here certainly is a circumstance in which a new arrhythmia has occurred. An individual with presently a normal sinus rhythm has a mitral valvulotomy and in the immediate postoperative period has atrial fibrillation. This has been a widely reported observation. Something like one third of the subjects who have normal sinus rhythm prior to mitral valvulotomy develop atrial fibrillation on the first day, sixth day, or ninth day, for example, and in spite of intensive therapy, at least half of them will not revert to normal sinus rhythm. This has been our experience also. In other words, one sixth of all persons who have a normal sinus rhythm prior to a mitral valvulotomy will end with chronic atrial fibrillation. Attempts at conversion during the immediate postoperative period have definite drawbacks and in general it is better to wait a matter of months before trying for conversion.

Other things have occurred in recent years that actually have produced arrhythmias that really did not exist before; not new arrhythmias, but new causes. Hydralazine certainly does produce persistent tachycardia. On the other hand, reserpine produces a bradycardia which, with its slowness and the increased stroke volume of each pulse, often produces a concerned and heart-conscious patient.

Hypothermia, in the immediate postoperative period, presents frequently an arrhythmia due to the dislocation of the cardiac pacemaker. We had 10 consecutive cases of atrial septal repair under hypothermia, and in 8 of these, in the first few days following the chilling episode, there was a shift of the pacemaker into the atrioventricular node.

Still another example of iatrogenic arrhythmia is being seen in the elderly patient. In the well-digitalized patient the addition of compression dressings to the eye may result in excessive bradycardia and even in periods of cardiac standstill.

In the postabsorption period, after a meal rich in glucose, a heavily digitalized patient may actually develop arrhythmias indicating digitalis intoxication. The movement of potassium into the cells and depletion of readily available potassium at the cell membrane are perhaps the provoking causes.

Cortisone in large doses has a tendency to shorten the P-R interval and if potassium loss is high, ventricular arrhythmias may result.
Finally, the Brock procedure, the procedure in which the right ventricle is actually transected and entered, may well alleviate the cause for the right heart overload, but a surprising percentage of these individuals end with right bundle-branch block.

As you look back over the comments I have made, one must note how each new step forward has in turn created its own new hazard.

**Dr. Blumgart:** Are there any comments or further questions?

**Dr. Sokolow:** I think Dr. Dimond’s comment that it is unwise to try to convert an arrhythmia in the acute postoperative state is very important. It has been our practice to delay conversion until the patient has convalesced from the mitral valvulotomy and has been stabilized with respect to his activity, diet, nutrition, medication, and so on. Some patients who become toxic with quinidine, if given on the second or third postoperative day, will tolerate the drug at a later time without toxicity and with successful conversion.

**Dr. Dimond:** There is another characteristic item about the use of quinidine that is not often stressed in teaching: the peculiar experience of gradually increasing the quinidine dosage to full tolerance and to definite toxicity and yet with no success at conversion. When the drug has been stopped and the drug level has been allowed to dissipate, the patient will convert to normal sinus rhythm. This may occur at 6 to 8 hours after the last dosage, for example. I call this the “make or break” phenomenon and I have no reasonable explanation for it.

**Dr. Logue:** I would like to comment on a recent patient who was not digitalized and who developed atrial fibrillation in the postoperative period during the infusion of glucose. The intravenous administration of 3.0 Gm. of potassium chloride over a period of 2 hours caused a reversion to normal sinus rhythm.

**Dr. Blumgart:** Time is going on, and we have so many subjects here that I think we will go right on to the next one, which is the subject proposed by Dr Logue, namely, “The Control of Arrhythmias by Decreasing Thyroid Metabolism.”

**Dr. Logue:** The usefulness of radioactive iodine in the management of arrhythmia in the patient with thyrotoxicosis is well known. However, its usefulness in the management of recurrent supraventricular arrhythmia in the euthyroid patient is less widely appreciated.

It has been well demonstrated that the production of hypothyroidism by $^{131}$I may be quite helpful in decreasing the frequency or preventing recurrent attacks of either atrial tachycardia or atrial fibrillation. Dr. Elliot Corday and his co-workers have reported excellent results in a group of 33, 28 having excellent results following such therapy. This treatment should be utilized for those patients who cannot be controlled by customary forms of therapy. It has been useful in patients with normal hearts, in patients who have arteriosclerotic heart disease, and in those with rheumatic heart disease. Occasionally patients with chronic atrial fibrillation will undergo spontaneous reversion to normal sinus rhythm following the production of hypometabolism.

An example of the use of $^{131}$I is illustrated in figure 6. This patient, 74 years old, had arteriosclerotic heart disease with frequent attacks of angina pectoris, accompanied by congestive failure, and attacks of syncope. These symptoms were not controlled with combinations of procaine amide and quinidine, but following the administration of $^{131}$I the patient became hypothyroid. The attacks ceased, the patient’s angina was greatly improved, heart failure subsided, and over a follow-up period of 1½ years she has been active and free from attacks of atrial fibrillation.

**Dr. Blumgart:** In our studies of the effect of hypothyroidism induced by $^{131}$I in the treatment of congestive failure in angina pectoris, we were interested in observing a small group of patients who had been fibrillating, and who had had atrial fibrillation for many years, and we were surprised to
WHAT'S NEW IN ARRHYTHMIAS?

Fig. 6. H.W.B. a 74-year-old woman with frequent disabling attacks of atrial fibrillation (upper strip) and angina pectoris. Protein-bound iodine was 4.4 mg. per cent and I\textsuperscript{131} uptake was 26 per cent. I\textsuperscript{131} (20 mc.) was given and there has been no recurrence of atrial fibrillation in 1\textfrac{1}{2} years since hypothyroidism was induced. Lower strip shows usual sinus rhythm.

find that when they returned on some occasion after treatment, they had persistent normal sinus rhythm.

In relation to Dr. Logue's comments, it might be interesting to relate some of the experiences of our group, particularly carried out by Dr. George Kurland, Dr. Arnold Golodetz, and Dr. A. Stone Freedberg, which will be reported on Sunday morning. They studied particularly 39 patients in whom the usual parameters of thyroid function were entirely normal, including the protein-bound iodine and clinical signs. However, in about a third of those patients there were 2 abnormalities in regard to thyroid function that were disclosed by further studies, namely, the turn-over of thyroxine in the blood was greatly increased, and the red-blood-cell uptake was greatly increased. These changes were found in about a third of the patients. In the 39 patients studied, 5 patients were favorably influenced by the administration of ordinary stable iodine; all but 2 had normal thyroid function. In 2 of these patients, when the stable iodine was stopped, atrial fibrillation recurred, to be replaced again by normal sinus rhythm when the iodine was resumed. I think a very important practical point in the treatment of some patients with arrhythmias, even though they seem to be entirely euthyroid, is that if you are not going to think of using radioactive iodine in the next 6 to 8 months, it might be worthwhile to try ordinary stable iodine.

Dr. Logue: Did any of these patients have abnormal protein-bound iodine?

Dr. Blumgart: No.

Dr. Logue: We have been interested in a group of those who have normal uptakes and normal protein-bound iodine, who have nodules demonstrated by radioactive scanning, and who have responded following administration of I\textsuperscript{131}. Did some of the cases discussed by you have nodules?

Dr. Blumgart: No, not by clinical examination. I say that with reservation because, as we know, there may be small adenomas present on pathologic examination that may not have been disclosed on physical examination.

Dr. Sokolow: There is one aspect of hyperthyroidism about which I should like to comment. In the development of hyperthyroidism there is a period, sometimes perhaps weeks or months, during which time the usual tests for hyperthyroidism may be negative, only to become positive at a later time. We have seen at least 2 patients with paroxysmal atrial arrhythmias, 1 with fibrillation, and 1 with flutter, in whom we suspected the possibility of an overactive thyroid gland, but all the tests were negative; 6 months later it was obvious that the patients had
hyperthyroidism, and appropriate therapy prevented recurrent attacks of arrhythmia. In addition to the possibility, therefore, that the patient may not show overt hyperthyroidism, he may present himself with paroxysmal atrial arrhythmias during the process of developing thyrotoxicosis.

**DR. BLUMGART:** I would state in regard to the series that I mentioned, Dr. Freedberg and Dr. Kurland have examined these patients repeatedly.

It is too bad that our time is getting short. We have time for only 2 more subjects. The next one will be discussed by Dr. Harvey, namely, "The Present Treatment and Prevention of Ventricular Tachycardia."

**DR. HARVEY:** In the treatment of ventricular tachycardia early recognition of the presence of this arrhythmia is most important, as this should be regarded as a medical emergency requiring prompt treatment. The choice of drugs is generally between quinidine or procaine amide. As a rule, procaine amide intravenously is used first and the following precautions are taken: The ampule containing 1 Gm. of procaine amide is diluted to 100 to 125 ml. with 5 per cent dextrose in water. A vasopressor such as Levoephed is also available in another solution for intravenous use, if necessary, for any hypotension that develops. The separate solutions of procaine amide and Levoephed are connected to the intravenous tubing so that either solution can be stopped or given when necessary. On the opposite arm a blood pressure cuff is attached so that frequent blood pressure determinations may be taken during the infusion. A direct-writing electrocardiograph to enable constant monitoring of the electrocardiogram is also employed. Procaine amide is then started and we attempt to give the 1 Gm. over a period of approximately 30 minutes. If hypotension develops, the procaine amide is stopped temporarily and replaced by the infusion of Levoephed. On the other hand, if no hypotension develops, the vasopressor is merely held in readiness. At the first sign of reversion of ventricular tachycardia procaine amide is discontinued. Occasionally, the entire amount may be given without reversion to normal sinus rhythm. If such is the case, before giving an additional amount of procaine amide it is preferable to wait one half to 1 hour, or even longer, as reversion may take place during this period of waiting. If after this period, however, the ventricular tachycardia persists, then an additional gram of procaine amide is diluted in the same manner as described and with similar precautions. As a rule, reversion takes place during the infusion of the first gram of the procaine amide. Once reverted, we have found oral quinidine preferable for maintenance, and generally give 0.3 Gm. at intervals of approximately every 6 hours.

The patient who initially presents with ventricular tachycardia and shock or significant hypotension is first given the vasopressor agent, as we have seen occasional instances in which reversion has occurred merely by elevating the blood pressure, thereby making administration of procaine amide unnecessary. In fact, any type of arrhythmia associated with shock or severe hypotension may possibly be reverted or aided by the initial use of a vasopressor agent.

If ventricular tachycardia is a manifestation of digitalis toxicity, then the use of potassium is indicated with or without additional procaine amide.

Time does not permit a more detailed discussion of the more complicated or resistant types of ventricular tachycardia. The treatment as discussed has, in our experience, proved useful and generally successful.

**DR. BLUMGART:** The next subject on our program is "The Use of External Electric Current for Cardiac Standstill and Ventricular Fibrillation," which will be taken over by Dr. Logue.

**DR. LOGUE:** One of the most significant advances in the management of cardiac standstill and arrhythmias such as ventricular fibrillation has been the development of the external cardiac pacemaker and the external defibrillator by Dr. Paul Zoll and
his co-workers. It has certainly been lifesaving in patients with Stokes-Adams seizures, whether naturally occurring or induced by intracardiac surgery. I think it is most useful in patients with coronary disease in whom cardiac standstill or ventricular fibrillation occurs. Most of the patients who die with rhythm disturbances due to coronary disease do so by standstill rather than by fibrillation. I think it’s use is important in every instance of cardiac arrest in which thoracotomy has been done. The patient should be hooked to the stimulator prior to closing the chest and this should be maintained for some days because of the frequency with which recurrent ventricular tachyarrhythmia, standstill, and ventricular fibrillation occurs. Its use is valuable when one is using toxic drugs such as quinidine, digitalis, potassium, or procaine amide. The electric stimulator should be available when procedures such as pericardial paracentesis is being done, since ventricular standstill or ventricular fibrillation may rarely occur.

I should like to present 3 cases to illustrate the use of the pacemaker and the external defibrillator. The first of these is a patient with Stokes-Adams disease. The second is a man of 40 with his first myocardial infarction who developed cardiac arrest. The third patient developed ventricular fibrillation due to quinidine therapy.

The first patient, a woman of 74, had coronary arteriosclerosis with angina pectoris and frequent Stokes-Adams attacks. For a time the attacks were controlled by the use of the external stimulator but death ultimately occurred. In many instances, however, the patient may be carried through a critical period of depressed atrioventricular conduction and recovery may ensue. Isoproterenol is frequently useful, given as a constant in-
travenous infusion or sublingually. The intravenous administration of molar lactate is valuable on occasion and may produce a return to normal atrioventricular conduction; however, its effects are of brief duration (fig. 7).

The second patient was a man of 40 with his first myocardial infarction. He was given procaine amide prophylactically by intramuscular injection because of ventricular premature beats. Approximately 24 hours later he developed sudden cardiac arrest. An intern notified the family of his apparent death but an alert fellow instituted artificial respiration and the cardiac pacemaker, with a return to normal sinus rhythm and complete recovery.

The third patient, a woman of 74, developed chronic atrial fibrillation while taking thyroid extract. Following the administration of quinidine, 0.4, 0.6, and 0.8 Gm. at inter-

vals of 2 hours, conversion to sinus rhythm occurred 6 hours after the last dose. A prophylactic dose of 0.4 Gm. was given but prior to this dose the patient had a convulsive seizure thought to be due to cerebral embolism. Some 14 hours after conversion she began to have ventricular fibrillation with respiratory arrest and continuous convulsive seizures. At this time, her physician, Dr. John Wade of Montgomery, began mouth-to-mouth resuscitation and the use of the external defibrillator. Figure 8 demonstrates selected strips showing ventricular fibrillation, electric defibrillation, and cardiac standstill; external electric stimulator was ineffective at this point, but an intrinsic idioventricular rhythm appeared that was soon followed by normal sinus rhythm. Recovery was uneventful.

Dr. Wade was curious about the sequence of events and upon checking found that the
pharmacist had sent enteric coated quinidine, so that delayed toxic levels were reached well after conversion occurred, some 14 hours after the last dose of quinidine. One should keep in mind the hazards of long-acting quinidine in the conversion of atrial fibrillation, particularly in situations in which absorption is for any reason delayed.

**Dr. Blumgart:** Are there any comments?

**Dr. Dimond:** I would like to warn about one very definite hazard from the cardiac monitor and pacemaker in a patient with a recent coronary occlusion. Frequently during the night the electrode contact to the skin will be broken, either through the patient's turning in his sleep or by the electrode paste's drying out. This broken contact will activate the stimulator and the sleeping patient will begin receiving a series of electrical jolts through his chest wall.

**Dr. Blumgart:** The other point is, you would not have to have it so arranged that the stimulator would go on automatically—just have the auditory signal.

Dr. Zoll tells me that now there have been 200 cases who have been treated successfully by means of the cardiac pacemaker, and he has had personal experience in the defibrillation of 8 patients, including 5 who also had at times paroxysmal ventricular tachycardia, which was also abolished by countershock.

I should like to thank the panelists for their effective presentations.

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Analyses were reported showing that sodium was a trace element in bananas with the mean sodium content being 0.41 μg./Gm., or approximately 0.002 mEq. per banana—a negligible amount. The variations in sodium content of fruit grown under ordinary conditions were found to be quantitatively insignificant. This data indicated that bananas were suitable as the staple portion of low-sodium diets. Two patients with salt retaining syndromes developed a profound diuresis when placed on a diet consisting of 10 bananas supplemented by 1.5 liters of low-sodium milk. The diet was palatable, nutritious, and well tolerated. It provided 1,980 calories, 62 Gm. of protein, and an average sodium content of 3.02 mEq.
What’s New in Arrhythmias?
HERRMAN L. BLUMGART, W. PROCTOR HARVEY, R. BRUCE LOGUE, MAURICE SOKOLOW and E. GREY DIMOND

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