RESECTION OF THE AURICULAR APPENDAGES*

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RESECTION OF THE AURICULAR APPENDAGES has been proposed as a means of preventing recurrent arterial embolism in patients with rheumatic heart disease and auricular fibrillation.[8] This procedure was suggested by the frequent demonstration at autopsy of mural thrombi in the auricles of such patients who had rheumatic heart disease and auricular fibrillation and by the frequency of pulmonary and peripheral embolism. Weiss[11] stated that "rheumatic heart disease, more than any other type of heart disease, is responsible for embolic manifestations."

The incidence of mural thrombi in rheumatic heart disease increases significantly with persistent auricular fibrillation. In examining 116 cases of rheumatic heart disease, Garvin[3] found mural thrombi in 37 (31.9 per cent). Twenty-six (43.3 per cent) of the 60 patients in whom auricular fibrillation had occurred were found to have mural thrombi and 86.5 per cent of the thrombi arose in the atria (Tables I and II).

In another report, Garvin[4] recorded that in cases in which thrombi were present in the left side of the heart, 48.7 per cent showed one or more infarcts in the brain, kidneys, spleen, intestines, and/or extremities. In those with thrombi in the right side of the heart, pulmonary infarcts were found in 56.5 per cent and in left-sided thrombi peripheral infarcts occurred in 50 per cent of the cases.

Intracardiac thrombi were recorded in 22 of 30 cases by Stone and Feil[10] and in 11 of 14 by Graef and his associates.[5] The latter authors reported that in no instance was a thrombus found in the chamber of the auricle proper and that the left auricle was uniformly affected more severely than the right. Thus the auricles, and particularly the left, harbor mural thrombi in the majority of cases (Table II).

The age of the patient as well as the presence of auricular fibrillation appears to have direct influence upon mural thrombus formation. Mural thrombi have been found to occur more often in older patients.[3]

The need for prevention has been stressed by the relatively unsatisfactory results of treatment of peripheral embolus. Frequently the embolus lodges in

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a portion of the arterial bed where surgical intervention is not feasible, and even in those cases in which embolectomy can be undertaken, morbidity and mortality have been high. In several large series,[9] mortality has ranged from 40 to 59 per cent, and amputation because of resulting gangrene has been required in 18 to 22 per cent. In only 22 to 37 per cent of these cases has good circulation been restored. Once a peripheral embolus has occurred, recurrent emboli are not infrequent and are all too often fatal.

TABLE I.—*Occurrence of Mural Thrombi in R. H. D.*

		No. of Hearts	Thrombi	Percent
Graef[5]	Total	178	24	13.5
	With AF	14	11	78.5
Garvin[3]	Total	116	37	31.9
	With AF	60	26	43.3
Stone[10]	Total	100	37	37
	With AF	30	22	73.4
Hay[6]	Total	186	63	33.9
	With AF	106	52	49

The serious nature of embolic manifestations in rheumatic heart disease has been emphasized by Weiss and Davis[11] in a study of 474 autopsied cases. In 164 of these, heart disease was the cause of death. Of this group 73 (45 per cent) showed visceral or pulmonary infarcts of single or multiple organs. Embolism was felt to cause or contribute to death in 44 cases, or 26.8 per cent, of the 164 autopsies.

The feasibility of resection of the atrial appendages has been studied experimentally.[1, 2, 7] It has been demonstrated in experimental animals that the

TABLE II.—*Location of Mural Thrombi in R. H. D.*

	Ventricles			Auricles		
	Left	Right	Bilateral	Left	Right	Bilateral
Graef[5]	..	..	..	14	5	5
Garvin[3]	1	3	1	13	12	7
Stone[10]	2	4	..	19	16	..

auricular appendage can be removed without interruption of the normal heart action, with prolonged survival and with complete endothelialization at the site of removal. There does not appear to be any tendency for thrombus formation at the site of suture.

In 1949 Madden[8] reported two cases in which the left auricular appendage was removed. Both patients had rheumatic heart disease with mitral insufficiency and stenosis and persistent fibrillation, and had suffered embolic phenomena. One patient died of undetermined causes one week following operation. The other patient was found to have a left hemiparesis immediately after operation, which showed progressive improvement during the eight months follow-up period reported. These cases demonstrated that auricular

appendectomy was feasible in the human being. Stimulated by this report, the authors undertook the following cases:

Case 1.—R. B., a 50-year-old white male, was admitted July 11, 1949, to Wadsworth General Hospital, complaining of lower abdominal pain of one week's duration. The patient gave a history of "heart trouble" of approximately 12 years' duration, beginning with a coronary occlusion in 1938, for which he was hospitalized for 3 months. During the succeeding years, he was under medical observation but was relatively symptom free, save for mild substernal discomfort on exertion and occasional ankle edema.

One week prior to admission, while walking, the patient had sudden severe pain in the lower abdomen resulting in syncope. Nausea and vomiting associated with diarrhea followed, lasting 3 days but without melena. The abdominal pain persisted and increased somewhat in severity. Examination revealed a well-nourished and developed, chronically ill appearing, white male. Blood pressure 130/90, pulse 68 and irregular. Neck veins were slightly distended. Lower two-thirds of the right chest were dull to percussion anteriorly and posteriorly; occasional sibilant râles were noted in the left base and moist râles were present over the right base. Cardiac dullness extended to the left anterior axillary line in the sixth interspace. Abdomen was tender to palpation in the right lower quadrant with muscle guarding. The liver edge was 3 cm. below the costal margin. There was one plus ankle edema.

Laboratory. Hemoglobin and red blood cell count were normal; white blood count 14,200. Corrected sedimentation rate was 16 mm/hr., serology negative. Stools were negative for occult and gross blood. Blood urea nitrogen 16 mg. per 100 cc. Roentgenogram of chest, July 12, 1949, showed increased density over the right lower lung fields, interpreted as pleural effusion. Plain film of the abdomen was negative. Fluoroscopy, July 20, with barium swallow, indicated mitralization of heart. Electrocardiogram demonstrated auricular fibrillation.

Course. The patient improved with digitoxin, mercuhydrin, and a low salt diet; however, on July 22, 1949, he complained of sudden pain in the left leg associated with pallor of the left lower extremity and absent popliteal and tibial pulsations. A left femoral embolectomy was performed 3 hours later without evidence of return of circulation to the extremity. A similar episode involving the right lower extremity occurred July 31, and right femoral embolectomy was performed five and a half hours later. This leg also remained cold and cyanotic. Three days after the second operation the patient developed numbness in the left arm lasting 48 hours but without demonstrable diminution of radial pulsations. Digitoxin dosage was increased and the patient's legs were refrigerated. Slowing of pulse followed this treatment.

On August 9, 1949, left auricular appendectomy was performed. After the heart was exposed the left auricle seemed to contain a thrombus, but following amputation of the auricle, thrombus formation was absent to gross inspection. The pathologic report, however, was "old and recent organizing mural thrombi, left auricular appendage; myocardial degeneration; thickening and fibrinoid degeneration of endocardium." He received oxygen for 3 days after operation.

The patient made a satisfactory recovery. On August 26 bilateral mid-thigh amputations were performed. The patient withstood this well but developed a superficial slough of the skin flaps of the left stump. On August 31 a thoracentesis was performed, 400 cc. of serosanguineous fluid being removed. Approximately 6 weeks after operation, the patient had an episode of congestive heart failure, from which he recovered, but he has had no evidence of further embolic episodes now more than 8 months after operation. He is maintained on 1 to 2 injections of mercuhydrin weekly and a salt free diet.

Case 2.—W. D., a 63-year-old, white, unemployed male was admitted Nov. 5, 1949, complaining of right chest pain and bloody sputum of two days duration.

The patient had developed a mild non-productive cough one week earlier and had noted mild dyspnea. Two days before admission, 5 hours after his evening meal, he developed a paroxysm of coughing productive of bright red blood. This was accompanied by left parasternal pain radiating to the left shoulder. The pain continued intermittently, and he had repeated small hemoptyses. There has been no ankle edema.

The patient gave a long history of rheumatic heart disease with mitral stenosis and auricular fibrillation. He was hospitalized first in 1941 for similar symptoms and a diagnosis of pulmonary infarction made. In 1947 he was admitted with hemoptysis and transient left hemiparesis which responded to conservative therapy. In April, 1949, an episode of mild congestive failure required hospitalization. He has since been in the Domiciliary Unit of the V. A. Center, where he has received a low salt diet, 0.1 mg. digitoxin daily.

Physical Examination. The patient was a thin, chronically ill, white male. Bright red sputum was expectorated during examination. There was moderate dyspnea present. Blood pressure was 148/86, pulse 84, irregular with pulse deficit of 20. The chest was emphysematous. Diminished breath sounds at right base posteriorly and moist râles present at both bases. The heart was enlarged both to right and left. There was a Grade II systolic and diastolic murmur at the apex. Liver edge 4 cm. below costal margin.

Laboratory. Serology was negative. Erythrocytes and hemoglobin were within normal limits. White count was 10,600 with 92 per cent neutrophiles. Urinalysis was negative except for one plus albumen and rare hyalin casts. Roentgenogram of chest demonstrated cardiac enlargement, pulmonary emphysema and a shadow in the right lower lung fields interpreted as pulmonary edema with effusion. EKG showed auricular fibrillation and digitalis effect.

Course. The patient improved on bed rest and general supportive measures. The amount of hemoptysis progressively decreased until 2 week after admission when a sudden episode of chest pain occurred accompanied by hemoptysis. Roentgenographic examination demonstrated no change. A week later a third episode occurred. Repeated examination of the legs revealed no evidence of thrombophlebitis.

On Dec. 16, 1949, a right auricular appendectomy was performed. Pathologic report: "auricular appendage showing small organizing mural thrombus and extensive myocardial degeneration and necrobiosis." The course following operation was satisfactory. The patient was ambulatory by the sixth day after operation but required occasional nasal oxygen for 12 days for dyspnea. Accumulation of left pleural fluid required thoracentesis on 2 occasions. On January 11, 1950, a bilateral superficial femoral vein ligation was performed under local anesthesia because of mild tenderness in the right calf muscles. No evidence of thrombus formation was apparent. The patient has been transferred to the Domiciliary, where he is taking a low salt diet and 0.1 mg. of digitoxin daily. He requires 1 cc. of mercuhydrin once or twice a week. He has remained free of chest pain or hemoptysis since operation.

Case 3.—H. B., a 53-year-old retired elevator operator, was admitted January 10, 1950, complaining of pain and numbness of the right hand and forearm of 4 hours' duration. The pain was sudden in onset and was followed by numbness associated with pallor of the right hand and forearm. The pain subsided gradually and the pallor and numbness regressed. Upon admission, pain and color changes had disappeared and there was only residual weakness and coolness of the hand remaining.

Past History. At the age of 23, the patient had rheumatic fever and was treated by bed rest for 8 months. The patient was not aware of cardiac involvement. In 1947 he developed sudden severe pain in the left leg associated with numbness and coldness. He was hospitalized elsewhere and treated with anticoagulants. On that hospitalization,

he was found to have auricular fibrillation and roentgen rays showed cardiac enlargement with "right heart strain." He was placed on digitalis at that time and has been maintained on digitalis since. A similar episode of left leg pain occurred in 1948 and this was followed by intermittent claudication. In April, 1949, the patient had a pulmonary embolus and was hospitalized here. On this admission a left lumbar sympathectomy was performed for arterial insufficiency of the left leg with improvement in symptoms. He suffered a transient episode of pain in the right leg in October, 1949, and had mild intermittent claudication following this. A few weeks later a sudden attack of RLQ pain of 6 hours duration was associated with nausea and vomiting. This subsided spontaneously. He has noted occasional dyspnea and ankle edema since 1936. Past history was not remarkable otherwise except for hemorrhoidectomy in 1927, appendectomy in 1931 and herniorrhaphy in 1938.

Physical Examination. This patient was a thin white male appearing somewhat older than his stated age. Blood pressure was 120/70. Chest was clear to percussion and auscultation. His heart was enlarged 2 cm. beyond MCL, fibrillating at rate of approximately 80 with pulse deficit of 8-10. No murmurs were detected. Peripheral vessels were sclerotic and dorsalis pedis pulse absent bilaterally. Popliteal pulse was strong on the right, absent on left. The right hand was dusky and cooler than the left, but the radial and ulnar pulses were palpable and equal bilaterally. The left leg was warmer than the right. Reflexes in both upper and lower extremities were physiologic.

Laboratory. A roentgenogram of chest showed cardiac enlargement with chronic pulmonary congestion and pulmonary emphysema. Urinalysis was negative. Blood count was normal except for leukocytosis of 11,000. Kahn was positive 8 units; Wassermann negative. EKG demonstrated ventricular rate of 60 with occasional extra-systoles.

Course. Following admission the patient was given intravenous heparin for 48 hours. The hand improved progressively and appeared entirely normal by this time. Cardiac consultation was obtained, and it was decided that the patient was a suitable candidate for left auricular appendectomy. Operation was performed January 17, 1950, under endotracheal ether anesthesia. A thrombus was demonstrated in the auricular appendage at operation. Postoperatively the patient developed a tachycardia of 140 for 24 hours. He was maintained on digitoxin and the apical pulse was maintained at 90 after the first day following operation. Postoperative EKG, January 19, showed no essential change from that taken prior to surgery. Six days after operation 550 cc. of serosanguineous fluid was removed from the left pleural cavity. Seven days after operation the patient developed mild numbness and coolness of the right foot with diminution of the popliteal pulse. A right paravertebral block relieved the symptoms and improved the pulsation. Thoracentesis was again performed on the thirteenth postoperative day, 250 cc. of straw-colored fluid being withdrawn. The patient was asymptomatic thereafter and was transferred to the Domiciliary on February 14, where he receives a low salt diet and digitalis.

PREOPERATIVE PREPARATION

Patients with chronic rheumatic heart disease and auricular fibrillation with history of embolism are considered candidates for this operative procedure. It is important that a careful evaluation of the cardiac status be made under the supervision of a cardiologist. Cardiac compensation must be achneved prior to operation in these patients, who are relatively poor operative risks. Whether or not an attempt should be made to convert the fibrillation to normal sinus rhythm must be decided by the cardiologist in each individual

case. The attempt was made unsuccessfully in one of our cases (R. B.). The second patient had been found to be sensitive to quinidine on an earlier admission. The first two patients were admitted with signs of mild cardiac decompensation and were treated with a low salt diet, digitoxin and mercuhydrin. In the first case a rapid rate of fibrillation persisted. The lower extremities had undergone gangrenous changes due to arterial insufficiencies following bilateral emboli, and it was not until refrigeration to the lower thigh was instituted that the tachycardia diminished.

The selection of patients requires individual consideration of each case. At the present time, the decision for operative intervention is made by consultation between the cardiac and surgical services. It will require further experience with the procedure and a longer follow-up study of those who have been subjected to this operation before definite criteria can be established.

ANESTHESIA

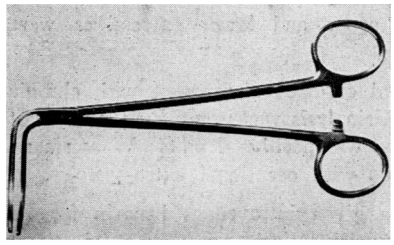


FIG. 1.—Modified right angle clamp used in resection of auricular appendage. Clamp devised from kidney pedicle clamp by removing serrations from jaws.

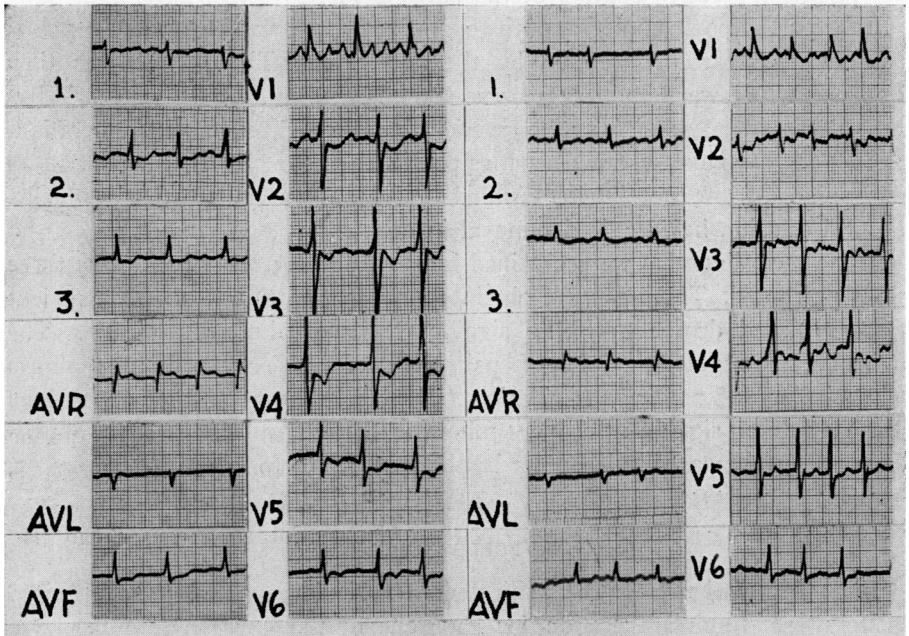
Preoperative medication and anesthesia must be selected in consultation with a competent anesthetist. In the cases reported here, morphine sulfate and atropine sulfate were administered preoperatively. Narcotics have been kept at a minimum to prevent respiratory depression. Endotracheal ether following induction with intravenous sodium pentothal has been satisfactory in this series. A high concentration of oxygen during operation is desirable.

OPERATIVE PROCEDURE

The chest is opened through the third interspace. The costal cartilage of the adjacent rib, either above or below the interspace, is divided and the ribs retracted. This provides good exposure to the left auricle, but it was found to be less satisfactory on the right side where the right auricle was found to lie in close proximity to the posterior aspect of the sternum. In this instance, it was necessary to remove a small portion of the sternum at this point. The lung is retracted gently laterally beneath a well padded broad retractor. The pericardial sac is opened in its longitudinal plane with an incision anterior to and parallel to the phrenic nerve. It has been necessary to make a perpendicular extension in the mid-portion of the pericardial incision to gain adequate access to the base of the auricle. The auricular appendage is manipulated gently during the application of the clamp to avoid accidental loosening of mural thrombi. A modified right angle, non-crushing clamp (Fig. 1) is placed across the base of the appendage and gradually closed. It is now helpful to place a smooth clamp, *e.g.*, sponge forceps, on the apex of the appendage. A

continuous suture of #00000 silk with an atraumatic needle is now placed across the base of the appendage distal to the clamp and tied at either end. The auricular appendage is now incised distal to this first suture line for a distance of approximately three-fourths its width. While the appendage is still attached, a second continuous suture of silk is placed over the end of the auricle and completed with the removal of the attached auricle. The clamp across the base is now cautiously loosened and the suture line observed for

FIG. 2



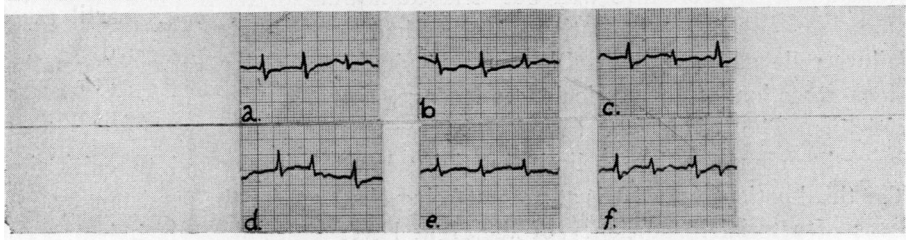


FIG. 3

FIG. 2.—Electrocardiogram from Case I (R. B.), demonstrating auricular fibrillation and right axis deviation. The tracings on the left were obtained August 8, 1949, prior to operation. The record to the right was made October 3, approximately two months after operation.

FIG. 3.—Single lead electrocardiographic records obtained during operative procedure in Case I (R. B.), August 9, 1949; (a) 8:45 A.M. following induction of anesthesia; (b) 9:00 A.M. pericardium entered; (c) 9:30 A.M. following application of clamp to base of atrial appendage; (d) 9:40 A.M. auricular appendectomy completed; (e) 10:00 A.M. pericardium closed; and (f) 10:20 A.M. operative procedure completed.

bleeding. It is usually necessary to place a few mattress sutures to obtain adequate hemostasis although bleeding has not been a problem in this series.

The pericardium is now closed with interrupted silk sutures, a defect being left in the lower end to prevent tamponade should pericardial effusion develop. The lung is allowed to expand and the chest wall closed in layers of silk. The pleural cavity is aspirated carefully and has been closed without drainage.

POSTOPERATIVE COURSE

The postoperative courses of the patients in this series have been quite satisfactory. Upon completion of the operative procedure, the tracheobronchial tree received careful attention by the anesthetist. The patient was then returned to the ward and oxygen was administered by nasal catheter. It has been necessary to administer oxygen for three to eight days after operation. Oral fluids have been given as soon as possible and the patients maintained on digitoxin and low salt intake. Ambulation has been undertaken gradually. Pleural effusion has required removal in each case.

Electrocardiograms were obtained before and after operation in the three cases presented. Tracings were obtained during operation in the first patient (Case 1) and demonstrated no significant change during operation. The pulse became regular according to palpation but the electrocardiogram shows a persistence of auricular fibrillation (Fig. 3). The postoperative tracings demonstrate no significant changes following the removal of the atrial appendage when compared with records obtained prior to operation (Figs. 2, 4 and 5).

FOLLOW-UP

The first patient (R. B.) subjected to auricular appendectomy required bilateral mid-thigh amputation for gangrene of the lower extremities. He withstood this procedure well but delayed healing of the right amputation stump prolonged hospitalization. He has had two minor episodes of cardiac failure relieved by aminophyllin and mercuhydrin. During the eight months since resection of the auricular appendage, there has been no evidence of embolus. The second case (W. D.) is now asymptomatic and is maintained in cardiac compensation on limited activity, low salt intake and mercuhydrin. The third patient (H. B.) is at present being observed for the persistent symptoms of arterial insufficiency of the right lower extremity which had been present prior to the removal of the left atrial appendage. The latter two patients have had no episodes suggesting embolism four and three months since operation, respectively.

DISCUSSION

It would now appear that removal of the auricular appendage can be accomplished in the human being with reasonable safety. It is realized that

FIG. 4

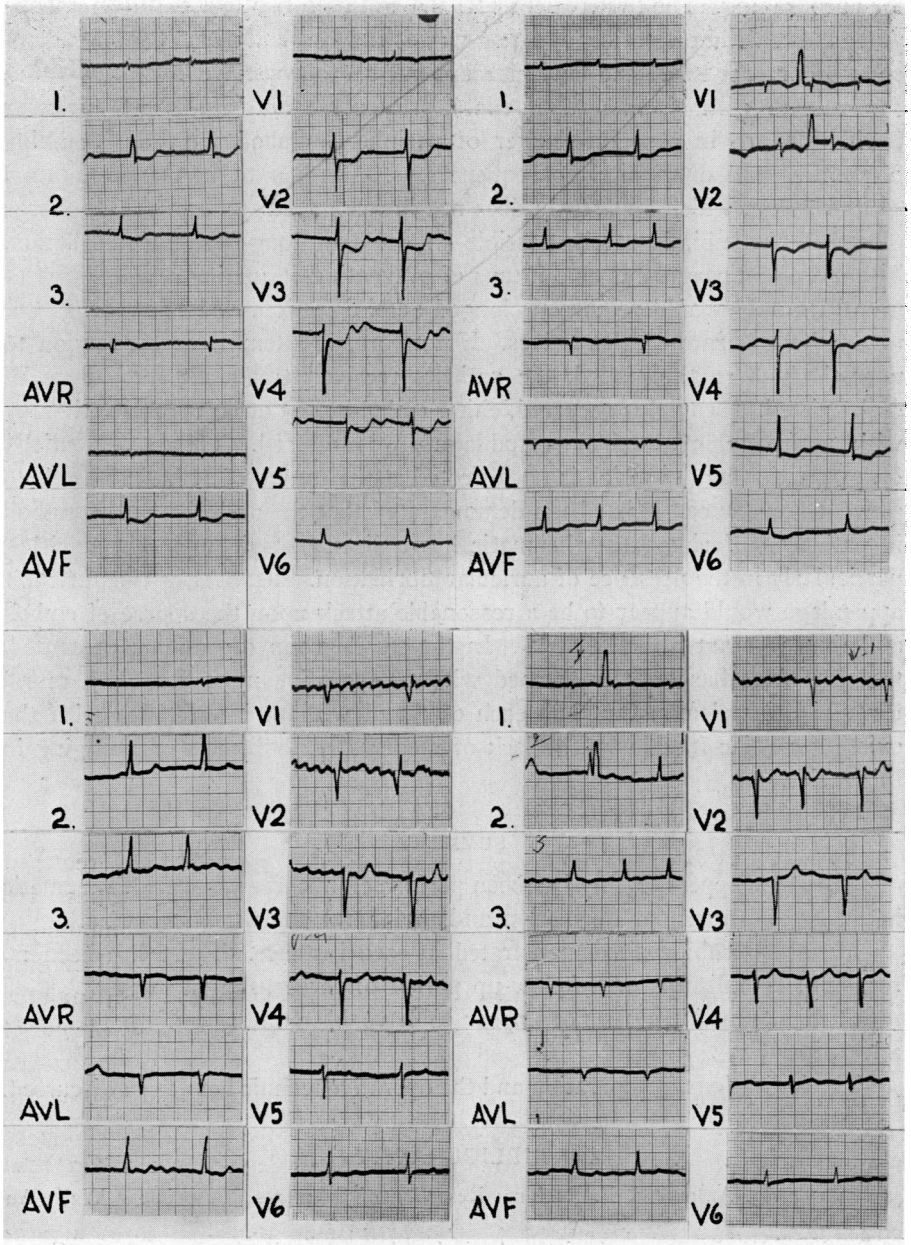


FIG. 5

FIG. 4.—Electrocardiograms from Case 2 (W. D.). The tracings to the left were obtained November 21, 1949, before operation, and those presented on the right February 3, 1950, seven weeks after operation.

FIG. 5.—Electrocardiograms from Case 3 (H. B.). The records on the left were made January 11, 1950, six days before operation, and the tracings on the right were obtained February 3, two weeks following resection of the left atrial appendage. In addition to auricular fibrillation, occasional ventricular extrasystoles are noted.

the follow-up studies are brief in the cases presented, but the results are thus far encouraging. The first patient (R. B.) in this series had multiple embolic phenomena within a period of a few weeks and has had a notable absence of such occurrence since the auricular appendage was removed.

Patients with chronic rheumatic heart disease and persistent auricular fibrillation are in constant danger of peripheral emboli with the resulting disabilities and often fatal consequences. Conversion to permanent normal sinus rhythm, which apparently does not in itself predispose to the discharge of mural thrombi, is rarely possible in cases of long-standing fibrillation. The use of anticoagulants as a means of preventing formation or discharge of mural thrombi would require prolonged administration, and these drugs are not without hazard in themselves. Heretofore, if attempts at conversion to normal sinus rhythm failed, the patients received treatment for failure, if such existed. If embolus occurred, either embolectomy or conservative therapy with anticoagulants and/or vasodilators was offered. This is essentially symptomatic therapy which is not directed at the source of emboli and fails to prevent recurrence. It has been demonstrated that the majority of the emboli in patients suffering from rheumatic heart disease originate in the auricles, particularly in the presence of auricular fibrillation. Removal of the auricular appendage would appear to be a reasonable attack upon the source of emboli and thus warrant a clinical trial. In the small group of patients presented, the procedure has been performed without operative mortality, with mural thrombi being demonstrated in each case and without interference with the existing cardiac function. The lack of recurrent embolism is encouraging in this series.

SUMMARY

Auricular appendectomy has been performed in three cases with rheumatic heart disease, auricular fibrillation and peripheral embolic phenomena.

Mural thrombi were demonstrated in each instance. There has been no recurrent embolism over a follow-up period of three to eight months.

Cardiac function does not appear to have been influenced by the operative procedures.

The indications for operation and the operative technic have been discussed.

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DISCUSSION.—DR. JOHN H. OLWIN: I should like to congratulate Drs. Longmire and Beal on the successful accomplishment of a difficult piece of surgery.

All too frequently, however, these patients are poor surgical risks, and as an alternative to surgery, I should like to present our limited experience with the use of anticoagulants in this condition.

We have given heparin and Dicumarol to six patients with auricular fibrillation and clinical evidence of peripheral emboli. In each patient the emboli apparently ceased after proper anticoagulant control was achieved.

In two instances the patients died of cerebral accidents, sudden in onset, several weeks after anticoagulants were discontinued.

A third patient remained on Dicumarol for nine months, and died three months after the withdrawal of the anticoagulant. At autopsy there was a recent coronary occlusion, with extensive myocardium infarct on, and ancient infarcts of the brain, spleen and kidneys. The latter were considered to be on the basis of old emboli. There was no evidence of recent embolism.

A fourth patient had a recurrence of his emboli ten days after anticoagulants were stopped. He was again free from them after reinstitution of the therapy, and has remained so for the past 18 months. He carries on as a laborer, and returns to the laboratory twice to three times each month for checks on his prothrombin level.

A fifth patient has been on therapy for six months, and has been free from evidence of emboli.

The sixth patient has been lost, and his present status is unknown.

In no instance was there observable evidence of emboli while the patients were on controlled anticoagulant therapy. It has been our experience that with proper methods for controlling Dicumarol administration, patients can be maintained on an out-patient basis over a period of years with safety. We have patients now who have been on as long as three and four years, with no evidence of Dicumarol poisoning being found.

It will obviously require much more extensive experience with this form of therapy before proper evaluation of it will be possible, but in patients with auricular fibrillation and recurrent emboli, who may not readily withstand surgery, this type of therapy is suggested as an alternative to surgery.

DR. IVAN D. BARONOFSKY: Dr. Longmire and Dr. Beal are to be congratulated on their excellent results. It occurred to us, at the time that Madden presented his results with auricular appendectomy, that perhaps a simpler expedient to the problem of prevention of recurrent embolization would be merely to ligate the left auricular appendage at its junction with the left atrium. In preliminary experiments in dogs, Dr. Abbott Skinner and myself found the auricular appendage actually fibrosing down to almost its disappearance in a matter of three months following the placement of a simple ligature at its base.