

Chronic Heart Block in Dogs. A Method for Producing Experimental Heart Failure

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A method is described for the production of chronic atrioventricular block in dogs, by incision of the region of the bundle of His through the open right atrium during temporary caval occlusion. Exercise tolerances, chest x-ray films, electrocardiograms, phonocardiograms, cardiac outputs, intracardiac pressures, femoral pressures, and left ventricular coronary flows were obtained pre-operatively and from 1 to 10 months postoperatively. The majority of the animals developed clinical, laboratory, and pathological evidence of congestive heart failure. All animals had generalized myocardial hypertrophy.

DESPITE the inherent limitations of transferring data on experimental heart disease from animals to man, it is possible, after surgical production of lesions in animals, to make controlled analyses of various syndromes which are analogous to human disease entities. In a contemporary study in dogs¹ the acute effect of surgically induced complete heart block on cardiocirculatory function was reported. The present communication is concerned with a study of the chronic effects of complete heart block in dogs, as well as with a more detailed description of the operative procedure.

During the study, the majority of the animals with chronic complete heart block developed right and left sided heart failure, either spontaneously or after several weeks of daily exercise. Because of the current interest in developing experimental means of producing congestive heart failure, the events and details of decompensation have been described as fully as possible.

METHODS

Twelve dogs were used, ranging in weight from 10 to 23 Kg. Intravenous Nembutal was used for all pre- and postoperative catheterization studies.

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Postoperatively, most animals were studied once with 20 to 23 mg. per kilogram, but all other pre- and postoperative determinations were done with 27 to 30 mg. per kilogram of Nembutal.

Left ventricular coronary flow in cubic centimeters per 100 Gm. muscle per minute was measured by the nitrous oxide desaturation method² after catheterization of the coronary sinus and femoral artery (which was also used to record blood pressure). Cardiac output was determined by the direct Fick method, using the pulmonary artery for the mixed venous sample. A closed system spirometer, connected to an airtight endotracheal tube, was used to measure total oxygen consumption. The blood samples were analyzed for nitrous oxide by the method of Kety and Schmidt³, and for oxygen content by the manometric method of van Slyke and Neill. Efficiency and other calculations were computed from the formulae summarized by Goodale and Hackel², assuming a respiratory quotient of 0.83.

Vascular pressures were detected with inductance or capacitance transducers, with electrical integration of the mean pressures when desired. The frequency response of the measuring and recording systems was in excess of 30 cycles per second when measured by recording pressures within a balloon during explosion. Electrocardiogram and vascular pressures were recorded on a direct writing oscillograph.

Specific details of other procedures are given under the appropriate sections.

RESULTS

The Technic of Surgical Production of Complete Heart Block

In 12 dogs, under ether anesthesia after morphine-atropine premedication, section of the region of the bundle of His was carried out. Permanent complete heart block was obtained in every case. One animal died on the third postoperative day, and all others

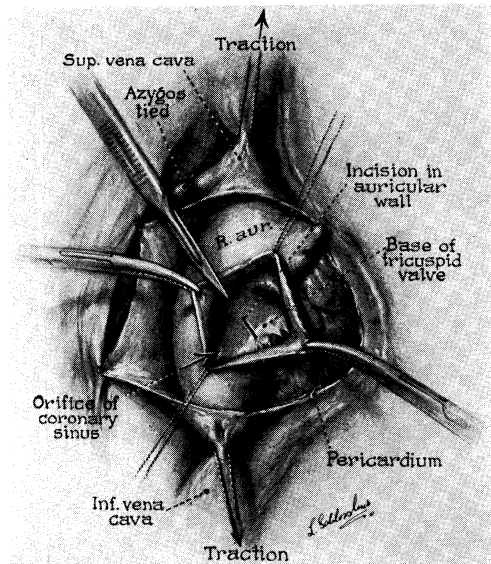


FIG. 1. Artist's view of the technique for surgical production of complete heart block. The auricle has been entered during temporary caval occlusion, and the cut made across the auriculoventricular junction at the posterior end of the base of the septal cusp.

survived for chronic study. The heart was approached through a thoracotomy in the right fourth costal interspace. Operative details are shown in figure 1. The azygos vein was ligated, and braided silk placed around the superior and inferior venae cavae, extrapericardially. The pericardium was opened anterior to the phrenic nerve. Arterial silk sutures were placed at the superior and inferior extremities of the proposed auricular incision, and the intervening auricular wall grasped in a curved non-crushing clamp. The auricular wall isolated by the clamp was incised. The caevae were occluded by traction on the braided silk, and the auricle entered. Residual blood in the right side of the heart and continuing drainage from the coronary sinus was sucked out and an incision was made across the auriculoventricular junction 5 to 10 mm. anterior to the coronary sinus (fig. 1). The cut was begun at the posterior end of the base of the septal cusp, and usually extended for a short distance into the contiguous auricle and ventricle. The relations of the incision at the auriculoventricular junction are shown in figure 2. It was similarly found by Erlanger and Blackman⁴ that complete heart block could be most easily produced by injury to the region illustrated.

Usually it was possible to be sure at what moment heart block was established for auriculoventricular dissociation was obvious even though the caevae were occluded, and the right side of the heart empty of blood. In all animals, a spontaneous ventricular

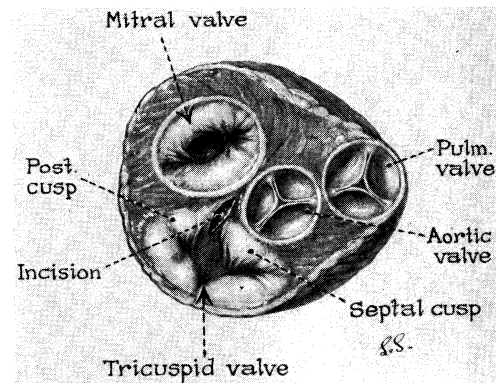


FIG. 2. Details of the anatomy of the intracardiac incision for heart block. The auricles have been removed, and the view is from above. Note the relation of the cut to the posterior end of the septal cusp, the left auricle, and the base of the aorta.

beat began immediately, and ventricular asystole at the operating table was never observed.

In most cases complete heart block was obtained during a single entry into the auricle with a total caval occlusion time of 50 to 60 seconds. In some animals in which block was not readily produced, it was necessary to reenter the heart several times to deepen or extend the cut. This was attended with an increased risk of accidental septal defects, and in 4 of the 12 dogs small communications were made through the membranous portion of the inter-ventricular septum or through the low interauricular septum. (See relations in fig. 2.) These defects were recognized by the presence of bright red blood in the base of the cut, and were closed immediately, usually by a single suture. In these instances in which complete block was obtained with some difficulty, multiple caval occlusions for short periods were well tolerated, and were not accompanied by any increase in postoperative difficulties.

At the conclusion of the intracardiac procedure, the caevae were released, allowing the right heart to fill with blood before placing the noncrushing clamp back on the auricular incision. The auricular wall was then closed with continuous over and over silk. With the slowed rate of complete block, the heart could be observed to be dilated, so markedly that the pericardium was generally not closed. X-ray films, taken immediately and for as long as the animals lived, revealed an invariable enlargement of the cardiac shadow (fig. 3).

The unanesthetized preoperative resting pulse rates of the animals in this study were from 90 to 140 per minute. After complete heart block, rates under the same conditions ranged from 30 to 65 per minute. Seven of the

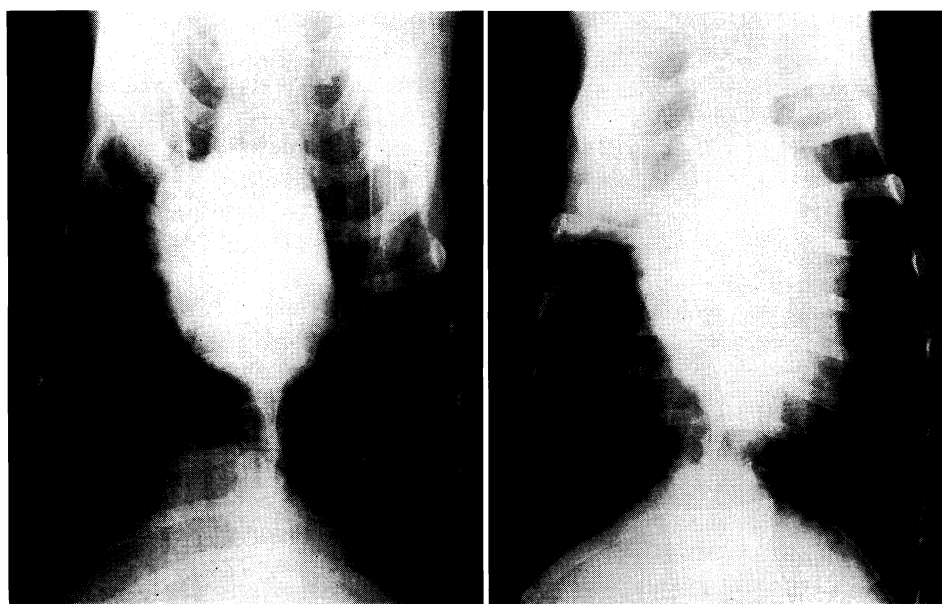


FIG. 3. Two-meter chest x-ray films preoperatively (left), and 12 days after performance of surgical complete heart block (right). Preoperative rate was 120 per minute and idioventricular rate was 40 per minute. Note cardiac enlargement and pulmonary vascular congestion after block.

11 dogs studied chronically had rates between 40 and 55 per minute, two animals consistently running lower and two higher rates than this usual range. The resting rate which prevailed for the balance of any dog's life had generally become evident within 48 hours, and remained about the same from day to day and month to month. It was not possible to explain the differences in the fundamental ventricular frequency within this group of dogs on the basis of different location of lesions, since all the cuts were made in essentially the same area.

It has been shown that the cardiac output of dogs is considerably reduced immediately after the surgical production of complete heart block (1, 5). This explains the clinical behavior of the dogs during the first two to seven days. They were easily fatigued, could be made to ambulate only with difficulty, and were so lethargic that an assisted feeding and watering program had to be initiated in several instances. The keynote of postoperative therapy was avoidance of situations which might tax an already dangerously low cardiac output. Water seal drainage was used in closing the thoracotomy, and vigilance was maintained for signs of pneumothorax and atelectasis. Heavy doses of anti-

biotics were given, since it was noticed early that even a superficial wound infection had a surprisingly detrimental effect on recovery. Activity was discouraged. The phlegmatic behavior characteristic of the early postoperative period gradually disappeared and after a week or 10 days it was difficult with casual observation to distinguish these animals from normal dogs. ●

Electrocardiographic confirmation of the complete heart block was obtained at frequent intervals postoperatively. It was possible, in addition, to study the auriculoventricular dissociation stethoscopically. Audible auricular sounds, at two and one-half to four times the frequency of the regular heart sounds, could be heard widely over the chest, most prominently at the anterior extent of the fourth intercostal space on the left. These auricular sounds were present in every animal. They were loudest immediately after surgery, and became fainter with the passage of time. The sounds were intensified after exercise, and softest during rest. Spectral phonocardiograms, which allow visualization of time, frequency, and intensity of heart sounds^{6, 7} were obtained in six of the dogs at 2 to 300 days after the performance of

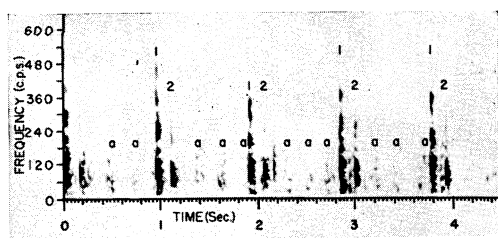


FIG. 4. Spectral phonocardiogram of dog which had been completely blocked for four months. The auricular sounds (a) seen in the record were easily detectable upon stethoscopic examination, although softer than the regular heart sounds (1, 2).

block.* An example is shown (fig. 4) in which by critical filtering, the auricular and ventricular sounds are boldly contrasted in a dog which had been blocked for four months. In several dogs, a prominent auricular diastolic murmur was detected on the spectral phonocardiogram in conjunction with the auricular sounds. The characteristics of this murmur were quite like those of a similar murmur described by Ryland in elderly patients with heart block.⁸

Clinical Course of Animals Which Developed Heart Failure

After the first few days of extreme lethargy, the blocked dogs began to eat well (diet consisted of standard grain-base dog meal) and superficially seemed normal. That complete compensation had not uniformly occurred, however, was evident from the fact that 5 of the 11 dogs studied chronically developed congestive heart failure within three months, essentially at cage rest, and two others failed later under the stress of controlled daily exercise. The animals with the slowest idioventricular rhythms were somewhat more prone to develop heart failure than dogs with faster rates, although this was not consistently the case. Of greater importance in the genesis of decompensation was the degree of spontaneous physical activity exhibited by the subject. For example, the dog which was observed for the longest time (10 months) had a resting rate of 30 to 35 beats per minute throughout his life, was slothful and never developed evidence of heart failure. The dog which first developed

* Spectral phonocardiography and analysis were done by Dr. Victor A. McKusick.

failure in this series (two weeks) had a rate of 65, but was hyperactive in the cage. The onset of symptoms and signs of heart failure occurred from 2 to 16 weeks after surgery and developed after the interlude of apparent adjustment described above. The first notable alteration was generally a return of the lassitude and anorexia which had been present immediately after surgery. Hepatomegaly could soon be detected, followed by ascites. Prolonged dyspnea followed the slightest exertion. Chest x-ray films usually showed pulmonary vascular congestion (fig. 3), a finding which in some cases was the first indication of impending failure.

Although there was evidence of both right and left heart failure, development of signs of right sided failure always dominated the ultimate picture, and terminally hepatomegaly and ascites were far more advanced than pulmonary edema and pleural effusion. The course of the decompensation was relatively benign. Only one of the seven dogs which decompensated died spontaneously. The rest were either sacrificed, or died as the result of accidents in catheterization or treadmill exercise. The majority of these animals were in mildly progressive failure for at least six weeks before their death.

To determine with certainty that the heart failure was primarily related to the lesion in the conduction system, rather than to nonspecific effects of the surgical procedure, six dogs were subjected to "sham" operations in which every step was taken except the actual section of the bundle of His. The animals were watched from three to six months, and none had any pre- or postmortem suggestion of heart failure.

The Effect of Chronic Heart Block on Vascular Pressures

It has been shown that the right auricular pressures of dogs with surgically induced complete heart block are only moderately elevated in the early postoperative period.¹ In the present series in which observations were started later, the dogs which developed clinical and pathological evidence of heart failure showed large increases in right auricular pressure to levels as high as 220 mm. H₂O. These rises were present in some cases at the end of the month, and in others appeared later (fig. 5),

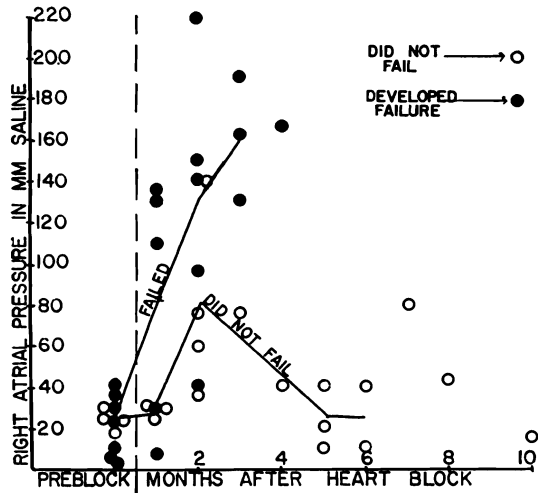


FIG. 5. Right atrial pressures in dogs with complete heart block, the open circles representing measurements from animals which did not fall, and the black dots being from animals which decompensated.

coinciding with the onset of clinical signs of decompensation. The pressure configuration (fig. 6C) did not suggest a significant tricuspid regurgitation in any of these dogs. The animals which did not develop heart failure either never had large rises in right auricular pressure or had early elevations which subsequently returned to relatively normal values (fig. 5).

With the extreme bradycardia of complete heart block, right ventricular ejection pressures were high, ranging from 40 mm. Hg to 70 mm. Hg (fig. 6B). The contribution of auricular systole to ventricular diastolic filling was demonstrable in every animal (fig. 6B). With the onset of failure the pressure rise during

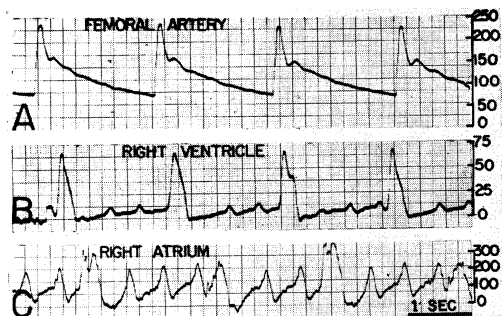


FIG. 6. Femoral arterial A, right ventricular B, and right atrial C pressures. Calibrations of the first two are in millimeters Hg and of the third in millimeters water. The records are all from animals in heart failure.

diastole became steeper and the auricular pressure waves more prominent. The example shown (fig. 6B) is from a decompensated animal in which the end diastolic pressure is 130 to 160 mm. H₂O. The pressure with ventricular systole returned to 0 in every animal except one (who was in far advanced failure), and in this exceptional case the end systolic pressure was 5 to 7 mm. Hg.

The femoral pulse, by palpation, was full and almost pistol shot in the dogs with the slowest rates. Direct arterial pressure recording generally showed an elevation of systolic pressure, with a wide pulse pressure (fig. 6A). In the case shown, the pressure was 230/80 mm. Hg. Mean blood pressure was in general slightly less than had been obtained in the preoperative controls (fig. 7). Significant differences in the arterial pressure levels, between the animals which failed and those which did not, could not be detected (fig. 7).

The Effect of Chronic Complete Heart Block on Exercise Tolerance

Before surgery, the exercise capacity of the dogs was obtained by use of a treadmill.⁹ A level running surface was used with a standard speed of 7 miles per hour. Tolerance was defined as the duration of running necessary to cause the dogs to collapse, and in the controls ranged from one to three hours.

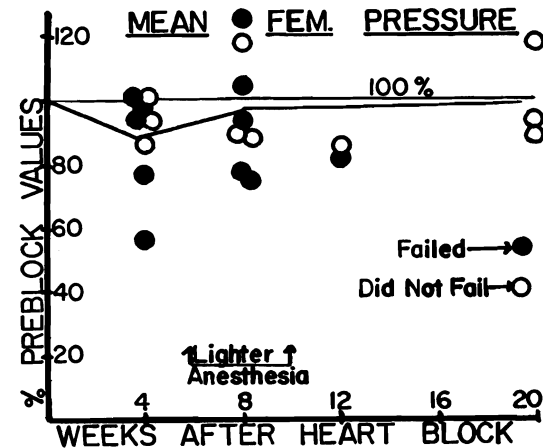


FIG. 7. Mean femoral arterial pressures in animals with chronic complete heart block, expressed in percent of preoperative controls (each animal thus serving as its own control). Note lack of correlation of values to presence or absence of heart failure.

Postoperatively, eight of the animals were tested monthly for exercise tolerance. During the first three months no exertion was allowed other than that necessary for the actual testing. At the end of three to four months, three of the eight animals had developed clinical signs of heart failure. At this time the remaining five animals were placed on a regimen of daily severe exercise. With this enforced exertional stress, two more animals developed heart failure, primarily right-sided, within two weeks. Both of these dogs died while running on the treadmill, in one case with rupture of a remarkably congested liver. The remaining three dogs were continued with daily exercise for five weeks, and far from developing any signs of decompensation, seemed to get stronger.

One month after the heart block, the exercise tolerance was greatly reduced in every animal, somewhat more severely in the animals which were failing or eventually failed (fig. 8). Endurance at this time ranged from 2 to 25 minutes. The animals which never developed heart failure manifested a steady return of

exercise capacity, until at the end of four months they could run about as well as before the block (fig. 8). In contrast the animals which were failing or ultimately failed had no such restitution of exercise tolerance, which remained low until death (fig. 8).

The pulse was counted before and after each treadmill determination. Rate usually increased 10 to 20 beats per minute during the run. The femoral pulse became much more forceful during and after exercise.

The Effect of Chronic Heart Block on Cardiac Output

Prior to surgery, cardiac outputs were obtained in nine of the dogs, using a Nembutal dose of 27 to 30 mg. per kilogram. After four weeks of complete heart block, cardiac outputs were again obtained under the same anesthetic conditions. Despite large stroke volumes of from 40 to 80 cc. the outputs were generally reduced both in the animals which failed and in those which remained compensated (fig. 9). At the end of eight weeks, cardiac output studies were

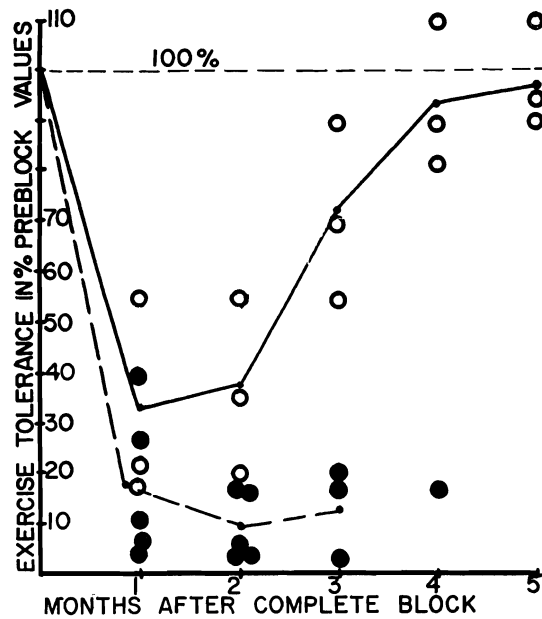


FIG. 8. Exercise tolerances in dogs with complete heart block, expressed in per cent of preoperative treadmill times. Points shown by solid circles are from animals which developed heart failure, and points shown by open circles are from animals which did not decompensate.

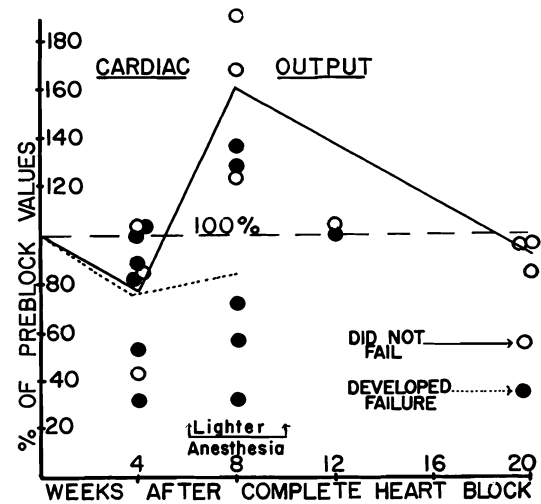


FIG. 9. Cardiac output in animals with complete heart block expressed in per cent of preoperative values (each animal serving as his own control). All pre- and postoperative determinations were done with 27-30 mg. per kilogram, Nembutal except the eight week tests which were done with 20 to 23 mg. per kilogram. With the lighter anesthesia note the inability of the animals which were failing or later failed to increase their output as markedly as the compensated animals.

again done, this time with 23 mg. per kilogram of Nembutal, an anesthetic alteration which in normal animals would be expected to increase the minute output of the heart. Under these new conditions, the animals which did not fail responded with large increases in cardiac output (fig. 9). The ventricular rates under the two levels of Nembutal anesthesia were about the same in any animal, so the rises in output were primarily due to stroke volume increases. Dogs which were failing or eventually failed either had output gains which were smaller than those of the compensated animals, or had no increases at all (fig. 9).

The Effect of Chronic Complete Heart Block on Left Ventricular Coronary Flow, Efficiency, Oxygen Consumption, and Work

In previous studies on the early effect of complete heart block in dogs, it was demonstrated that changes in coronary flow and left ventricular oxygen consumption rather closely paralleled alterations in the generally reduced left ventricular work, with little consequent alteration in calculated myocardial efficiency.¹ In the present study, similar determinations were done in eight dogs. Preoperative left ventricular weights, for use in the calculation of efficiency and left ventricular oxygen consumption, were estimated by the method of Goodale and Hackel.² Because hypertrophy developed after heart block, the measured postmortem weights of the left ventricular were used for all postoperative computations.

The results in the present series were similar to the acute studies, with two noteworthy exceptions which can be shown most clearly by demonstrating the interrelationships in a single case (fig. 10). In contrast to the earlier studies,¹ the left ventricular work in the present series returned to preblock levels in some cases. When this occurred, and the animals were not in congestive failure, the coronary flow and left ventricular oxygen consumption were reduced in relation to the work done with a resultant increase in myocardial efficiency (fig. 10—see values at four weeks), a change probably explicable on the basis of the slowed rate.^{10, 11} In the animal shown, heart failure subsequently

developed. With the onset of failure, coronary flow and left ventricular oxygen consumption increased despite a relatively unchanged work load leading to a marked fall in myocardial efficiency. All failing animals studied in the present series were characterized by declining efficiencies, a finding which is in agreement with considerable previous information on decompensation in heart-lung preparations^{10, 12} and in intact organisms.^{13, 14}

Effect of Chronic Heart Block on Heart Weight

At autopsy the total heart weights were measured by the method of Herrmann¹⁵ in 10 of the 11 dogs followed chronically. All animals had been blocked for at least six weeks, and the oldest had been blocked for 10 months. The total heart weights were greater by 5 to 50 per cent than predicted on the basis of body weight

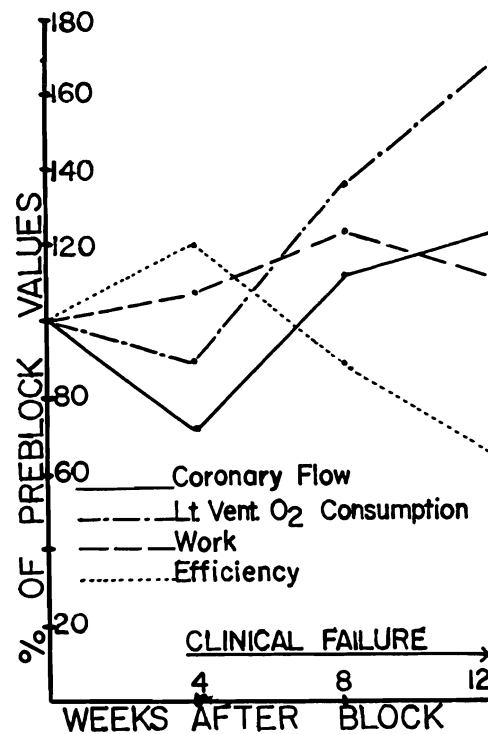


FIG. 10. Left ventricular coronary flow, work, oxygen consumption, and efficiency before (four week determination), and after the onset of congestive heart failure (8 and 12 week determinations). Note the progressive fall in efficiency after decompensation, from a level which was initially higher than obtained in the preoperative control.

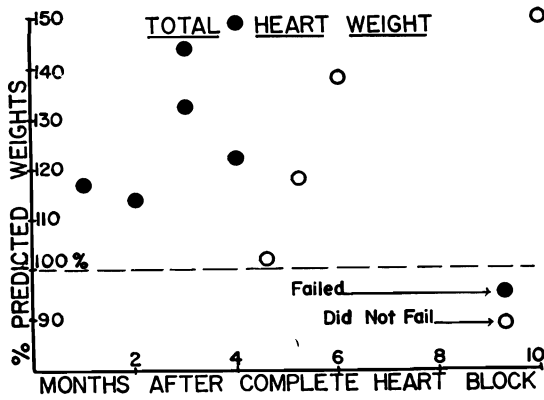


FIG. 11. Total heart weights of dogs with complete heart block, in per cent of values predicted by method of Herrmann. Note absence of correlation between the degree of hypertrophy and the presence or absence of heart failure.

with the Herrmann coefficient (fig. 11), a hypertrophy somewhat less extreme than described by Erlanger and Blackman.¹⁶ The degree of hypertrophy, within the time limits of six weeks to 10 months, was not clearly related to the longevity of the block, and was not significantly related to the presence or absence of heart failure (fig. 11).

The ventricles were then measured after dissection by the method of Goodale and Hackel,² and in every case left ventricular weight was greater than expected from the Goodale-Hackel coefficient.

Finally, in order to gain an idea of the relative hypertrophy of the different cardiac chambers, further division was carried out by Herrmann's technique in five hearts, and the weights compared with those expected from Herrmann's predictions. Both ventricles were found to be hypertrophied (fig. 12), the left slightly more than the right. The weight ratios of left to right ventricles were somewhat increased also (fig. 12), suggesting a slight predominance of left ventricular hypertrophy. The auricles, incidentally, were also increased in weight.

Postmortem Findings

Postmortem examinations were conducted on all animals immediately after death, excluding the head in all but two cases. Microscopic

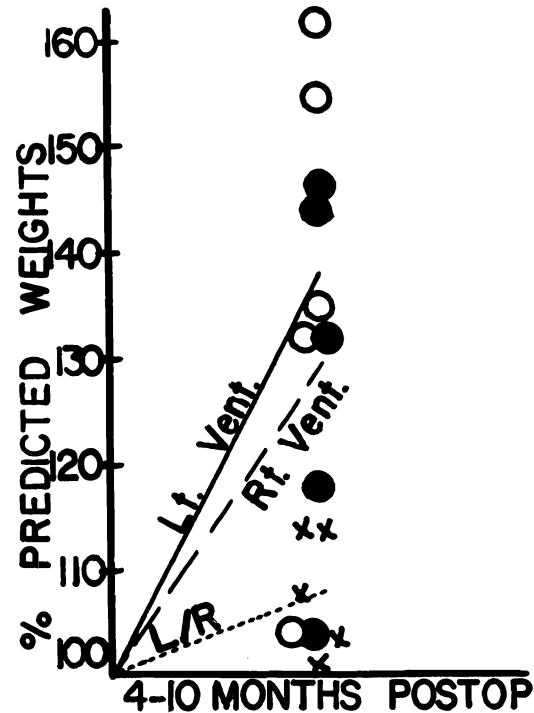


FIG. 12. The ratio of left to right ventricular weight (L/R) in per cent of the values predicted by the method of Herrmann. The changes in left (Lt. Vent.) and right ventricular (Rt. Vent.) weight are also shown. Solid circles represent left ventricular weights, crosses represent L/R ratios, and open circles represent right ventricular weights.

sections* were made of the lungs, heart, liver, kidney, adrenals, small bowel, aorta, and in two dogs (both of which were decompensated) the pituitary. The positively abnormal findings, which histologically were limited to the liver and lungs, are summarized in table 1.

Particular care was taken, in the gross examination of the hearts, to rule out the possibility that valvular damage, unrealized septal defects, or postoperative constrictive pericarditis might have played a role in the production of the symptoms and signs of congestive heart failure. None of these factors could be implicated. Pericardial adhesions were filmy or absent. No septal defects were present. All valves were found on testing to be functionally competent, and indeed no changes could be detected in any valve except the tricuspid which

* Histologic examinations were done by Dr. William Rienhoff, III.

TABLE 1.—*Gross and Microscopic Changes in Animals with Complete Heart Block. All Chronic Animals had, in addition, Generalized Cardiac Hypertrophy. Specific Mention is Made only of Positive Findings*

No.	Duration of Complete Heart Block	Gross	Microscopic	Remarks
1	12 weeks	100 cc pleural effusion; 900 cc ascites; pulmonary edema; enlarged congested liver	Diffuse pulmonary edema with many hemosiderin-laden macrophages in alveoli; chronic passive congestion of liver	Dog had terminal acute pyelonephritis
2	43 weeks	Normal	Normal	Sacrificed
3	3 days	Focal atelectasis of lungs	Focal atelectasis of lung	During operation IA defect made and closed. Postop animal had signs of brain damage, probably from air embolus. No brain post done
4	14 weeks	Equivocal pulmonary edema; 100 cc ascites; enlarged congested liver	Acute and chronic passive congestion of liver	Died suddenly during treadmill run
5	6 weeks	Liver enlarged and congested	Chronic passive congestion of liver	Fibrillated during catheterization
6*	14 weeks	1200 cc mixed blood and ascitic fluid. Liver enlarged and congested. Liver had linear rupture	Intrahepatic hemorrhage. Acute and chronic passive congestion of liver	Died during treadmill run
7	8 weeks	550 cc pleural effusion; pulmonary edema; 2050 cc ascites; large congested liver	Pulmonary edema; Chronic passive congestion of liver	Fibrillated during catheterization
8	22 weeks	Normal	Normal	Sacrificed
9	10 weeks	50 cc pleural effusion; 200 cc ascites; extensive retroperitoneal edema; pulmonary edema; large congested liver	Patchy pulmonary edema with hemosiderin laden macrophages in alveoli; chronic passive congestion of liver	Sacrificed
10	12 weeks	40 cc pleural effusion; 900 cc ascites; pulmonary edema; enlarged congested liver	Many hemosiderin-laden macrophages in lung but no demonstrable edema fluid; chronic passive congestion of liver	Sacrificed
11	21 weeks	Normal	Normal	Sacrificed
12	18 weeks	Normal	Normal	Sacrificed

* Failure precipitated by daily treadmill exercise.

in some animals had minor scarring at the point where the intracardiac incision had been made.

The four animals which had not developed clinical or laboratory signs of heart failure had no pathological abnormalities except for the hypertrophy noted above. The seven animals which had decompensated all had unmistakable changes (table 1). All seven had chronic passive congestion of the liver, characterized grossly by enlargement and nutmeg appearance on sec-

tion, and microscopically (fig. 13) by central lobular atrophy, distention of the sinusoids, and varying degrees of fibrosis. Five had significant ascites. Four of the dogs had small or moderate pleural effusions. In five cases gross pulmonary edema was seen. Microscopically, hemosiderin-laden macrophages were present within thick-walled pulmonary alveoli in four animals, and in three cases pulmonary edema fluid was unequivocally present (fig. 13).

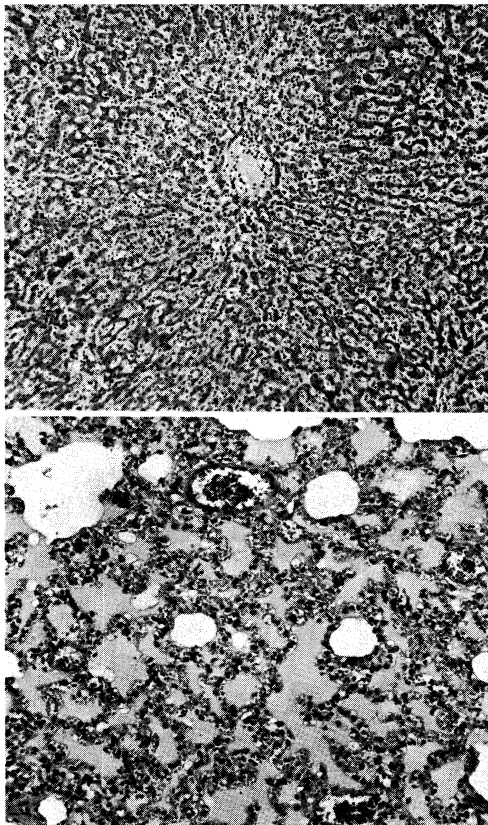


FIG. 13. Sections of the liver (upper) and lung (lower) showing central lobular atrophy and pulmonary edema. (liver (upper) $\times 75$ lung (lower) $\times 150$).

DISCUSSION

Chronic heart block in dogs is a preparation of experimental utility from several points of view. It affords a means of studying bradycardia. Blocked hearts are parasympathetically denervated as far as rate control is concerned,^{1, 17} although sympathetic stimulation can cause minor rate increases.¹⁷ Since in this and other respects the situation is more like a Starling heart-lung preparation than is the case in the intact animal, it is possible to obtain accurate information on *in vivo* stroke volume adjustments to alterations in rate^{1, 18} and output demand. Complete heart block shortly induces a generalized, although slightly asymmetrical hypertrophy. Finally, it is a preparation which leads to right and left sided congestive heart failure. In this series, decompensation occurred in the majority of animals,

and it is felt that if the animals had been exercised during the first few postoperative weeks most or all would have failed.

There is much evidence that the congestive heart failure is specifically due to the conduction defect (and the resultant bradycardia) rather than to any nonspecific effect of the operative procedure. At autopsy, essentially no deformation of normal anatomy could be demonstrated either within or around the heart. "Sham" operations were followed by quick and uneventful recoveries. Finally, in the classic work by Erlanger and Blackman,¹⁶ chronic heart block in dogs produced by a different technique likewise resulted in several instances of heart failure.

In several respects, chronic heart block presents a uniquely advantageous situation for the study of events of low output cardiac failure. There is a discrete onset of reduced cardiac output^{1, 5} following which a considerable time intervenes before large elevations occur in central venous pressure and before clinical signs of decompensation become manifest. The excessive and inadequately managed stroke work load is not regionally imposed upon selective areas of the heart as shown by the general hypertrophy and the evidence of both systemic and pulmonary vascular congestion. Changes in the myocardial metabolism, in the direction of impaired myocardial efficiency, are in accord with the classic concept of cardiac decompensation.^{10, 12}

Space does not allow a detailed comparison between complete heart block in dogs and in man, although general clinical opinion indicates that this entity is better tolerated in humans than might be inferred from the present study in dogs. However, Erlanger's analysis of the striking similarity of this condition in the two species¹⁷ suggests that the present data in dogs may have some application to the analogous human syndrome, at least in arriving at a better understanding of the directional changes which occur with bradycardia.

SUMMARY

A method has been described for the surgical production of chronic complete heart block in

dogs. This consists of incision of the region of the bundle of His through the open right auricle during temporary occlusion of the vena cavae. Exercise tolerances, chest x-ray films, electrocardiograms, phonocardiograms, cardiac outputs, intracardiac pressures, femoral pressures, and left ventricular coronary flows were obtained preoperatively and from 1 to 10 months postoperatively. The majority of the animals developed clinical, laboratory, and pathological evidence of heart failure either spontaneously or after a period of enforced treadmill exercise. The congestive failure was characterized by an elevated central venous pressure, reduced cardiac output, falling myocardial efficiency, hepatomegaly and cardiac cirrhosis, ascites, pulmonary vascular congestion, and pulmonary edema. All animals had generalized myocardial hypertrophy.

ACKNOWLEDGMENT

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SUMMARIO IN INTERLINGUA

Es describe un methodo pro le production chirurgic de chronic bloco cardiac complete in canes. Le methodo consiste in executar un incision in le region del fasce de His a transverso le aperite auricula dextere durante le occlusion temporari del vena cave. Le sequente studios laboratorial esseva executate ante le operation e a periodos de inter 1 e 10 menses plus tarde: tolerantia a exercitios, roentgenogramma thoracic, electrocardiogramma, phonocardiogramma, rendimento cardiac, pression intracardiac, pression femoral, e fluxo ventriculo-coronari sinistre. Le majoritate del animales disveloppava signos clinic, laboratorial, e pathologic de dysfunctionamento cardiac, o spontaneemente o post un periodo de exercitio fortiate. Le dysfunctionamento congestive esseva caracterisate per le sequente tractos: elevate pression venose central, reduction del rendimento cardiac, abassate efficacia myocardiac, hepatomegalia e cirrhosis cardiac, ascites, congestion pulmono-vascular, e edema pulmonar. Omne le

animales habeva generalisate hypertrophia myocardiac.

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