

A Study of Methods and Their Pathological Changes for Surgical Intervention of Atrial to Ventricular Conduction in Dogs

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Introduction

Surgically induced heart block is nearly always the result of direct trauma to the conduction bundle (MC GOON et al., 1964¹⁴). The threat of its inadvertent occurrence during cardiac surgery increases the demand on the surgeon for precise anatomic orientation and surgical technique. However, when permanent block does develop as the result of surgery, it should be treated in most instances by permanent electrical stimulation of the heart (FURMAN, 1977⁵).

Anatomically the atrio-ventricular node (AV node) is situated in the inferodistal aspect of the atrial septum and the His bundle begins as a continuation of the AV node near the right fibrous trigone. (LEV, 1964¹²) OTSUKA and KAWAMURA, 1974¹⁶) (Fig. 1)

Previous methods of experimental production of permanent heart block have included:

1. Mechanical crushing (ERLANGER and BLACKMAN, 1910;³) MEAKINS, 1913¹⁵).
2. Local injection of formalin or iodine in alcohol into the tissue surrounding the His bundle (ERLANGER and BLACKMAN 1910;³) STEINER, 1968²¹).
3. Ligation of tissue near the AV node (TAUFIC et al., 1954;²²) FOLKMAN and WATKINS, 1958⁴).
4. Electrocautery of the AV node and His bundle (WIEBERDINK, 1966;²³) SEALY, 1977¹⁹).
5. Heat coagulation around the His bundle (PRUETT, 1967¹⁷).
6. Surgical transection or excision of the His bundle (STARZYL and GARTNER, 1955²⁰).
7. Septal infarction by occluding the septal artery (HASHIBA, 1967⁸).
8. Freezing of the AV node-His bundle (HARRISON, 1977⁷).

These intervention have been associated with variable success in achieving permanent A-V block. Our study was designed to devise a safe and dependable surgical method of

Key words : Complete A-V block, His bundle, Heat coagulation, Soldering gun, Left ventricular pressure.

索引語 : 完全房室ブロック, ヒス束, 熱凝固, ハンダゴテ, 左室圧.

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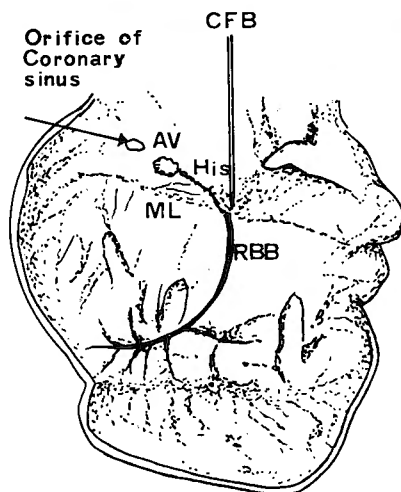


Fig. 1 Network of Purkinje fibers in subendocardium of right ventricle by modified PAS procedure (above) and its diagram (below) in dog.

AV=A-V node, His=His bundle, RBB=Right bundle branch, CFB=Central fibrous body, ML=Medial leaflet of tricuspid valve (excised)

acute and chronic heart block in dogs to be used for further investigations.

Materials and methods

Experiments were performed in 82 mongrel dogs of both sexes weighing 8 to 28 kg. They were anesthetized with intravenous Nembutal (25-30mg/kg). Each dog was ventilated with room air via an endotracheal tube attached to a Harvard pump respirator (20 ml/kg). A control electrocardiogram was recorded continuously. The chest was then opened via a right thoracotomy through the 4th intercostal space. The lung was retracted

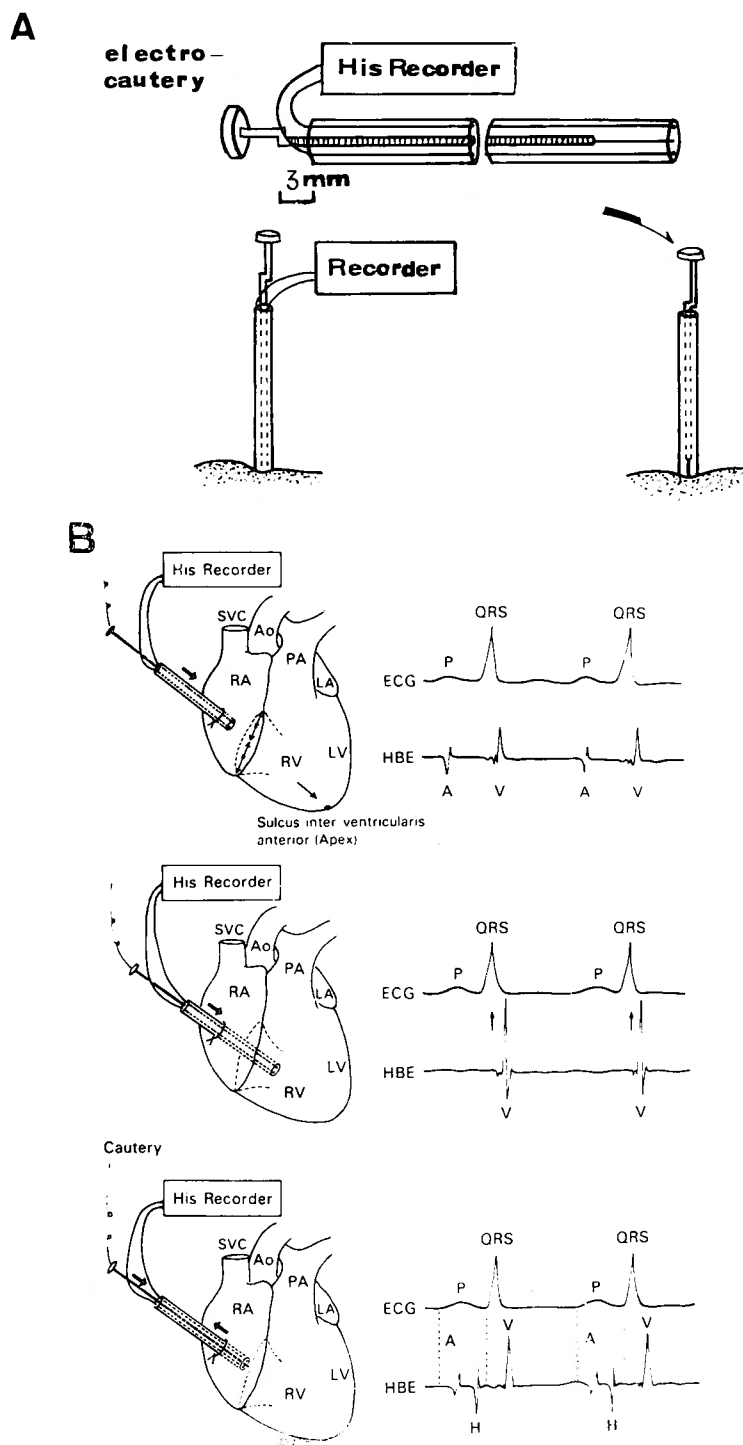


Fig. 2 A) Diagram of the intracardiac His recorder with electrocautery¹⁹⁾ As the His bundle was identified, the electrocautery was pushed into the area, and it was cauterized for 5 to 10 seconds.

B) His bundle identification in atrium, ventricle and His bundle.

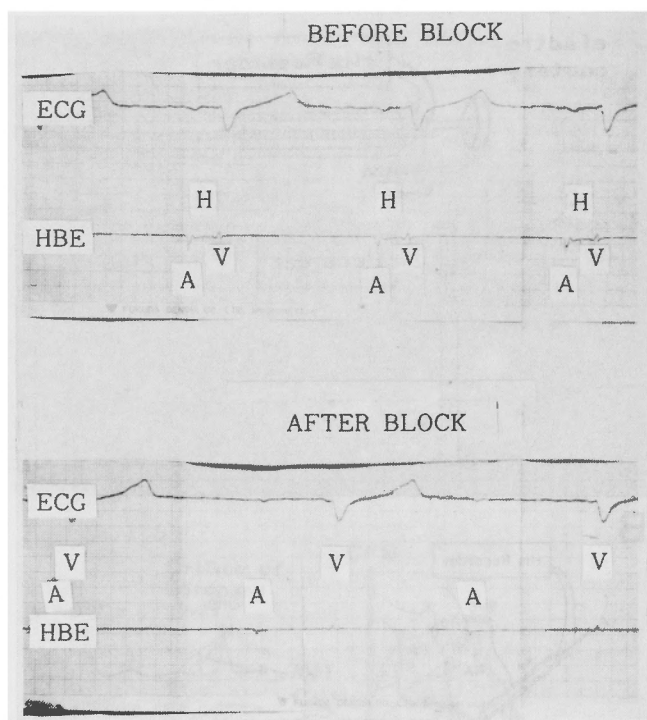


Fig.13 Electrocardiogram and His bundle electrogram (HBE) before and after heart block.

posteriorly and the pericardial sac opened in front of the phrenic nerve and retracted with stay sutures. Carotid arterial pressure was recorded through a fluid-filled polyethylene catheter. Left ventricular pressure (LVP) was measured with a Konigsberg's transplantable microtransducer (Model p-16, 6.5 mm in diameter) which was inserted directly into the left ventricle through an incision wound at the apex.

The transducers were connected to a SAN-EI polygraph (model PH 41-6) which gives a continuous display and can record when necessary. Central venous pressure measurement was set up through the external jugular vein. Cardiac output was measured by Statham's electromagnetic flow meter (model SP 201) with its probe placed around the ascending aorta.

We first tried to interrupt the His bundle via a closed heart technique by using the electrocautery within a His bundle detector probe (Dr. SEALY's method)¹⁹⁾. The probe was inserted into the right atrium through a purse string suture. The cautery point could be inserted through a sheath which had a bipolar electrogram probe at its end. (Fig. 2A) The position of the His probe could be guessed by electrocardiogram (ECG) and His bundle electrogram (HBE) as shown in Fig. 2B. After the His bundle was identified on the electrogram, the electrocautery probe with a 3mm long exposed point was inserted through the sheath, and the area was cauterized for 5 to 10 seconds or more until block was produced (Fig. 3). A lead II ECG and reference electrograms from the atrial (with a clip

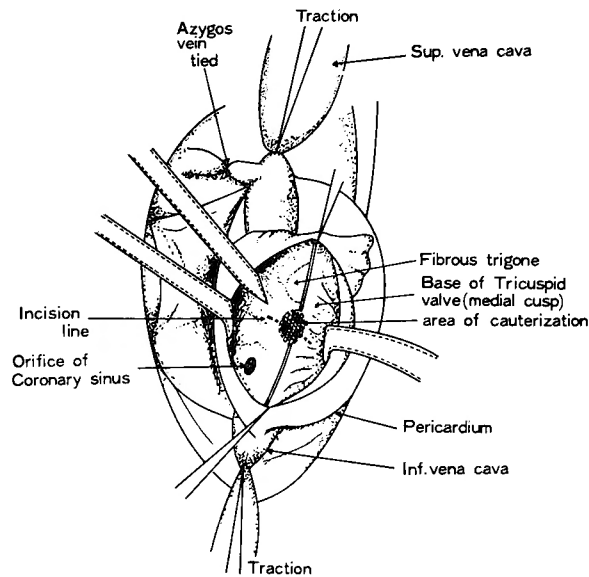


Fig. 4 Artist's view of the technique of surgical production of complete heart block.

type electrode applied to the right and/or left appendage) and right ventricular wall were recorded simultaneously to confirm the fact that the sharp spike was due to the His electrogram and to make sure of A-V dissociation and block. The relationship of the His electrogram to the ECG is shown in Figs. 2 & 3.

In the next series of His interruption experiments, an incision was made above the annulus of the tricuspid valve beginning at a point beneath the coronary sinus and extending to the right fibrous trigone (Fig. 4). The right atrial wall isolated by a curved vascular clamp was incised. The azygos vein, the superior vena cava and the inferior vena cava were occluded by traction with braided silk, and the right atrium was entered. After releasing the curved clamp at the right atrial wall, residual blood in the right side of the heart and continuous drainage from the coronary sinus was sucked out and the His bundle was incised. In acute experiments, the occlusion was no longer than 180 seconds with less than 60 ml of blood loss. If A-V block was not produced by the first incision, a second occlusion-incision attempt was undertaken fifteen minutes later. When block was achieved the atrium was sutured closed and a demand pacemaker set at 110 beats per minute was prepared for use if necessary.

The third method we tried was electrocauterization around the AV node-His bundle. A Neo-med solid state electrosurgery model 3000 (15 VAC 50/10 Hz 15A) was used. Electrocauterization was performed at the middle portion of the base of the septal cusp and usually extended for a short distance about 2 to 5mm into the contiguous region to the atrium and the ventricle (Fig. 4).

The fourth method we used was a soldering gun (2 mm in diameter of its tip) in-

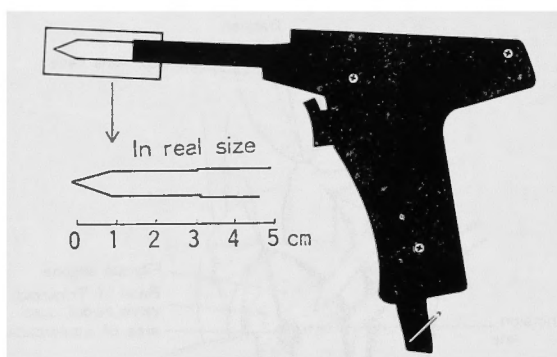


Fig. 5 Soldering tip for heat cautery. $d=2\text{mm}$

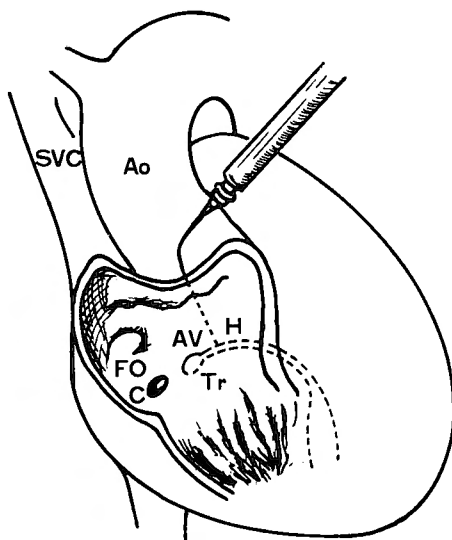


Fig. 6 Diagrammatic representation showing the technique and placement of the needle.
H : bundle of His, C : coronary sinus (orifice), FO : fossa ovalis, AV : AV node,
Tr : septal leaflet of tricuspid valve.

stead of the electrocautery probe (Fig. 4 & 5). The temperature of the soldering tip could be as high as 400°C . The technique was the same as used in the second method. After identification of the suspected location of the His bundle under right atriotomy, the area of the AV-His connection was coagulated for seconds until heart block ensued.

The fifth method was a surgical cryothermia unit (Takeda Cryobar model CS 45-3) instead of the electrocautery probe. Expansion of nitrous oxide within the probe tip (5mm in diameter) resulted in cooling. With the cryothermia unit, the area of the AV-His connection was frozen to -60°C for two minutes; and then the tip was rewarmed to remove it.

The sixth method was injection of 40% formaldehyde (0.1 to 2.0 ml) into the His bundle (Dr. STEINER's technique; Fig. 6)²¹.

After successful heart block had been produced, the hemodynamic effect was recorded. In acute studies, the dog was sacrificed and the pathological changes of the conduction system were studied.

Results

In this study, we investigated the degree of success of different methods, the problems of the methods, the hemodynamic changes before and after heart block and the gross and microscopic pathological changes.

The degree of success of different methods:

In our study, six kinds of surgical intervention were associated with variable degrees of success in achieving permanent A-V block (Table 1).

The first method was electrocautery within the His detector probe which caused complete heart block in only 40% (8/20). The major causes of failure were acute heart failure, fistula formation between the aorta and the subepicardial trigone, artificial septal defect between right and left atrium, and bleeding from the right atrium.

The pre-operative QRS duration was 0.040 second and the post-operative average was 0.091 second (Fig. 3).

With the second method, despite complete incision through the right atrial septum carried to the membranous ventricular septum the His bundle was successfully interrupted in

Table 1 Methods of surgical interruption of atrial to ventricular conduction and their rate of success

Method of A-V block	1. Intracardiac His probe with electrocauterization	2. Incision	3. Electrocauterization	4. Heat coagulation	5. Freezing	6. Local injection
No. of dogs	20	10	9	23	10	10
Successful block	8(40%)	7(70%)	7(78%)	23(100%)	6(60%)	8(80%)
Transient block	4	2	1	0	2	1
Failure	8	1	1	0	2	1
Reversion to sinus rhythm	3	1	0	0	1	0
Other arrhythmias	1	1	1	0	1	1
Complications	8	4	2	0	4	3
a) Acute heart failure	4	1	1	0	3	1
b) Bleeding	2	1	1	0	0	1
c) Fistula or septal defect	2	2	0	0	0	0
d) Others	0	0	0	0	1	1

only six of ten dogs. Careful review at autopsy revealed that the incision was too high and too anterior to cut through the conduction system.

The third method (direct electrocauterization after inflow occlusion) was successful in seven out of nine dogs. The causes of failure were acute heart failure, bleeding after repeated trying and impaired general condition.

Heat coagulation had a 100% success rate (23/23). Although destructive of tissue, it preserved the connective tissue network. No fistula or residual septal defect was produced. In nearly all of the dogs (22/23) complete heart block occurred at the first attempt. There were no serious complications.

The fifth method, cryothermia, resulted in complete A-V block in only six out of ten dogs (60%). In the others block was transient and/or acute heart failure developed after several attempts.

The sixth method, injection of 40% formaldehyde into the His bundle, produced complete heart-block in eight of ten dogs. One of the eight heart-blocked dogs developed myocardial damage due to injection of excess formalin into the ventricular septum.

Chi Square tests of the success of the fourth method (heat coagulation) in relation to the other methods revealed significant difference (a p value of less than 0.05 was taken as indicative of statistical significance) (BAHN, 1972)¹⁾.

Electrographic confirmation of the block was obtained (Fig. 3). In no case did a ventricular rhythm fail to occur after the A-V block.

The resulting preparation was a very stable one. The heart rate, cardiac output, arterial and venous pressure, dp/dt and left ventricular pressure were monitored.

Hemodynamic change:

a) Effect of complete heart block on cardiac output

In all 51 dogs with successfully produced permanent complete heart block, the cardiac output or cardiac index was reduced. The magnitude of the decline was related to the idioventricular rates which ranged from 25 to 110 per minute. The majority had rates from 40 to 55 per minute, and in this group the cardiac output fell quite regularly to about one half of the pre-operative value.

The foregoing data provide strong evidence that the decrease in the cardiac output was due specifically to the bradycardia resulting from complete heart block. To further rule out the possibility of non-specific effects due to the operative procedure, cardiac pacing (ventricular pacing in demand mode) at the pre-block heart rate was performed. Such repetitive stimulation restored the cardiac output to about 90% of the levels present before the block in most cases. In a few cases, the cardiac output did not return to the pre-block value because of myocardial dysfunction (due to injection of formaldehyde into the ventricle) or heart failure (excess bleeding and impaired general condition). The state of reduced output after complete heart block is quite stable in the first few hours.

b) The effect of complete heart block on arterial pressure

During occlusion of the venae cavae and right atriotomy, the arterial pressure dropped

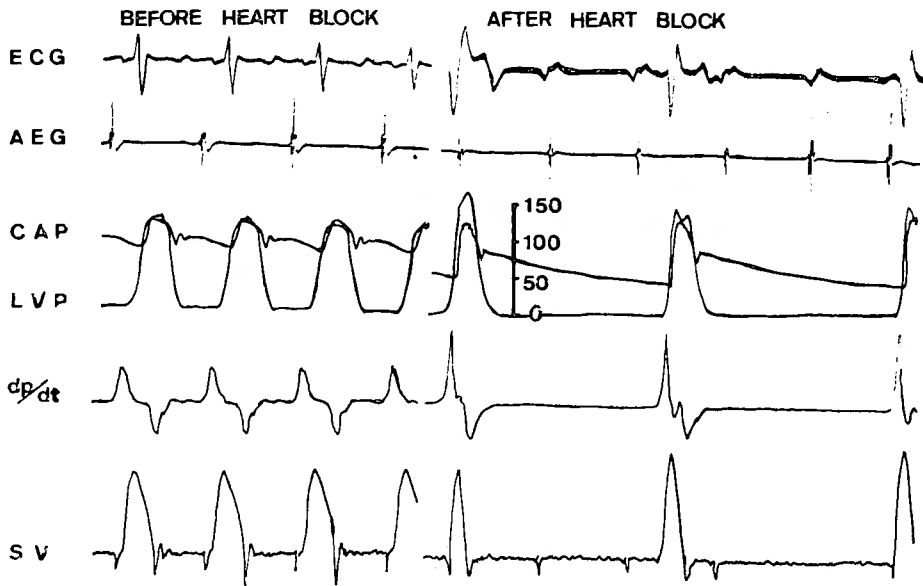


Fig. 7 Hemodynamic change before and after complete heart block. AEG : atrial electrogram, CAP : Carotid arterial pressure, LVP : Left ventricular pressure, SV : stroke volume.

sharply from an average 150 to 40 mmHg. Small pulses continued to appear in the arterial tracing.

After closure of the atrium and release of the venae cavae, strong arterial pulses immediately reappeared (Fig. 7) with idioventricular rates of 25 to 110 per minute. There was, in general, a transient period of hypertension, presumably due to the release of catecholamines.

A few minutes after complete block was established systolic pressure usually stabilized at about the preoperative level. The diastolic pressure at the slowed rate was considerably reduced, causing a wide pulse pressure. Thus, the mean arterial pressure was reduced. The decline being roughly proportional to the reduction of the rate.

c) Effect of complete heart block on left ventricular work

Left ventricular work was computed from cardiac output times mean carotid pressure. The calculation ignores the work done in imparting velocity to blood, a factor which is ordinarily lower than two percent of total work (BROCKMAN, 1965)²⁾. This kinetic factor may increase with the pronounced bradycardia and large stroke volume seen with heart block. So it is possible that the work as calculated was falsely lower by about 5 to 10 percent. Acutely, left ventricular work was diminished to 40% of the pre-block value.

d) Effect of complete heart block on central venous pressure

There was small elevation of central venous pressure ranging from 0.5 cm to 3.0 cm H₂O. The augmentation of stroke volume mediated by atrial contraction through the Starling mechanism occurred only in beats in which the A-V intervals fell by chance within

the physiologic range. This caused a phasic variation in stroke output that was reflected in changing heights of the arterial pressure waves and central venous pressure.

e) Factors influencing heart rate after complete heart block

The heart rates after complete heart block was reduced to about 40 to 50% of the pre-block rate with a median value of 52 per minute. Two dogs had heart rates lower than 30 and three had heart rates higher than 65.

Several other factors may influence idioventricular rate : vagal activity, the catecholamines, extension of the lesion, pre-block heart rate, manipulation of preparation (i. e. pacing electrodes, microtransducer, etc.), block producing procedures (time of inflow occlusion, blood loss, times of re-entry attempt and others) and overdrive suppression of cardiac pacing, etc..

Pathological changes :

At death, the atrial and ventricular septal areas were removed and sectioned for microscopic study. Tissue sections were 5 μ thin, step sectioned through the AV node, His bundle and proximal bundle branches (LEV, 1958)¹²⁾. These sections were stained with hematoxylin and eosin. No other stains are necessary for identification.

On gross examination, these specimens had a right atrial sutured-lesion. In seven of them, (7/53), the epicardial surface of the right atrium was slightly to moderately burned.

In the cauterization groups (methods 1, 3 and 4), gross examination revealed an area of cauterization extending over the atrial septum, tricuspid valve leaflet and both the membranous and muscular septum. In the microscopic sections, the His bundle was identified at the upper edge of the septum (GLOMSET & GLOMSET, 1940,⁶⁾ JAMES & SHERF, 1971)⁹⁾.

Macroscopic endocardial changes involved only the area of cauterization, which looked burned. In two of these animals a small mural thrombus was present over the area of probe placement.

Microscopically, the lesion showed necrosis of myocardial cells and conduction fibers, a polymorphonuclear leucocytic infiltration and marked hemorrhage in the periphery of the lesion (Fig 8).

The lesion generally involved the His bundle and proximal bundle branches but frequently spared the left bundle branch entirely. Sometimes the lesion of cauterization extended so far and deep that the AV node and/or the His bundle could not be identified.

In two of the animals the septal leaflet of the tricuspid valve was burned and moderately to severely edematous.

No aortic wall changes was seen in any of these groups.

Two dogs that were not subjected to A-V block treatment served as controls. They underwent a right thoracotomy. The right atrium was opened and the soldering tip set at room temperature was held on the His bundle area for 30 seconds. No evidence of A-V block was found clinically. These animals were sacrificed immediately. Their A-V conduction systems, prepared and examined in a similar fashion, were investigated. One of them



Fig. 8 Heat coagulation of the A-V node-His bundle area revealed marked burned and necrosis of conduction fibers, polymorphonuclear leucocytic infiltration and marked hemorrhage. (HE stain, $\times 100$)

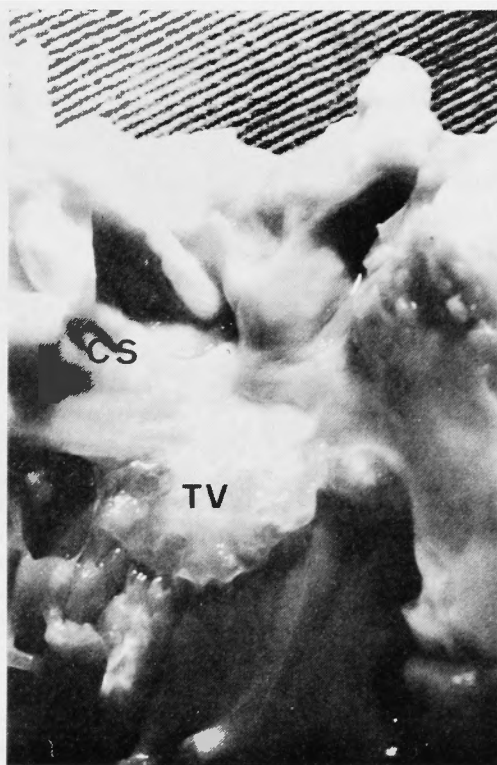


Fig. 9 Photograph of the area of heat coagulation in a dog sacrificed two years later. The area of coagulation had scar formation with good demarcation. Tricuspid valve was not involved.
TV : Tricuspid valve (septal leaflet)
CS : Coronary sinus

showed no lesion and the other a small hemorrhage near the AV node.

In the coagulation group only, two years later, the dogs were sacrificed for examination of the conduction system and the myocardium. Grossly, the area of heat coagulation had scar formation with good demarcation. Tricuspid valve was not involved. (Fig. 9). Microscopically, the endocardium showed fibrosis of myocardial cells, diffuse necrosis of the conduction cells, perivascular cellular infiltration consisted of fibroblast and fibrocytes. (Fig. 10). The myocardium showed eccentric hypertrophy.

In the incision group, endocardial changes were seen grossly only in the vicinity of the wound. Microscopically the lesion showed moderate hemorrhage and polymorphonuclear leucocytic infiltration. One dog showed artificial atrial septal defect formation and mild valvular damage.

In the freezing group, the epicardial surface was slightly hemorrhagic. The macroscopic endocardial change was a lesion 6 to 10 mm in diameter with a circular hemorrhagic focus. Microscopically, it showed destruction of myocardial cells and conduction

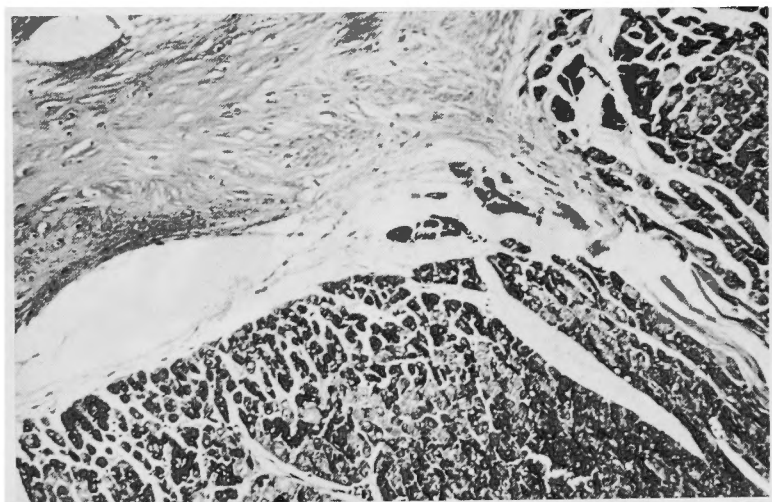


Fig. 10 Photomicrograph of heat coagulation in the AV node-His bundle area in a dog sacrificed two years after production of complete heart block and insertion of a demand ventricular pace-maker. Burned areas around the AV node separated from the atrial septum by fibrosis, strands of fibrous tissue, fibroblast and capillaries. (HE stain, $\times 100$)

fibers, a polymorphonuclear leucocytic infiltration and marked hemorrhage in the periphery of the lesion.

In the formalin injection group, the endocardial changes seen grossly involved the AV node-His bundle area and ventricular septum which was dark gray. Microscopically, there was necrosis of myocardial cells and conduction fibers, red blood cell extravasation and polymorphonuclear leucocytic infiltration.

Discussion

The dog with complete heart block is a useful preparation for the study of hemodynamics, electrophysiological changes, cardiac pacing and cardiac function.

The present study was carried out in order to find the reliable and simplest method of interrupting the atrio-ventricular conduction system in dogs to be used for further studies and to gain an outline of the natural history and hemodynamic changes in acute complete heart block.

We first tried to use a closed heart method of electrocautery within the intracardiac His probe similar to the technique described by Dr. SEALY. Although this method avoids the need for cardiopulmonary bypass, our studies led us to abandon it. Not only was there the potential for damage to the aorta, to both the A-V valves and to the ventricular and atrial septa, but also there was the cauterization were made too superficially or were done far enough away from these structure.

In our opinion, electrocauterization with a specific intracardiac His probe does not easily produce complete heart block, because of the blood filling the right atrium and the

movement of the heart. In fact, in our experiment the success rate was only 40%.

WISBERDINK used a non-penetrating cautery point applied blindly to the area of the His bundle in dogs, i.e. coagulation with diathermic probe. But in our hands this technique was difficult in a moving heart and did not consistently produce heart block.

Among the other methods that have been advocated for producing A-V block is suture ligation of the AV node-His bundle junction, but this has been associated with a high incidence of return to normal sinus rhythm in both experimental and clinical studies. (FOLKMAN et al, 1957)⁴. We did not use this method in our study.

The oldest method for the experimental production of a permanent heart block is mechanical crushing from outside of the right atrium with a special frocep (MEAKINS, 1913.)¹⁵ We did not use it in this study, either, because functional or permanent heart block is not always produced by this method. It also decreased the vigor of the heart and interfered with making records.

Suture ligation, incision and cautery are all associated with the risk of inducing septal defect, tricuspid insufficiency and aneurysms and fistulae of the aortic sinus of Valsava. As in the 2nd method used in this study, in making the anterior portion of the incision, the surgeon runs the risk of perforating the right atrial epicardium or entering the left atrium.

The major advantage of the cryosurgical method of effecting A-V block is the reversible nature of the technique: it is possible to identify the appropriate area and observe the functional effect of conduction block (at 0°C) prior to production of an irreversible state of A-V block. Also the cryosurgical ablation, although destructive, preserve the connective tissue network until scar tissue has time to form and replace it. Thus, the dangers that are associated with the electrocautery are avoided. This technique does have the disadvantage of icy adhesions among the tip, the blood and the endocardium. The period of inflow occlusion should never exceed three minutes. This makes freezing of the His bundle and rewarming of the probe impractical without cardiopulmonary bypass. Moreover, we did not know the exact extension and the depth of the frozen area at -60°C. Clinically, its reversible nature would be useful in blocking the normal or abnormal conduction system under extracorporeal circulation.

There is a simple and quick technique for the production of heart block in dogs by the injection of 40% formaldehyde into the His bundle. This approach avoids entering a cardiac chamber and requires no special apparatus. It can produce stable complete heart block with a high degree of consistency. However, repeated attempts may be needed to produce complete block, so a dog might receive more than 2.0 ml of formaldehyde which could influence myocardial function. After many attempts, residual bleeding from the site of entry of the needle in the right atrium was also noted, but it could be stopped by direct suture.

In our study, heat coagulation had a 100% (23/23) success rate in achieving permanent complete heart block. This method does have the advantage of short duration of

intervention, under direct vision, does not need a bloodless field of operation, causes few complications and produces a permanent, not a functional, A-V block.

Although clinically the use of heat coagulation may have some obvious disadvantage in that heat denatures collagen, destroys fibroblast and cannot cause a reproducible and reversible state of dysfunction, it is the most reliable of the methods we tested in this experimental study.

Acknowledgement

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References

- 1) Bahn AK : Basic medical statistics, New York, Macgraw Hill Book Company, Inc. pp. 63-85, 1972.
- 2) Brockman SK : Cardiodynamics of complete heart block. *American Journal of Cardiology* **16** : 72-77, 1965.
- 3) Erlanger J, Blackman JR : Further studies on the physiology of heart-block in mammals. Chronic auriculoventricular heart block in the dog. *Heart* **1** : 177-212, 1910.
- 4) Folkman J & Watkins E : A artificial conduction system for the management of complete heart block. *Surg. Forum* **8** : 331-338, 1958.
- 5) Furman S : Cardiac pacing and pacemakers I. Indication for pacing bradyarrhythmias. *Am. Heart J.* **93** : 523-530, 1977.
- 6) Glomset DJ & Glomset ATA : A morphologic study of the cardiac conduction system in ungulates, dogs, and man. Part II : The Purkinje system. *Am. Heart J.* **20** : 677-710, 1940.
- 7) Harrison L : Cryosurgical ablation of the A-V node-His bundle : A new method for producing A-V block. *Circulation* **55** : 463-470, 1977.
- 8) Hashiba K : A-V block produced by ligation of septal arteries in the dog. *Japanese Heart Journal.* **6** : 256, 1967.
- 9) James TN & Sherf L : Fine structure of the His bundle, *Circulation* **XIV** : 9-28, 1971.
- 10) James TN : Anatomy of the A-V node of the dog. *Anatomical Record* **148** : 15-26, 1964.
- 11) Levinson DC, Guather L, et al : Hemodynamic finding in heart block with slow ventricular rates. *American Journal of Cardiology* **4** : 440-446, 1959.
- 12) Lev M : The normal anatomy of the conduction system in man and its pathology in A-V block. *Annals of New York Academy of Science.* **III** : 817-829, 1964.
- 13) Lev M, Widran J, et al : A method for the histopathologic study of the AV node, bundle and branches. *A.M.A. Archives of Pathology* **65** : 73-83, 1958.
- 14) Mc Goon DC, Ongley PA, et al : Surgically induced block. *Annals of New York Academy of Sciences* **III** : 830-834, 1964.
- 15) Meakins J : Experimental heart block with A-V rhythm. *Heart* **5** : 281-196, 1913.
- 16) Otsuka N & Kawamura K : Anatomy, in Cardiac Conduction System, 1st ed., Edited by Sano TI., Igaku Shoin Ltd., pp. 1-50. (in Japanese) 1974.
- 17) Pruett J : Technique for experimental heart block, *Journal of Applied Physiology.* **22** : 830-832, 1967.
- 18) Samet P, Jacob W, et al : Hemodynamic sequelae of idioventricular pacemaking in complete heart block, *American Journal of Cardiology* **11** : 594-599, 1961.
- 19) Sealy WC : A study of methods for surgical interruption of the His bundle. *Journal, of Thoracic*

- and Cardiovascular Surgery, 73 : 424-430, 1977.
- 20) Starzyl TE & Garther RA : Chronic heart block in dogs : A method for producing experimental heart failure. Circulation 12 : 259-270, 1955.
- 21) Steiner C : A simple technique for production of chronic complete heart block in dogs. Journal of Applied. Physiology 25 : 631-635, 1968.
- 22) Taufic M, Bashour FA & Lewis FJ : Production of heart block under direct vision. Surgical Forum 5 : 96, 1954.
- 23) Wieberdink J : Experimental production of permanent heart block (total or bundle branch block) without circulatory arrest or extracorporeal circulation. Thorax 21 : 401-404, 1966.

和文抄録

実験的完全房室ブロックの作成方法とその 病理学的変化の研究

京都大学医学部外科学教室第2講座（指導：日笠頼則教授）

高 欽 澤

82匹の雑種成犬を用いて下記の方法による心房心室刺激伝導系切断を行った。即ち

1. 改良式心臓内ヒス束プローベを以てする電気焼灼術
2. ヒス束切断
3. 電気メス焼灼
4. ハンダゴテによる熱凝固
5. ヒス束冷却
6. 40%ホルマリン局所注入

これらの手術手技とそれによるブロック成功率並びに病理学的変化を比較検討し併せてブロック前後の血行動態にも検討を加えた。

ブロック発生に関しては熱凝固の成功率が一番高く

(23/23, 100%), 次はホルマリン局所注入 (8/10, 80%), 電気メス焼灼 (7/9, 78%), ヒス束冷却 (6/10, 60%), 改良式ヒス束プローベ電気焼灼 (8/20, 40%) の順であった。

ブロック術後は、当然のことながら、毎分40~55の固有心室調律を示し、心拍出量及び左心仕事量の低下がみられた。その他拡張期血圧低下による脈圧の増大、中心静脈圧の上昇傾向が認められた。

病理学的変化としては心筋細胞と伝導線維の壊死、多核球の浸潤と種々の程度の出血が認められた。

結論として実験的完全房室ブロックの作成はハンダゴテによる熱凝固法が最も確実であり合併症は殆んどみられず、最もよい方法と考えられる。