Selective Roentgenographic Contrast Examination and Electrokymography of the Left Heart in Experimental Mitral Insufficiency

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Difficulty in the accurate preoperative assessment of mitral insufficiency, clinically and by several laboratory methods, suggested the desirability of direct visualization of the regurgitant flow. Direct puncture of the left ventricle for the injection of radiopaque contrast material was used in dogs before and after the creation of mitral insufficiency by division of 1 chorda tendinea. The regurgitation was accurately recognized radiographically. Consistent alterations in left atrial border movement were also recorded by electrokymography.

Until recently, attempts to improve the preoperative recognition of mitral insufficiency have been concerned with the correlation of observations on physical examination, electrocardiography, roentgen examination, electrokymography, angiocardiography, and right heart catheterization with the findings at surgery when the valve was palpated. Lately, left heart catheterization by left atrial puncture has been introduced, but it seemed to us that an even more direct sign of mitral insufficiency was needed—visualization of the regurgitant flow itself. This possibility was originally demonstrated by Nuñez and Pons-dornenech in 1 patient, later confirmed in patients by Smith and associates, and recently extensively studied in dogs by Wilder's group.

For this purpose a technic for the selective introduction of contrast material into the left ventricle of the intact animal by controlled percutaneous puncture has been developed. The verification of experimentally created mitral insufficiency by this means has provided an opportunity to observe corresponding alterations in the electrokymogram. Electrokymographic studies on patients with mitral disease have been described and analyzed extensively, but no account of the changes produced by pure experimental mitral insufficiency has been available.

In a series of 8 dogs an attempt was made to create mitral insufficiency by division of 1 of the chordae tendineae. In the 5 surviving dogs preoperative and postoperative left ventricular cardioangiography and electrokymography were used to study the effects of the surgical procedure.

Methods

The animals were anesthetized with intraperitoneal sodium pentobarbital and electrokymography was carried out in lateral projection with an Elema electrokymograph* and direct writer (Mingograph 24).* Tracings were obtained with electrocardiographic control over the superior, middle, and inferior portions of the left atrium and the posterior portion of the left ventricle. Then left ventricular cardioangiography was accomplished by the previously mentioned percutaneous puncture technic: 1 ml. of 70 per cent Hypaque† was rapidly injected by pneumatic syringe for serial filming at a speed of 2 per second, with electrocardiographic control.

Some time later, mitral insufficiency was produced by severing a chorda tendinea with a cutting hook that was inserted through the ventricle at first, but later more successfully through the left atrium. Successful production of mitral insufficiency was followed immediately by systolic ballooning of the atrium, a palpable systolic thrill in the atrium, and a postoperative systolic murmur.

After the 5 dogs had recovered from the surgical procedure, electrokymography and left ventricular cardioangiography were repeated. In 1 animal left atrial and ventricular pressures were also recorded.

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* AB Elema-Järnhs, Industrivägen, Stockholm, Sweden
† Provided through the courtesy of Winthrop Laboratories, New York, N. Y.
by percutaneous needle puncture through a right paraspinal approach under fluoroscopic guidance. Three of the dogs were eventually sacrificed and a fourth died of pulmonary edema and the results of the surgical and diagnostic procedures were compared with the gross pathologic findings.

**Results**

The preoperative contrast study of dog 1 showed a competent mitral valve with none of the material entering the left atrium (fig. 1A),

![Fig. 1. Dog 1. A. Preoperative, left ventricular cardioangiography in ventricular systole. The normally competent mitral valve prevented contrast material from entering the left atrium. B. Film after division of 1 chorda tendinea in ventricular systole. Large reflux into the enlarged left atrium and pulmonary veins identifies mitral insufficiency.](image)

![Fig. 2. Dog 2. Postoperative left ventricular cardioangiography after division of 1 chorda tendinea. Left atrial opacification shown in frontal projection in early ventricular diastole, and lateral projection in late ventricular systole.](image)
whereas postoperatively a large atrial reflux identified mitral insufficiency (fig. 1B).

The preoperative electrokymograms from the midportion of the posterior left atrial border and the left ventricle were normal, while postoperative tracings from the left atrium showed marked changes following the division of 1 of the chordae tendineae. Inward movement with atrial systole was prolonged and the normal inward movement during the phase of rapid ventricular ejection was relatively reduced. A late systolic peak was prominent.

In dog 2 the contrast injection clearly showed the mitral insufficiency produced by division of a single chorda tendineae (fig. 2). The material entered the left atrium and pulmonary veins. Electrokymographic alterations were essentially the same as in the first animal except that the left atrial wall moved outward throughout all portions of ventricular ejection until late ventricular systole (fig. 3). Simultaneous recording of left atrial pressure and left atrial movement showed the peak of atrial pressure to correspond with the most posterior position of the atrial wall.

The preoperative studies in dog 3 showed normal competency of the mitral valve and no abnormality in the electrokymogram. That mitral insufficiency was created was clearly shown by atrial reflux (fig. 4), but postoperative electrokymography was unsuccessful. This difficulty was attributed to the massive pulmonary edema that was visible on films and later confirmed at necropsy. One chorda tendinea had been divided.

In dog 4 no preoperative studies were made. Postoperatively the creation of mitral insufficiency was confirmed by demonstration of atrial reflux from the left ventricle. Electrokymographic curves of left atrial movement were similar to those already described. At postmortem examination 1 of the chordae was found to be divided (fig. 5). The puncture site was identified by a small scar.

Preoperative contrast injection and left atrial electrokymography in dog 5 were normal and remained so after an attempt to produce mitral insufficiency by the transventricular approach (fig. 6). Postmortem examination disclosed a normal mitral valve, but a laceration of an aortic valve cusp, which caused aortic insufficiency. Corresponding left ventricular enlargement was seen by cardiography.

Finally, experiences in a normal dog need to be described. A left atrial electrokymogram obtained prior to left ventricular puncture was normal. An error was made in the procedure of localization so that it was necessary to puncture the left ventricle several times before successful placement of the needle.

Fig. 3. Dog 2. Electrokymograms before and after production of mitral insufficiency. A. Left atrial, before operation. B. Left ventricular, before operation. C. Left atrial, after operation. D. Left ventricular, after operation. E. Left atrial, after operation, with left atrial pressure. F. Left atrial, after operation, with left ventricular pressure.
Subsequent injection of contrast material demonstrated a localized inward bulging of the lateral wall of the left ventricle (fig. 7). Although no contrast material was detected in the left atrium in the frontal projection, the lateral projection obtained about 15 minutes later clearly showed some mitral insufficiency. In contrast to the other operated dogs, the left atrium here was about normal in size. Electrokymography of the left atrium after the puncture showed persistent normal inward movement in early systole, but a high peak in late systole had appeared.

The dog was sacrificed and on postmortem examination a blood clot 2 cm. in diameter was found in the pericardial space. The mitral valve and chordae tendineae were normal except for some ecchymoses on the mitral leaflets. The mitral insufficiency in this case seems most reasonably explained on the basis of laxity of 1 of the chordae tendineae as a result of displacement of the ventricular wall by the clot.

**Discussion**

This series of experiments, which were carried out to evaluate the roentgenologic possibilities of demonstrating mitral insufficiency, is too small for extensive analysis. In an earlier study during development of a technique for controlled puncture of the left ventricle for injection of contrast material, reflux into the left atrium from the ventricle was not seen in normal dogs except for the 1 instance in which pericardial bleeding occurred. In all the animals with division of a chorda tendinea such leakage was clearly and unmistakably demonstrated. From the roentgenologic point of view left ventricular cardioangiography appears to be a most direct and reliable method for identifying mitral insufficiency. As yet no quantitation of the volume of regurgitation has been attempted.

In the presence of experimental mitral insufficiency considerable and fairly constant changes are also seen in the electrokymographic tracings obtained from the posterior border of the left atrium. These are of interest with regard to Wiggers’ earlier experimental studies of mitral insufficiency in the dog. The variations in left atrial volume that he recorded bear a striking resemblance to the movements of the left atrial border observed by electrokymography. At present it is not
possible to state how far these observations can be applied to the clinical problem of evaluating diseased mitral valves in patients. It is recognized that these tracings are dissimilar to the "early plateau type" curves generally described as characteristic of clinically significant mitral insufficiency. The differences may result because these experimental studies are on mitral insufficiency of acute, relatively short duration in otherwise normal hearts.

**Summary**

The possibilities of investigating mitral insufficiency by roentgenologic means have been studied in a series of dogs in which pure mitral insufficiency was created by division of 1 chorda tendinea of the mitral valve. The demonstration of regurgitation of contrast material into the left atrium after injection into the left ventricle seems to represent the most positive roentgenologic sign for establishing the presence of mitral insufficiency.

In this study consistent changes in movement of the posterior border of the left atrium after the creation of mitral insufficiency were recorded by electrokymography. The pattern of these changes is described and a close relation to volume variations is suggested.

**Summario in Interlingua**

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**Fig. 5. Dog 4. Heart showing division of 1 chorda tendinea (arrow)**
Fig. 6. Dog 5. A. Preoperative, left ventricular cardioangiography in ventricular systole. The mitral valve is competent. B. Postoperative film in ventricular diastole, 4 heartbeats after start of injection. No atrial reflux has occurred. The left ventricle is dilated as a result of aortic insufficiency.

Fig. 7. Left ventricular cardioangiography of normal dog with pericardial blood clot (arrow). A. Frontal; no recognizable mitral regurgitation. B. Lateral; mitral regurgitation into normalized left atrium and pulmonary veins.

In le presente studio, alterationes de occurrentia regular in le movimento del margine posterior del atrio sinistre post le creation del insufficientia mitral esseva registrate electromagnosticheramente. Le disposition de iste alterationes es descritbe. Un intime relation inter illos e variationes de volumine es suggerite.
REFERENCES


The thinker makes a great mistake when he asks after cause and effect. They both together make up the indivisible phenomenon.—Goethe, 1749–1832.
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