An Analysis of Normal and Abnormal Left Atrial Pressure Pulse in Man

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Pressure pulses were obtained under direct vision, in the left atrium of subjects without clinical evidence of cardiac disease and in patients with mitral valvular disease, after exposure of their hearts during the course of surgical intervention. It was found that the c wave is produced by valvular closure and the subsequent fall of pressure during early ventricular systole (x wave) is related to atrial relaxation. Evidence is presented that mitral incompetence cannot be determined from direct atrial recordings, unless the degree of insufficiency is of great magnitude.

LEFT ATRIAL PRESSURE PULSES, for many years relegated to the experimental laboratory, have recently assumed an important clinical role in the assessment of the degree and type of mitral disease present preoperatively, and of the effectiveness of surgical intervention postoperatively. This study was undertaken to define more clearly the characteristic cyclic variations recorded from the left atrium in human subjects with normal and diseased mitral valves, and to relate these changes to underlying hemodynamic events.

MATERIAL AND METHODS

Catheters of various sizes and lengths attached to wide bore lead tubing were tested to determine their frequency and sensitivity, and it was found that a no. 7 or 8 cardiac catheter reduced to 35 cm in length gave a response (50 cycles per second) that was adequate to accurately record pressures from either a low or high pressure system. This was also found to be true for a no. 20 needle attached to lead tubing.

Normal left atrial pressures were recorded in 10 patients during the course of limited excisional surgery for pulmonary tuberculosis. In six patients a 35 cm. no. 8 cardiac catheter was introduced into the left atrium via a segmental pulmonary vein. In five of these the catheter was subsequently advanced through the mitral valve into the left ventricle. In four other patients the use of a no. 7 double lumen catheter, 30 cm. long, again introduced via a pulmonary vein, made possible the simultaneous recording of left atrial and ventricular pressure pulses. In three patients with mitral valvular disease, 20 gage needles attached to lead tubing were used to puncture the atrial wall in order to record pressures from this chamber.

Pressures in all instances were recorded by attaching the catheters or needles to Statham strain gauge transducers via lead tubing. The recording apparatus included high sensitivity carrier amplifiers and a photographic device for duplicating the oscillographic image. Zero level for all manometers was adjusted to the estimated height of the left atrium. Thus all recorded pressures are relative to this estimated zero level and are therefore not quantitatively absolute.

RESULTS

The Normal Left Atrial Pressure Pulse: Figure 1A and B show simultaneous recordings of left atrial and ventricular pressure curves in normal hearts obtained through double lumen catheters. The two tracings are virtually superimposed since the manometer sensitivities are similar and the base lines are approximately 2 mm. apart. The atrial tracings are labeled according to the schema used by Little and associates. In figure 1B, the catheter tip is apparently located at the level of the mitral valve, for during late ventricular diastole the tip assumes a ventricular position as evidenced by the deviation from the normal atrial contour and the recording of the onset of ventricular contraction. These records are shown to demonstrate that the c wave is a function of the relative pressures within the atrium and ventricle with the onset of ventricular systole. If at the start of ventricular contraction, the ventricular pressure is less than the atrial pressure, a prominent c-wave appears as in
LEFT ATRIAL PRESSURE IN MAN

Figure 1A. If, however, at this moment, the ventricular pressure is higher than the atrial, no c-wave is seen in the atrial curve (fig. 1B). A similar observation has been made by Little in dogs.

The hemodynamic events that are responsible for the normal left atrial pressure pulse are further elucidated by a study of premature ventricular beats illustrated in figure 2 from a patient with a normal heart. With the onset of premature beat number 1, there is a sharp rise in atrial pressure resembling, in some respects, the c-wave of the normal sinus beat. Since this ventricular beat occurred while the mitral valves were open, i.e., following the v-wave this atrial pressure rise is due to closure of the mitral valves as the ventricular pressure suddenly rises. After a slight fall, the atrial pressure is further elevated by an atrial contraction (a) against a closed mitral valve. Beat number 7 also shows atrial contraction occurring against a closed mitral valve.

Beats 1 and 7 show a considerable fall in atrial pressure following atrial contraction. This decrease corresponds in time to the x-wave of normal beats and occurs even though the venous inflow continues, atrial outflow is arrested by closed mitral valves, and there is no descent of the base during this phase of ventricular systole. It follows, therefore, that this fall in atrial pressure is a function of atrial relaxation following its contraction. Consistent with this explanation is the observation that the elevated atrial pressure occurring just after

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Fig. 1. Left atrial and ventricular pressure pulses recorded through double lumen catheters in normal subjects. Ordinate values in mm. Hg. For discussion see text.

Fig. 2. Left atrial and ventricular pressure tracings and ECG recorded in a normal subject through double lumen catheter. Extra-systoles produced by tip of catheter, stimulating the endocardium of the left ventricle. a = atrial systole, P = electric atrial systole.
FIG. 3. Left atrial pressure pulses: (A) mitral stenosis with normal sinus rhythm and no regurgitation detected by palpation; (B) mitral stenosis with atrial fibrillation and no palpable regurgitation and (C) mitral stenosis with atrial fibrillation and a large regurgitation by palpation.

extrastyle number 6 is sustained in spite of ventricular contraction and supposedly descent of the base. In this instance, the pressure does not fall since its rise was not immediately preceded by atrial contraction as is evident from the electrocardiogram and therefore atrial relaxation did not take place. In figure 3 of Little's paper a similar rise in atrial pressure is sustained throughout ventricular systole after the preceding atrial contraction had been prevented by left vagal stimulation.

**Left Atrial Pressure Tracings Modified by Mitral Valvular Disease:** Figure 3 shows three atrial tracings taken from different patients undergoing mitral valve surgery. Record A is from a patient with mitral stenosis, normal sinus rhythm and no regurgitation palpable by the surgeon. This tracing is similar to a normal atrial pressure pulse except for elevated mean pressure and a prominent c-wave. The prominent c-wave is as expected, since the atrial pressure in mitral stenosis is much higher than the ventricular at the beginning of ventricular systole. Note that the stenosis did not affect the x-wave.

Record B is from a patient with mitral stenosis, atrial fibrillation and no detectable regurgitation. Besides the absence of the a-wave, the striking change from the normal is the prominent c-wave, and the absent x-wave. In the absence of atrial contraction, and therefore, relaxation of this chamber, no x-wave is seen. However, immediately following the c-wave, there is a gradual rise in pressure forming a definitive v-wave. This type of tracing has been published by others, and is apparently characteristic of either left or right atrial curves in the presence of fibrillation.

Record C is from a patient with mitral stenosis, atrial fibrillation, and a fixed mitral valve resulting in a large amount of palpable regurgitation. As was shown at postmortem examination, the valves were fixed so that there was essentially a common atrio-ventricular cavity on the left side of the heart. Thus the atrial curve is similar to a ventricular tracing. Note the early rise in pressure, absence of a spiked c-wave as is usually seen with fibrillation and for stenosis, and no pressure rise that appears to be similar to a v-wave.

**DISCUSSION**

With the absence of atrial contraction and relaxation (atrial fibrillation), the atrial pressure pulse markedly deviates from the normal. Besides the absence of an a-wave, there is a prominent c-wave and absence of an x-wave. Without partial closure of the mitral valve normally afforded by atrial contraction (Henderson, Dean), the leaflets remain widely separated just prior to ventricular contraction. Consistent with this postulation is the visual photo-fluoroscopy demonstration by Ravitch, that with fibrillation a minute wisp of dye regurgitates back into the atrium from the left ventricle with the onset of ventricular contraction. Thus, the prominent c-wave recorded with atrial fibrillation is due to a minute amount of incompetence as well as encroachment upon the atrial volume by the closing valve leaflet from a widely separated position.

With the absence of atrial relaxation there is no fall in atrial pressure during early systole (x-wave). Thus the ascent of the v-wave begins immediately after the elevated c-wave. The
resulting atrial tracing (fig. 3B) could easily be interpreted as showing significant mitral regurgitation unless the underlying hemodynamic events resulting from fibrillation were well understood. This type of tracing also illustrates the necessity for using a system with adequate frequency and sensitivity response so that an impact-like pressure change such as the c-wave can be accurately and clearly transcribed.

The diagnosis of significant mitral valve incompetence can be made with some certainty from the type of atrial tracing which shows a rise in pressure during the early phase of ventricular systole (Fig. 3C). Similar atrial tracings have been recorded in the experimental laboratory. However, many published atrial tracings recorded from cases with proven insufficiency show little change during early ventricular systole, but merely demonstrate an enlarged v-wave. These clinical observations are consistent with the experimental findings of Wiggers and Feil.

The difference between the ventricular type and the enlarged v-wave type of atrial curve must be the degree of incompetence of the mitral valve. It is conceivable that the larger the opening, the more ventricular in character will be the atrial pulse and the less will be the pressure drop across the mitral valve. The smaller the opening in the valve, the less will the atrial cyclic variations deviate from the normal and the greater will be the atrioventricular gradient. Perhaps further study of the pressure drop across the mitral valve will yield information that will help to determine the degree of stenosis and presence or absence of significant insufficiency.

Since it is true that hemodynamically significant insufficiency can be present with only minor alterations of the left atrial pressure pulse, direct left atrial pressure measurements are of limited value in establishing the presence of mitral valve incompetence. It is only in those cases in which changes in the atrial pulse are marked that the diagnosis of incompetence can be made with certainty by this method.

**SUMMARY**

Left atrial pressure pulses were recorded with a system of adequate sensitivity and frequency in 10 subjects without heart disease undergoing limited resectional surgery and in three patients with mitral valvular disease. In 9 of the 10 normal cases, left ventricular pressures were also recorded.

An analysis of left atrial and ventricular pressure tracings showed that the c-wave is produced by valvular closure and the x-wave (fall in atrial pressure during early ventricular systole) is related to atrial relaxation.

By virtue of the above observations, it is possible to explain the prominent c-wave and absence of the x-wave in cases with atrial fibrillation.

From a review of clinical and experimental atrial pressure studies, it is concluded that mitral valvular incompetence cannot be determined from direct left atrial recordings unless the degree of insufficiency is of great magnitude.

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**SUMMARIO IN INTEHLINGUA**

Le pulsos del pression sinistro-atrial esseva registrate per medio de un systema de adequate sensibilitate e frequentia in 10 subjectos sin morbo cardiac qui se subjiceva a un limitate chirurgia resectional e in 3 patientes con morbo de valvula mitral. In 9 del 10 casos normal, etiam pressiones sinistro-ventricular esseva registrate.

Un analyse del registracions de pression sinistro-atrial e -ventricular monstrava que le unda C es producite per clausura valvular e que le unda X (abassamento del pression atrial durante le systole ventricular initial) es connectite con relaxation atrial.

Le supra-reportate observationes permitte le explication del prominentia del unda C e del absentia del unda X in casos con fibrillation atrial.

Un revista de studios clinic e experimental de pressiones atrial supporta le conclusion que incompetentia del valvula mitral non pote esser determinate super le base de directe registracions sinistro-atrial excepte si le grado del insufficicntia es multo alte.
REFERENCES

4 HENDERSON, Y. AND JOHNSON, F. E.: Two modes of closure of heart valves Heart 4: 69, 1912.

Compulsory Research

"Happily there are plenty of men you cannot keep from original research; others only need putting on the scent and they’re off on the track of truth. But must every ambitious graduate be forced by custom or authority to prosecute research in order to obtain a university post or to succeed as a practising cardiologist? Research ability used to be regarded as a rare gift, something of a phenomenon. It was search with some prospect of finding; it connoted both a spirit of curiosity and a capacity for investigation. That is not the attitude today, though one must admit that it is largely the expansion of organized research that has transformed medical practice. My question concerns the universality of the capacity for research; and I almost believe that the true investigator, great or small, is born not made. That famous literary physician, Sir Thomas Brownie, wrote: “Every man is not a proper Champion for Truth, nor fit to take up the Gauntlet in the cause of Verity.”

"Though commoner than aptitude for research, the power to apply new (and old) knowledge judiciously is not given to everyone. It also requires a gift, namely, clinical judgment. This will ripen to clinical wisdom only by constant association with the sick. Young physicians of this type, so badly wanted, need to keep themselves abreast of the world literature in their specialty, and devote time to postgraduate clinical work at other centers and in other lands. Must they invariably be forced to shorten the time required for this large purpose to participate in minor and often ephemeral research before they are considered for any important staff appointment as a teacher or consultant? Not every young physician or cardiologist should be under compulsion to prosecute individual research in order to succeed."

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