Unusual Diastolic Heart Beat in Pericardial Effusion

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SUMMARY

Unusual, hitherto undescribed form of diastolic heart beat was reported in a case of tense pericardial effusion and in another case of seroconstrictive pericarditis. It consists of large and sharp early diastolic thrust, the peak of which coincides in time with the protodiastolic extrasound (pericardial knock). The mechanism and the diagnostic significance were discussed. Failure to recognize this diastolic heart beat in most cases of pericardial effusion or seroconstrictive (effusive-constrictive) pericarditis may be due to the absolute faintness of the precordial movement in these conditions. In addition, inspiratory widening of the split second heart sound due to the shortened electromechanical systole was described in a case of tense pericardial effusion without constriction.

Additional Indexing Words:

Diastolic heart beat  Pericardial knock  Splitting of the second heart sound  Pericardial effusion  Seroconstrictive pericarditis  Effusive-constrictive pericarditis  Phonocardiography  Ultrasound cardiogram  Pulsus paradoxus

The purpose of this report is to describe an unusual diastolic heart beat found in 2 cases who had effusive pericarditis. This rare heart beat is seldom mentioned in the texts on cardiology, so that it would be worthy of reporting.

CASE REPORTS

Case 1. A 30-year-old woman was admitted to the Second Dept. of Internal Medicine, Tokyo University Hospital, with a history of general edema on March 25, 1971. She has been treated at the outpatient clinic for about 10 years as having chronic nephritis which was diagnosed by clinical ground as well as the biopsy finding of the kidney.

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B: X-ray film taken on Dec. 24, 1971. Pericardial effusion increased, whereas pleural effusion disappeared. UCG (see Fig. 3) was taken under this state.

Fig. 2. Electrocardiogram of Case 1.
Fig. 3. Echocardiogram (UCG) of Case 1.
Phonocardiogram and electrocardiogram are simultaneously recorded.
Pericardial effusion is evident by the wide separation of pericardial echo and
posterior wall echo of left ventricle. Time scale: 0.4 sec., vertical scale:
2.0 cm.

Physical and laboratory findings included general edema with ascites and pleural
effusion (Fig. 1A), albuminuria, hepatomegaly and hypertension (180/120 mm.Hg).
Anemia was evident (Hb. 5.8 Gm./100 ml., RBC 1.82 million/cmm.), and high
erthrocyte sedimentation rate (121 mm./hr.), hypoalbuminemia (5.4 Gm./100 ml.)
with lowered A/G ratio (0.4), hypercholesteremia (630 mg./100 ml.), elevated BUN
(70.5 mg./100 ml.) and high creatinine level (11.5 mg./100 ml.) were noted. Other
biochemical and serological studies were within normal range. Renal biopsy and
renal clearance findings further supported the diagnosis of renal nephrosis. Electro-
cardiogram (Fig. 2) showed prolongation of Q-T interval, though the voltage of QRS
complex was at lower limits. Echocardiogram (Fig. 3) disclosed the free space be-
tween the posterior wall and the pericardial echoes, indicating the presence of peri-
cardial effusion. At this time, the cardiac shadow was slightly enlarged as shown in
Fig. 1B. Blood pool scan further confirmed the effusion.

Auscultatory and phonocardiographic findings including pulse studies:
Inspection and palpation of the precordium revealed the well-visible tapping
impulse diffusely located from the anterior axillary line to the lower left sternal border and maximal in the 5th intercostal space in the mid-clavicular line (5LMCL), which was thought the site of the apical impulse. This heart beat was composed of definite systolic retraction followed by sharp and huge outward movement which coincided in time with the protodiastolic extrasound (pericardial knock) (Fig. 4). The graphic recording of the apex cardiogram (ACG) taken in a supine position revealed that the respiratory variation of the amplitude and the timing of the sharp outward movement during early diastole. The maximal amplitude of this diastolic heart beat was observed in late inspiration or early expiration (Fig. 5) and minimal in the expiratory apnea. The interval between the peak of the impulse and the second heart sound (IIS) also varied during respiration (0.05–0.09 sec.), shorter in early expiration. Generally speaking, the larger the amplitude, the shorter the interval. On the other hand, the systolic wave showed no appreciable change by respiration. Characteristically, the diastolic heart beat observed in this patient was not sustained as in usual case of constrictive pericarditis, but it was rather an impulse which is similar to the water hummer pulse. The pulse tracings recorded anywhere between the anterior axillary line to the lower left sternal border showed essentially the same pattern.
Moreover, the use of the left lateral decubitus position for recording the ACG gave the same result.

The jugular phlebogram showed the systolic collapse (x descent), and y descent was not sharp all the time (Fig. 6). Venous pressure was not elevated at the time of this recording (105 mmH₂O). Thus, there was no Friedreich's sign. Kussmaul's venous sign was also not noted.

The carotid pulse tracing showed normal pattern, but slight pulsus paradoxus was observed regularly, giving the small pulse during inspiration (Fig. 7).

Auscultation and phonocardiogram disclosed a typical protodiastolic extra-sound (pericardial knock), which was maximal at the site of maximal impulse (Figs. 6 and 8). This extrasonde contained both low-pitched and high-pitched components, but had predominantly lower frequency component as in the case of usual third heart
Neither positive systolic wave nor sharp y descent was observed. Phonocardiograms are taken from apex (upper 3 tracings) and 3L. Now, the pericardial knock (protodiastolic extrasound) is clearly inscribed.

sound. However, the acoustical feature was medium-pitched, probably because of the nature of the mixed frequency span. It was nearly merged with the second heart sound at the beginning of the expiration (Fig. 8), but separated during expiratory apnea (0.09 sec.). This was the same behavior as the peak of the diastolic heart beat (cf. Fig. 5). No friction rub was observed by daily monitoring during past 6 months.

Another important phonocardiographic finding was the splitting of the second heart sound best observed in the third intercostal space along the left sternal border (3L). The split interval varied from 0 to 0.065 sec. during respiration (Fig. 7). The inspiratory splitting occurred rather abruptly by inspiration, and this was due to the sudden movement of IIA (aortic component) towards the first heart sound (shortened electromechanical interval) as well as less marked delay of IIP (pulmonic component). The shortened ejection time was also observed in the carotid pulse tracing. In general, both IIP and K (knock sound) moved simultaneously during respiration, but the movement of the former was much more abrupt.

This patient was treated by peritoneal dialysis twice a week and the clinical course remained almost constant. Then, she was transferred on Feb. 29, 1972, to Mitsui Memorial Hospital, Tokyo, for further treatment of her chronic state. Peritoneal dialysis and artificial kidney were performed regularly. However, cardiomegaly was insidiously progressive and isotope angiography and echocardiography proved the increased fluid in the pericardium. Pericardial paracentesis was attempted, however, it failed to obtain the fluid. She died of shock following the
puncture on April 18, 1972.

Autopsy disclosed 1,500 ml. of serous pericardial fluid which contained bloody coagula due to the puncture of the left ventricular wall. No pericardial adhesion and also visceral pericardial restriction were observed. Pericardium was smooth except for the bleeding site. Heart weight was 370 Gm. and the thickness of the left ventricular wall was 13 mm. There was left ventricular dilatation. Cardiac valves were not pathologic except slight atheroma of aortic and mitral valves. Coronary arteries were patent, but slight atheroma was present. Both kidneys weighed 70 Gm. for each. Lung congestion was noted. Generalized arteriosclerosis was present.

Case 2. A 28-year-old man was admitted to the Third Dept. of Internal Medicine, Tokyo University Hospital, in May, 1966, because of dyspnea, general edema
and hepatomegaly. Since Nov., 1964, he has been treated as having lymphadenitis tuberculosis of the left lung hilus, which subsided by the administration of streptomycin. However, in July, 1965, lymphnode of the neck swelled and the marked pericardial effusion was noted on the chest roentgenogram. Positive C-reactive protein (2 plus) and elevated erythrocyte sedimentation rate (112 mm./hr.) were noted. Pericardial effusion was once disappeared by the appropriate therapy, but it relapsed into polyserositis including pleural effusion and ascites with hepatomegaly. At the time of admission, he was dyspneic and ascites was apparent, but the peripheral edema was lacking. Jugular vein was distended and the venous pressure was 300 mm.Hg.

He was referred to the Second Dept. of Internal Medicine on July 4, 1966, for further graphic study including phonocardiography and other pulse tracings (mech-
On the phonocardiogram, pericardial knock (K) is clearly inscribed at the apex (upper 2 tracings). The tracings taken from 3L show splitting of the second heart sound with accentuated IIP (pulmonic component). ACG shows early systolic retraction followed by mid-systolic outward movement. Conspicuous early diastolic heart beat is observed, the peak of which coincided in time with the knock sound. Though the amplitude of ACG seems large, however, the precordial pulse by palpation was not prominent.

anocardiography). At this time, right-sided pleural effusion was present, but no cardiomegaly was noted, so that the pericardial effusion was questionable or of slight degree. On physical examination, the carotid pulse was feeble, while the jugular vein was strongly distended. However, Kussmaul’s sign was not observed. Jugular phlebogram revealed systolic wave (x descent), high v wave, and sharp y descent which was slightly deeper than the x wave. At any rate, the positive systolic wave was not observed. Apex beat was weak. However, the apex cardiogram (ACG) disclosed the dominant deflection was early diastolic outward movement as seen in Case 1 (Fig. 9). Systolic wave was composed of sharp inward deflection followed by mid-systolic outward motion. Sharp diastolic movement was, as in Case 1, coincided with the protodiastolic extrasound (pericardial knock: K), and it was not sustained, but it was rather water hummer pulse. The interval between the second heart sound (IIS) and the peak of the diastolic beat was 0.08–0.09 sec. during the held expiration. Auscultation and phonocardiography revealed the typical protodiastolic extrasound, which was widely audible over the precordium and was maximal in the site of apex beat. The second heart sound was split at the base, and the pulmonic component was accentuated. The split interval was 0.04 sec. in expiratory apnea. Ausculta-
tion over the epigastrium revealed a series of 3 sounds, IIA, IIP, and K, which were almost fixed in timing irrespective of respiratory phase.

Other laboratory data were prolonged circulation time, dip and plateau of the right ventricular pressure pulse (40/12–22 mm.Hg), normal cardiac output, no pulsus paradoxus, no Kussmaul's sign in the right atrial pressure pulse, not so restricted cardiac movement on roenigen-kymography, cirrhotic liver on peritoneoscopy, and inverted T waves with lowered ST segment in limb and precordial leads on the electrocardiogram. Pleural effusion and ascites were exudate. Liver function tests were within normal range except BSP test (20%).

DISCUSSION

For many years, well marked physical findings have been described in pericardial effusion, tamponade and constrictive pericarditis. Of these, the signs indicating constrictive pericarditis are usually against those indicating pericardial effusion. For example, Lange et al. studied systemically the physical findings in 37 patients of pericardial diseases, in which the Friedreich's sign (early diastolic drop in venous pressure) was mainly observed in constrictive pericarditis, whereas pericardial effusion gave no such a sign. Kussmaul's sign (inspiratory rise in venous pressure) was not observed in pericardial effusion, whereas it appeared in one-third of constrictive pericarditis.

Although the characteristic feature of the apex beat in constrictive pericarditis was often mentioned, the precordial pulsation in pericardial effusion has not been described in detail. Usually, the precordium is silent or shows no apparent pulsation in pericardial effusion. Only Friedberg mentioned that the strong impulse may be present in such cases, though it is often weak and wavy or it may be absent. Wood did not comment on it. Moreover, the prominent third heart sound (protodiastolic extrasound or pericardial knock) would exclude the presence of lax cardiac tamponade or pericardial effusion, and an associated diastolic heart beat also indicates the pericardial constriction.

So far as the authors' knowledge concerns, the presence of protodiastolic extrasound in pericardial effusion was firstly documented by McKusick and Harvey. Our previous experience in a case of prominent pericardial effusion (34-year-old male) showed relatively low-pitched pericardial knock which was 0.08 sec. behind the second heart sound. Thus, it seems likely that pericardial effusion itself may produce the extrasound similar to that of constrictive pericarditis.

On the other hand, the diastolic heart beat, a counterpart of the pericardial knock, has not been described in pericardial effusion. As previously described, it is extremely rare to find the exact feature of the precordial pulse including apex cardiogram in cases of pericardial effusion. Kay et al.
Fig. 10. Example of the phonocardiogram, apex cardiogram and jugular phlebogram in a case with classical signs of constrictive pericarditis. Atrial fibrillation, 51 year-old female.

Loud protodiastolic extrasound (pericardial knock) is recorded widely from apex to base. The second heart sound is split with accentuated pulmonic component (IIIP). Apex cardiogram (ACG) shows typical systolic retraction followed by the diastolic heart beat, which sustains throughout diastole. Jugular phlebogram (Jug.) shows systolic positive wave followed by sharp y descent. A series of diastolic waves including sharp h wave are also recorded. On chest roentgenogram, calcification of pericardium is widely observed. Dip and plateau pattern in left and right ventricles was typical.
reported a case with "late systolic" apex beat with systolic sound, but it seems to be an exceptional occurrence.

Recently, Radel et al.19 and Hancock20 reported the intermediate clinical state, which is a midpoint between pericardial effusion and constrictive pericarditis, and gave it a name of relapsing seroconstrictive pericarditis19 or subacute effusive-constrictive pericarditis.20 In this situation, the visceral pericardium is constricted but tense effusion is present in a free pericardial sac. The Case 2 in this report is quite similar to this state, because of no apparent cardiomegaly, elevated venous pressure, ascites and hepatomegaly, and other signs related to chronic constrictive pericarditis. However, it is quite difficult to determine whether the peculiar apex beat is consistent with seroconstrictive pericarditis.

Classically, the precordial movements including apex beat in constrictive pericarditis are characterized by systolic retraction followed by "sustained" diastolic heart beat (cf. Fig. 10). It is of note, however, the diastolic heart beat in this report is "sharp" diastolic thrust which coincided in time with distinct pericardial knock. Moreover, the Case 1 had no visceral or parietal constriction during her course or at autopsy. On the other hand, the Case 2 had probable pericardial constriction in the presence of effusion. Therefore, the similar phonocardiographic and mechanocardiographic findings of these 2 cases might be discussed on the same ground.

Fig. 11 illustrates the difference between the classical heart beat and the "non-sustained" one described in this report. In constrictive pericarditis, the systolic retraction takes place as the ventricular volume decreases during systole by increased centripetal movement of the free wall of the ventricles, which pulls the chest wall through the window where the scar tissue and calcification are sparse or absent.14 The presence of annular constriction takes part in the occurrence by the interference of free movement of the atrioventricular ring.15 In diastole, venous blood entering under the high atrial pressure causes abrupt distension of the ventricle followed by the sudden halt because of the diminished capacity of the constricted ventricles. This is reflected in the diastolic heart beat and also in the pericardial knock.6,14 On the other hand, in tense pericardial effusion and in seroconstrictive pericarditis, ventricular ejection may produce the negative pressure in the pericardial cavity which contains the fluid. This, in turn, may cause the traction of the chest wall during systole. This may not be uniform throughout systole in seroconstrictive pericarditis, because of the possible irregular pooling of the fluid in the pericardial sac. Thus, the systolic retraction may be partly replaced by outward movement as seen in the Case 2. With completely surrounding pericardial fluid, the retraction may be uniform as in the Case 1. In diastole, ventricular relaxation
Fig. 11. Schematic representation of constrictive pericarditis (upper figure) and of pericardial effusion or seroconstrictive pericarditis (lower figure). Details: see text.

reduces the negativity in the intrapericardial pressure, and then, rushing venous blood expands the ventricles, but the expansion may be promptly impeded by the positive pressure developing in the pericardial sac in early diastole. This is similar to the case of constrictive pericarditis, in which the impedement of the venous blood inflow is in the pericardial constriction. Tense pericardial effusion per se stands to prevent further increment of inflow as a thick fibrous pericardium. However, once the ventricular expansion comes suddenly to a halt, the accumulated pericardial fluid might be expelled to the other place, and localized development of high intrapericardial pressure will be cancelled promptly, giving a water hummer movement of the pericardial surface. With
a gross accumulation of the fluid (Case 1) and/or partial adhesion of the pericardium and the chest wall (Case 2), the chest wall will be struck by this series of pericardial movement. If the effusive-constrictive condition is present as in Case 2, the diastolic thrust with sharp peak in early diastole may be more exaggerated by the constrictive action of the visceral restriction of the ventricles, whenever the adequate effusion is present.20)

At present, the diagnostic significance of the diastolic heart beat with sudden thrust in early diastole and pericardial knock remains obscure. Radel et al.19) mentioned about the gallop, but not about the precordial movement in their case report. Hancock20) also noted pericardial knock in 3 of 13 cases of effusive-constrictive pericarditis and in 2 of 7 of pericardial effusion, but no comment on the precordial movement. This might be due to the absolute faintness of the precordial pulse in most cases of pericardial effusion. However, the result of the present report indicates the importance of the graphic recording of the precordial movement in every case of pericardial effusion.

Finally, it should be emphasized that the Case 1 with massive pericardial effusion without pericardial constriction showed peculiar type of splitting of the second heart sound. Beck et al.21) described the same observation in 4 cases of constrictive pericarditis, in which inspiratory widening (IIA-IIP interval) was proved to be due to the shortened R-IIA interval (electromechanical interval) during inspiration. This coincidence in either pericardial effusion or constrictive pericarditis may be explained by the presence of a common denominator in each condition, i.e. pulsus paradoxus. Therefore, this type of splitting should be investigated carefully in every case of pericardial disease, because of frequent association of pulsus paradoxus with tense pericardial effusion.51

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References