Indirect Pulmonary Artery Pulse

Diagnostic Significance in Transposition of Great Vessels

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SUMMARY

The indirect pulmonary artery pulse in the second right intercostal space, hitherto undescribed, was reported in a case of complex congenital heart disease with transposition of great vessels, atrial septal defect, tricuspid atresia, ventricular septal defect and pulmonary hypertension. It was illustrated that the indirect pulmonary artery pulse in the second right intercostal space indicates the existence of the transposed great vessels. In addition, the significance of the precordial impulse in the third left sternal border was discussed.

Additional Indexing Words:

Indirect pulmonary artery pulse Transposition of great vessels
Precordial impulse Carotid artery pulse

TO diagnose complex congenital heart disease, non-invasive methods such as phonocardiogram, electrocardiogram and chest X-ray play an important role, while heart catheterization and angiocardiogram are often the method of physician's choice.

From our daily clinical experience, we have recognized that the precordial pulse tracing plays a very important role in the diagnosis of heart diseases. In this paper, we report our recent experiences of complex anomaly with transposition of great vessels in which the diagnosis was estimated by means of the recording of the indirect pulmonary artery pulse. Furthermore, it is attempted to mention the interesting precordial pulse similar to the jugular venous pulse.

CASE REPORT

A 15-year-old junior high school student was admitted for precise examination. He was noticed cardiac murmur soon after the birth, but no care was taken. Since

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the 8th month after birth, cyanosis has been noticed and the increase in weight has not been good. At the age of 9 he underwent an operation of cerebral abscess and often suffered from common cold. During his primary school days, exertional dyspnea was observed and has increased recently.

On examination he was 143 cm in height, weight 24.7 Kg and minimally cyanotic. No clubbed fingers were observed. Blood pressure levels on the right and left were balanced indicating 90/40 mmHg. The jugular veins were distended bilaterally, but hepato-jugular reflux was negative. The liver was palpable 2 finger breadths below the right costal margin. No edema was noticed in the lower limbs.

There were precordial pulsations in the second right intercostal space (2R), the third left intercostal space (3L) and apex. Particularly, the pulse in 2R appeared suddenly during the systolic period. At the end of systole, the second sound was detected by palpation. A grade 4 ejection systolic murmur was heard along the left sternal border and was loudest at the second or third left intercostal space. In addition, there was a grade 2 early diastolic murmur at the second and third

Fig. 1. Chest X-ray. Marked cardiac enlargement and increased pulmonary vascular markings are evident.

left intercostal space. A pulmonic ejection sound was heard. The second sound was nearly single and accentuated. No third or fourth sound was heard.

The chest X-ray (Fig. 1) showed a high degree of cardiac enlargement (cardiothoracic ratio=73%) and increase in the pulmonary vascular markings in right and left lungs.

Electrocardiogram (Fig. 2) showed first degree atrioventricular block (PR interval=0.24 sec), broad and notched P waves in leads II, III, and aVF. In lead V₁, P wave was diphasic and the negativity was broad and prominent.

Fig. 2. Electrocardiogram. Left atrial overloading and biventricular hypertrophy are suspected. A PQ interval is prolonged.

There was a deep S wave in leads V_1 through V_5 and a tall R wave ($=5.0$ mV) in lead V_6 . From this electrocardiogram, left atrial overloading and biventricular hypertrophy were estimated.

Phonocardiogram and precordial pulse tracing: Fig. 3 is the phonocardiogram recorded at the 2L and apex. At the 2L, ejection systolic murmur, early diastolic murmur and ejection sound were inscribed. The splitting of the second sound was narrow while the accentuation of the pulmonic component of the second sound was noticed. These findings were consistent with pulmonary hypertension.

Fig. 3. Phonocardiogram (PCG). Upper tracing is taken from the second left intercostal space (2L) and lower tracing from the apex. There are ejection systolic murmur, early diastolic murmur and ejection sound at the 2L. The second sound is almost single and the pulmonic component is accentuated. L=low-frequency PCG, M=medium-frequency PCG, H=high-frequency PCG. Time lines: 0.01 and 0.1 sec.

As is illustrated in Fig. 4, the precordial pulse in 2R showed the pattern of arterial pressure curve. Its dicrotic notch showed very coincident in time with the pulmonic component of the second heart sound. The simultaneously recorded carotid artery pulse and the precordial pulse in 2R (Fig. 5) showed quite different patterns each other. The dicrotic notch of carotid artery pulse tracing showed advancement of 0.01 sec than that of the precordial pulse tracing in 2R. Taking the pulse transmission time into consideration, it became clear that these 2 tracings came from com-

Fig. 4. The precordial pulse tracing in the second right intercostal space (2R 1.f.). The configuration looks like arterial pressure curve and its dicrotic notch coincided with the pulmonic component of the second heart sound. 5L=the fifth left intercostal space.

Fig. 5. Simultaneous recordings of the precordial pulse in 2R (2R 1.f.; lower) and carotid artery pulse (CAR; upper). The 2 show different patterns. The dicrotic notch of carotid artery pulse tracing shows slight advancement than that of the precordial pulse tracing in 2R.

pletely different artery. From these observations, it was estimated that the precordial pulse tracing in 2R was not of aorta but of pulmonary artery (indirect pulmonary artery pulse tracing). This indicates the anatomical existence of pulmonary artery to the right side of the sternum, namely, the existence of transposition of the great vessels (TGV).

The upper column of Fig. 6 shows jugular venous pulse while the lower column shows the precordial pulse tracing in the third left intercostal space (3L). The precordial pulse in 3L showed, at a glance, similar pattern to jugular venous pulse. The deflection estimated as "a" and "v" waves showed near time correspondence to "a" and "v" waves of the original jugular venous pulse, respectively. Interestingly enough, the compression of the jugular vein to record the phlebogram gave the distortion of the pulsation in 3L, and *vice versa*. This indicates the common

Fig. 6. Jugular venous pulse (Jug; upper) and the precordial pulse in the third left intercostal space (3L; lower). The precordial pulse in 3L shows similar pattern to jugular venous pulse.

Fig. 7. Angiocardiogram. Transposition of great vessels is evident. The main trunk of pulmonary artery extrudes to the right and looks like aneurysm. AO=aorta, PA=pulmonary artery, RPA=right branch of pulmonary artery, LV=left ventricle, RV=right ventricle.

source of the pulsation does exist between jugular phlebogram and the pulse in 3L, indicating the large right atrium is situated beneath the 3L.

From the above-mentioned examinations, the existence of TGV, large atrium and pulmonary hypertension were thought to be highly suggestive prior to angiocardiogram and heart catheterization. Actually angiocardiogram and heart catheterization disclosed the presence of TGV, atrial septal defect, ventricular septal defect, tricuspid atresia and pulmonary hypertension. The size of atrial septal defect was large, giving an impression of common atrium. The main trunk of pulmonary artery extruded to the right side as an aneurysm (Fig. 7). And it was thought that the pulse in 2R was caused by this aneurysmal pulmonary artery.

DISCUSSION

The detection of the pulse by palpation in 2L is a well-known fact in

atrial septal defect, anomalous pulmonary venous drainage, etc, in which the increase in the pulmonary blood flow and/or pulmonary hypertension is observed. This objective description is known as the indirect pulmonary artery pulse tracing. Sakamoto et al¹⁾ have made detailed report on this subject from the view point of hemodynamics. However, the indirect pulmonary artery pulse tracing was so far recorded only from the left side of precordium (mainly in 2L). As far as we know, we were the first in making a record of the pulmonary artery pulse in 2R. Whenever, the pulmonary artery pulse tracing is obtained in 2R or in the right side of the precordium, the existence of pulmonary artery anatomically to the right side should be indicated. This, in turn, indicates the possibility of the existence of TGV. However, the indirect pulmonary artery pulse tracing will not always obtainable in all cases of TGV. This will be limited only to those cases which has the increase in blood flow in the pulmonary artery and/or increase in intravascular pressure, and thin chest wall, etc. In spite of these restrictions, however, it can be said that the record of the indirect pulmonary artery pulse tracing in 2R is a very attractive and powerful mean of diagnosis for TGV.

The recognition of the pulmonary artery pulse tracing obtained in second intercostal space can be made by the fact that the dicrotic notch of that pulse tracing and the pulmonic component of the second sound have close time coincidence. This can be recognized more clearly if simultaneously recorded carotid artery pulse is analyzed.

It is also a well-known fact that the pulse in the left sternal border is detected in the cases of right ventricular overloading^{2),3)} or mitral insufficiency.⁴⁾ Analysis based on hemodynamics has been conducted. In such cases of right ventricular overloading, so-called "parasternal lift" in systolic period is observed. Also in the cases of mitral insufficiency, the pulse through the end of systolic period to the early diastolic period due to rapid dilatation of atrium are observed. They are both recognized as the important precordial pulse for clinical diagnosis.

In the present case, the pulse tracing in 3L is similar to that of the pattern of the jugular venous pulse, and different from the above-mentioned parasternal lift. In this case, high possibility exists that the atrial movement was transmitted to the chest wall (particularly in diastolic period) due to the increase of size and activity of the atrium, which was caused by the common atrium and the tricuspid atresia. The same function as that of the transmission of atrial movement to chest wall in the case of mitral insufficiency is considered. This, however, would ask for further investigation.

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