Pulsations of the Arm Veins in the Absence of Tricuspid Insufficiency*

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Pulsations of the peripheral veins have so far been known to be synonymous with tricuspid insufficiency (TI). Three patients who had been using heroin intravenously for 3 to 25 years and had no evidence of TI were found to have pulsations of the veins of the upper extremities. Recording of intravenous pressure of the peripheral vein and jugular venous pulse (JVP) showed that both were identical in their wave contour and timing and showed indirectly that right atrial pressure waves were being transmitted to the veins of the arm. It is proposed that the venous valvular insufficiency produced by thrombophlebitis of the arm veins in heroin addicts and chronically elevated right atrial pressure in the patient with primary myocardial disease, was the primary cause of retrograde transmission of the right atrial pressure. The presence of normal “a” and “c” wave with good “x” descent and “v” with a normal “Y” descent rules out significant TI while a prominent “v” wave preceded by a truncated “x” descent or regurgitant wave and followed by a sharp “Y” descent are characteristic of TI.

PATIENT MATERIAL

Four patients with pulsations of the superficial veins of the arms were studied. Three patients had been chronic users of intravenous heroin for periods of 3 to 25 years. The pulsations were best seen in right cephalic vein. (Practically all the veins of the upper extremity have been used for intravenous injections in heroin addicts.) The fourth patient was a known case of alcoholic cardiomyopathy and had been in biventricular failure for five years with evidence of mitral and tricuspid insufficiency.

METHOD

Right cephalic vein in three patients and basilic vein in one patient was cannulated with a No. 1817 Bardic inside needle catheter* and pressures were recorded using Statham strain-gauge transducers with patients in supine position. Where possible external jugular vein and carotid pulse tracings with the use of suction cup and pressure cup assembly,** surface phonocardiogram from fourth left parasternal area using Sunborn phono-pickup*** along with an electrocardiogram were recorded simultaneously with the cephalic or basilic vein pressure. The position of the intracath was confirmed fluoroscopically to be in cephalic vein and it was further confirmed angiographically that the cephalic vein drained into the subclavian vein and not into the jugular vein. In two patients undergoing diagnostic cardiac catheterization, indicator dilution curves were obtained by injecting indocyanine green into the right ventricular inflow tract and sampling was performed from the right atrial outflow area.10-11 In both of these patients appearance time of the dye in the right atrium was 10 to 12 seconds, thus ruling out tricuspid regurgitation.

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**Case Reports**

**Case 1**

A 28-year-old man who had been a heroin addict for five years was admitted on March 25, 1971, for uncontrolled diabetes. Physical examination revealed a blood pressure of 120/80 mm Hg, pulse rate 90/min, and temperature 98.4°F. Positive findings were limited to the cardiovascular system which revealed an atrial diastolic gallop along with a grade 1-2/6 early systolic murmur best heard at the fourth left intercostal space parasternally. S1 and S2 were within normal limits. His cephalic and antecubital veins were found to be pulsating almost synchronously with jugular venous pulse (JVP). Excepting the scar of thyroidectomy and sclerosed veins over both arms, the rest of the physical examination was unremarkable. On the basis of venous pulsations a clinical diagnosis of TI was made. Results of blood cultures were reported to be negative. Cardiac catheterization performed later showed that right atrial (RA) mean pressure was 3 mm Hg, right ventricular (RV) 22/8 mm Hg, and pulmonary artery (PA) pressure 22/8 mm Hg. Indicator dilution curves did not substantiate the diagnosis of TI.

Figure 1, panel A, shows that cephalic vein and jugular venous tracings are synchronous and identical in their wave form. All the “a,” “c,” and “v” waves can be recognized. There is a good “x” descent which rules out significant TI. Panel B shows the relationship of the peripheral venous pressure with carotid artery pulse. Panel C shows relationship between JVP and right atrial (RA) pressure waves. JVP is identical with right atrial pressure curve in its timing and contour, and thus indirectly proves that the peripheral venous (PV) pressure waves (panel A) are a reflection of right atrial pressure changes and further that the right atrium freely communicates with the peripheral veins.

**Case 2**

A 21-year-old man, a heroin addict for three years, was admitted on January 15, 1971, for treatment of bacterial endocarditis.

On admission, his blood pressure was 120/50 mm Hg, pulse 82/min, and temperature 100°F. Positive physical findings were limited to the cardiovascular system. Peripheral arterial pulses were of normal character. His JVP was interpreted to be of normal character. Most of the superficial veins of both arms were riddled with needle marks and were sclerosed but both the cephalic veins and a few of its tributaries showed pulsations almost synchronous with the JVP. His peripheral venous pulsation was better seen on the right side and in supine position. Cardiac size was grossly within normal limits. S1 and S2 were within normal limits. A grade 1/6 early systolic murmur, a grade 2/6 early diastolic murmur and S4 were heard at fourth left intercostal space parasternally. Lungs were clear to auscultation and percussion. Spleen was not palpable. A clinical diagnosis of aortic and tricuspid insufficiency was made.

His admission hematocrit was 44 percent, white blood cell count 9,300 per mm³ with normal differential, blood urea nitrogen 15 mg percent and electrolytes were within normal limits. Blood cultures revealed Strep fecalis. His chest x-ray film and electrocardiogram were within normal limits.

He was treated with ampicillin and streptomycin and left the hospital against medical advice after three weeks but continued taking oral ampicillin for three more weeks.

He was readmitted on March 16, 1971, for diagnostic catheterization. He had remained afebrile. Physical examination and laboratory findings were essentially unchanged from the last admission. After results of blood cultures were reported negative, he underwent cardiac catheterization on March 19, 1971. Simultaneous RA and RV pressures showed “a,” “v,” and RV end diastolic pressure of 5 mm Hg. PA pressure measured 22/8 mm Hg. Left ventricular pressure was 105/10 mm Hg. Indicator dilution curves did not substantiate TI. Aortic root angiograms confirmed the diagnosis of mild aortic regurgitation. Pressures were also recorded from the right basilic vein and compared with right

**Figure 1.** Panel A shows lead I, surface phonocardiogram (phono) from lower left sternal border (LSB), JVP and peripheral venous (PV) pulse. In panel B, JVP is replaced by carotid artery pulse tracing and in panel C, relationship between JVP and RA pressure recording is seen and lead II replaces lead I. JVP and PV in panel A are similar in their contour and in panel C, JVP is identical with RA pressure waves; therefore, PV reflects directly the pressure changes in RA. Absence of discernible systolic murmur and presence of a good “x” wave rule out TI.
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Figure 2. Panel A shows lead II and right basilic venous pressure. Panel B shows RA pressure. Both the pressures were recorded one after the other. In basilic vein "a" and "c" waves seem to be squeezed together while a distinct "v" remains separate. Presence of distinct "ac" and "v" waves in a peripheral vein must indicate their transmissance from right atrium. Normal "x" and "Y" waves point against TI.

atrial pressures (Fig 2). As may be noted, two major waves can be seen in the basilic vein pressure recording. Both "a" and "c" seem to be together with a good "x" wave which is against significant TI. In the basilic vein "ac" and "v" waves measured 10 mm Hg. The mere fact that "ac" and "v" waves can be easily recorded from basilic vein and that they appear to be synchronous with right atrial "a" and "v" waves (when the two tracings are measured against ECG) is sufficient proof that the right atrium was in direct communication with the vein.

Case 3

A 38-year-old woman who had been using heroin for 25 years was admitted for diagnostic work-up of anemia. Her blood pressure was 100/70 mm Hg, pulse 88/min, and temperature 98.4°F. Practically all the veins of the arms were found to be sclerosed but in spite of this, blood could be withdrawn from cephalic and antecubital veins. With the patient in the supine position, pulsations of cephalic and antecubital veins could be seen. Pulsations were better appreciated on the right side than on the left. Heart size was grossly within normal limits. S1 and S2 were within normal limits. No gallop or murmur was heard. Other physical examination was within normal limits.

On admission her Hct was 23 percent, HB 7 gm percent, reticulocyte count 0.4 percent, and WBC 7,000 per mm3, with a normal differential count. Rest of the laboratory findings were within normal limits.

Figure 3 shows that jugular and cephalic venous pulsations are identical in contour and timing. Both "a," "c," and "v" waves are seen. There is a prominent "x" wave and "Y" descent is not sharp; these rule out significant TI. This tracing shows clearly that the right atrial pressure changes are being transmitted to the peripheral veins without any resistance and delay.

Case 4

A 41-year-old man was being observed in the cardiac clinic for biventricular failure secondary to alcoholic cardiomyopathy since 1966. There was no history of any drug addiction. His blood pressure was 100/80 mm Hg, pulse 110/min, respiration 22/min, and temperature 98.4°F. His head showed side to side bobbing synchronous with jugular venous pulsations. With the patient lying at an angle of 45° the jugular veins were distended along their whole length. The veins over the head, face, and arms were found distended and pulsating. Both "a" and "v" waves were recognized with sharp "Y" descent. Examination of the chest revealed crepitations at both lung bases. The precordium showed point of maximum impulse at left anterior axillary line in sixth intercostal space. A parasternal lift was both visible and palpable. S1 and S2 were within normal limits. A grade 3/6 pansystolic murmur was heard at the apex. This murmur radiated posterolaterally. Another grade 3/6 pansystolic murmur which increased during inspiration was audible at the fourth left intercostal space parasternally. Prominent atrial and ventricular diastolic gallop sounds were heard both at apex and left lower border of sternum. Peripheral arterial pulses were weak in force and low in volume. Examination of abdomen revealed a pulsating liver about three fingerbreadths below the right costal margin. Both the lower extremities showed +2 pitting edema.

Figure 4, panels A and B show a pansystolic murmur at the lower sternal border. JVP shows an "a," "c" wave with a foreshortened "x" wave which is followed by a regurgitant wave (s). Intravenous pressure recording shows "v" wave measuring 32 mm Hg which is followed by a rather sharp "Y" wave. All of the features are characteristic of moderate to severe TI and are reproduced in the cephalic vein proving indirectly that right atrial pressure waves are being freely transmitted to the cephalic vein.

DISCUSSION

Normally, superior and inferior venae cavae have no valves and, therefore, a raised right atrial pressure is transmitted without any impediment to jugular vein and hepatic veins (having no venous
FIGURE 3. This figure shows lead II surface phonocardiogram from left lower sternal border (LSB), peripheral venous (PV) pressure, JVP and carotid pulse tracings. Phonocardiogram fails to show any systolic murmur. S1 is preceded by an atrial diastolic gallop. PV and JVP show similar waveform and reveal "a", "c" and "v" waves. "X" wave is prominent and "Y" wave has a normal descent thus ruling out TI. Since JVP reflects volume changes in right atrium and here PV is identical with JVP, therefore, peripheral venous pressure can be taken to depict right atrial pressure changes. This must mean direct communication between PV and RA without any barrier.

FIGURE 4. Panel A shows lead II surface phonocardiogram from left lower sternal border (LSB), JVP and PV pressure. In panel B, carotid artery pulse has been added. Phonoc shows a pansystolic murmur. JVP is similar to PV and shows "a", "c", "s", and "v" waves. "X" wave has been truncated and "c" is followed by a positive wave, the regurgitant "s" wave. "Y" wave has a sharp descent. All these facts point towards moderate TI. Since PV is similar to JVP, the former may be said to be representing RA pressure changes and be in direct communication with right atrium.
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...valves) causing pulsation of jugular veins and its tributaries and the liver respectively. Functional insufficiency of venous valves in the lower extremities whether of congenital origin or acquired after thrombophlebitis is supposed to cause varicosity of the veins. It is, therefore, understandable that in patients with TI pulsations of the veins of the legs have been reported only in patients with varicose veins. As long as the balance between the venous pressure and the resistance of the venous walls and the valves is maintained, the volume and pressure changes in the right atrium cannot be transmitted to the cephalic, axillary, femoral, and saphenous venous system because of the presence of functionally competent venous valves. In addition to conditions which could upset the balance between venous pressure and resistance of venous walls and the valves and cause the peripheral veins to pulsate, the veins can also show pulsations transmitted from the terminal arterial system under conditions where peripheral vascular resistance is decreased, and terminal arterioles open up, eg severe aortic regurgitation, anemia, application of heat to the limb and ingestion of alcohol. But in these instances it can be surmised that venous pulsation would look more like arterial than venous.

Increased incidence of intravenous use of heroin has provided most favorable circumstances for thrombophlebitic process of the arm veins. It is postulated that (1) repeated heroin injections into the arm veins cause thrombophlebitis of both superficial and the deep veins, (2) the venous thrombi over a period of years are resolved and the venous channels recanalized, (3) and during this process valves are destroyed thus putting the right atrium in direct extension with the veins of the arms which would pulsate like right atrium and jugular veins. There have been no postmortem studies of the veins of heroin addicts to verify this thesis.

Patient 3 illustrates point 2 well in that we were able to aspirate blood from previously thrombosed and noninjectable veins. The first three patients are characterized by a long history of intravenous use of heroin providing enough time for recanalization and destruction of venous valves after phlebothrombosis thus transmitting right atrial pressure waves to the peripheral veins under conditions where peripheral venous pressure approached the right atrial pressure. As can be seen from the illustrations, peripheral venous pressure tracings are identical with the tracings of jugular venous pulse and thus indirectly with right atrial pressure changes. In cases 1 and 2 peripheral venous pressure changes are identical with those of the right atrium.

That chronically increased right atrial pressure, especially in the presence of TI, can upset the balance between the resistance of the venous walls, valves, and the intravenous pressure and thus make the venous valves functionally incompetent (although anatomically intact), can be appreciated in case 4 and is further corroborated by the necropsy findings in Friedriech's case, mentioned by Kerr and Warren. In our case there was no historic or objective evidence of intravenous drug use but the patient had chronic biventricular failure with functional tricuspid and mitral insufficiency and showed the cephalic veins which pulsed synchronously with JVP. Patients without TI showed normal "a," "c," "x," "v," and "y" waves as opposed to case 4 with clinical TI whose arm veins showed a short "x" wave, a regurgitant wave (s) and a prominent "v" wave with sharp "y" descent. The reason why the venous pulsations were better seen on the right than on the left side is probably related to the right arm veins to right atrium and their relatively less curved course towards right atrium.

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