Clinical assessment of calibrated jugular pulse recording¹

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SUMMARY Calibrated jugular pulse tracings and cardiac catheterisation were performed on 50 consecutive patients with organic heart disease. Twenty normal adults had calibrated jugular pulse tracings and served as controls. The height of the jugular 'A' and 'V' waves was quantified as a percentage of a known displacement, rendering measurements independent of the amplification used in recording technique. A close correlation existed between the size of the calibrated jugular 'A' and 'V' waves and the actual magnitude of the right intra-atrial pressure changes (A wave, $r = 0.72$; V wave, $r = 0.80$). Patients who were free of pulmonary hypertension or tricuspid disease had calibrated 'A' and 'V' waves similar in height to the control population. An enlarged 'A' wave was usually associated with conditions restricting flow into the right ventricle. An enlarged 'V' wave generally indicated tricuspid regurgitation. These changes are often subtle and are not apparent in conventional venous tracings or at the bedside. We conclude that the calibrated jugular pulse tracings can accurately separate normal from abnormal venous waves and expand the diagnostic potential of the jugular pulse.

Examination of the jugular venous pulse allows the clinician to assess right heart haemodynamics. The 'A' wave, normally the tallest wave, has been noted to be abnormally enlarged relative to the 'V' wave in conditions restricting flow from the right atrium to the right ventricle (Luisada, 1954; Gibson and Wood, 1955; Kitchin and Turner, 1964; Wood, 1968), such as right ventricular hypertrophy and tricuspid stenosis. Similarly, the 'V' wave may be larger than normal in tricuspid regurgitation (Müller and Shillingford, 1954; Hartman, 1960), atrial septal defect (Reinhold, 1955; Haroutunian *et al.*, 1958; Tavel *et al.*, 1968), and congestive heart failure (Tavel, 1972). Identification of an enlarged 'A' or 'V' wave by conventional pulse recording depends upon the relative height of each wave. For this reason, a large 'A' wave might appear normal in the presence of an equally enlarged 'V' wave. Similarly, a normal 'A' wave might appear abnormally large in the presence of an abnormally

small 'V' wave. In order to circumvent these difficulties, we have developed a method to estimate the absolute height of each wave.

This report describes our experience with a new instrument to calibrate the pulse amplifier and to quantify the jugular venous pulse, expressing jugular 'A' and 'V' waves as a percentage of a known displacement. We validated this method by correlating it with actual intra-atrial pressure changes taken during catheterisation. In this manner, we were able to determine the upper limits of the normal 'A' and 'V' waves and to clearly separate normal from abnormal.

Subjects and methods

Calibrated jugular pulses and phonocardiograms were recorded on all patients undergoing cardiac catheterisation at the Indiana University haemodynamic laboratory during a 5-month period. Patients who had coronary angiography only were excluded. Sixty-seven consecutive patients had both jugular pulses and right atrial pressures recorded. There were 17 patients discarded because the jugular pulse tracing (10 cases) or right atrial pressure (7 cases) were not adequately recorded. The jugular pulse tracing was done within 24 hours of catheterisation in all cases.

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Fig. 1 Pulse transducer with attached pick-up device (funnel)—the distal system.

A group of 20 normal, healthy adults without cardiopulmonary disease (age range 20 to 53 years) acted as controls. A calibrated jugular pulse was obtained in each individual and was used to establish the limits of the normal 'A' and 'V' waves. None underwent cardiac catheterisation.

Jugular pulses were recorded using the standard method in our laboratory (Tavel, 1972). The pulse was recorded with the patient supine and the head slightly raised to relax the cervical musculature. Patients were instructed to suspend breathing in mid-expiration. The pick-up system consists of a new air-coupled, crystal microphone (Siemens-Elema Pulse/Phono Transducer 860, Fig. 1). This is connected to a pulse amplifier (Siemens-Elema EMT 30). With this system, the recorded pulse can be reduced or amplified with a 12-step selector. The electrical signals were recorded

Fig. 2 Volume calibrator attached to sensing head of the pulse transducer. Pressure applied manually will create a total displacement equivalent to 20 mm³.

graphically with an Electronics for Medicine recorder (Model DR 8) at a paper speed of 100 mm/s, and time lines set at 0.1 s intervals. All jugular pulses were recorded with a time constant of 4 s. After conclusion of each jugular recording, and with the amplification setting unchanged, the sensing head of the pulse/phono transducer was applied to a Siemens volume calibrator (calibration volume 20 mm³) (Fig. 2). The height of the resulting signal (in mm) was arbitrarily called 'y' (Fig. 3). The 'A' and 'V' waves were then quantified in mm and expressed as a percentage of 'y', independent of the amplification of the recorder. The equipment distal to the microphone was also standardised with the same amplification by carefully immersing the

Fig. 3 (A) Jugular pulse recording from control patient illustrating technique of measuring the 'A' and 'V' waves. (B) Displacement (y) created with volume calibrator with same amplification used to record jugular pulse. LSB, phonocardiogram from left sternal border; JUG, jugular pulse.

Fig. 4 (A) Jugular pulse recording in patient with tricuspid stenosis and regurgitation. In this example, the onset of the 'A' wave is taken 0.02 s from the peak of the 'P' wave on the electrocardiogram (see text). (B) Displacement (y) used to calibrate jugular 'A' and 'V' waves (0.48 y and 0.50 y , respectively). (C) Right atrial pressure tracing illustrating technique of measurement of 'a' and 'v' waves (see text). Note similarity in contour of the jugular pulse and the right atrial pressure recording.

funnel of the pick-up vertically into water to a preset depth and withdrawing quickly, taking care to avoid introduction of water into its interior. The resulting displacement signal was determined to bear a fixed ratio to our calibration signal, thereby ensuring that the distal system was working properly and was free of air leaks. Most jugular pulse tracings exhibited a clear-cut onset of the 'A' wave and no difficulty in measurement was encountered. In a small number of tracings, the onset of the 'A' wave was not clearly discernible. Based on experience with other tracings, in these it was taken 0.02 s after the peak of the 'P' wave on the electrocardiogram. The 'A' and 'V' waves were measured as seen in Fig. 3. The 'V' wave was measured in this manner and not from the 'X' trough because the 'X' trough is dependent on atrial relaxation and is attenuated in atrial fibrillation and tachycardia (Tavel, 1972). Each patient had several jugular pulse tracings performed with minor repositioning of the pick-up device. The best technical tracing with the greatest magnitude was used in all cases for calibration purposes. Several control patients had tracings performed on separate days by different technicians to ensure reproducibility of results.

Standard catheterisation techniques were used. Right atrial pressures were obtained during expiratory apnoea with saline-filled catheters connected to a Statham Model P23 pressure transducer and recorded on an Electronics for Medicine recorder (Model DR 8) at a paper speed of 50 mm/s. The reference level for all pressures was 5 cm below the angle of Louis. Cardiogreen dye curves were done for detection of intracardiac shunts or tricuspid

regurgitation if clinically suspected. An electrocardiogram was recorded during catheterisation. As seen in Fig. 4C, the peak of the 'P' wave of the electrocardiogram corresponds to the onset of atrial contraction in the pressure tracing. In our laboratory, pulmonary hypertension is considered present if the pulmonary artery systolic pressure is 38 mmHg or greater.

Results

Satisfactory jugular pulse tracings were obtained in 57 of 67 patients (85%) and right atrial pressure tracings in 60 of 67 (90%). Fifty patients had both recorded adequately and these were used in this study. In 4 cases, tachycardia (greater than 85 per minute) obscured the jugular 'V' wave and 'y' descent and in one case attenuated the 'A' wave (rate 100 per minute), though in most patients with tachycardia, however, the tracing was not distorted. Acquired valvular disease was present in 37 patients, congenital heart disease in 6, idiopathic hypertrophic subaortic stenosis and cardiomyopathy in 2 each, and 1 each with cor pulmonale, systemic hypertension, and mild constrictive pericardial disease. Sinus rhythm was present in 35, and atrial fibrillation in 15. Pulmonary hypertension or right ventricular pressure load was present in 28 (16 with sinus rhythm) and tricuspid regurgitation in 11.

Figs. 5 and 6 show the relation between the intra-atrial pressure recordings and the calibrated jugular waves. A significant correlation was obtained in each instance between the externally recordable calibrated pulse tracing and the intra-atrial tracing

Fig. 5 Relation between measured right atrial 'a' wave and calibrated jugular 'A' wave ($P < 0.001$).

Fig. 6 Relation between measured right atrial 'v' wave and jugular 'V' wave ($P < 0.001$).

(A waves, $r = 0.72$; V waves, $r = 0.80$). The correlation coefficient of the 'V' waves was calculated by excluding the single patient with severe tricuspid regurgitation and a 'V' wave of 33.5 mmHg. Though this calibrated jugular 'V' wave correlated well, it was felt that including this measurement would falsely increase the 'r' value. There was no relation between the mean right atrial pressure and the jugular pulse morphology.

In the 20 normal controls, the average calibrated 'A' wave was 0.26 y (range 0.12 to 0.38 y) and 'V' wave 0.22 y (range 0.07 to 0.36 y). The patients with organic heart disease in the absence of pul-

monary hypertension or tricuspid regurgitation had similar values. The average calibrated 'A' wave was greater in patients with pulmonary hypertension or right ventricular pressure load, but there was no direct relation between the degree of pulmonary hypertension and the height of the calibrated jugular 'A' wave. The largest 'A' waves were found in those patients with presumed reduced right ventricular compliance, such as subpulmonary

Fig. 7(a) Calibrated jugular 'A' wave in normal controls, pulmonary hypertension, or right ventricular pressure overload, and organic heart disease without pulmonary hypertension. (b) Calibrated jugular 'V' wave in normal controls, tricuspid regurgitation, and organic heart disease without tricuspid regurgitation.

Open circles represent patients with tricuspid stenosis. Both had pulmonary hypertension and tricuspid regurgitation.

stenosis (1), pulmonary stenosis (1), cardiomyopathy (1), and in one patient with idiopathic hypertrophic subaortic stenosis. Large 'A' waves were also seen in both patients with tricuspid stenosis, each also having pulmonary hypertension. A calibrated 'A' wave above 0.40 y was always associated with right ventricular pressure overload or tricuspid stenosis (Fig. 7A). The calibrated 'V' wave was greater in patients with tricuspid regurgitation than in either the controls or in the patients with organic heart disease without tricuspid regurgitation. In contrast to the 'A' wave in pulmonary hypertension, there was a direct relation between the estimated degree of tricuspid regurgitation and the height of the calibrated 'V' wave. Patients with minimal tricuspid regurgitation had 'V' waves which fell into the normal range and the calibrated pulse could not differentiate these from normal. A calibrated 'V' wave above 0.40 y was associated with tricuspid regurgitation with only 3 exceptions: endocardial cushion defect (1), mild constrictive pericardial disease (1), and severe mitral regurgitation of recent onset (1) (Fig. 7B).

Discussion

The jugular pulse tracing mimics closely the events in the right atrium, and tracings from both areas are similar in contour (Fig. 4). Morphological changes in the jugular pulse can help in the detection of haemodynamic disturbances in the right heart. Right ventricular pressure overload (Luisada, 1954; Gibson and Wood, 1955; Kitchin and Turner, 1964; Wood, 1968), tricuspid valvular disease (Müller and Shillingford, 1954; Gibson and Wood, 1955; Hartman, 1960; Kitchin and Turner, 1964), atrial septal defect (Reinhold, 1955; Haroutunian *et al.*, 1958; Tavel *et al.*, 1968), and constrictive pericardial disease (Lisa *et al.*, 1972) have been described as having characteristic changes in pulse contour. Recording a simultaneous phonocardiogram has allowed use of the jugular pulse in timing the right-sided third and fourth heart sounds (Tavel, 1972), the closure of the pulmonary valve, and the opening of the tricuspid valve (peak of 'V' wave) to assess the presence and degree of pulmonary hypertension (Hartman, 1960; Bamboa *et al.*, 1965; Burstin, 1967).

Although many attempts have recently been made to calibrate other pulse tracings (Reale, 1967; Sutton and Craige, 1967; Sutton *et al.*, 1970; Vetter *et al.*, 1972; Denef *et al.*, 1973, 1975; Van de Werf *et al.*, 1976), current methods of recording jugular pulses have not included calibration. Despite the numerous expected technical difficulties, such as obesity, respiratory variation,

and muscular tone, we have been able to obtain reproducible calibrated jugular pulse tracings in 57 of 67 patients (85%) and have validated their accuracy with intra-atrial pressure recordings (Figs. 5 and 6).

Calibrated jugular pulse tracings from 20 normal healthy controls were used to establish the upper limits of the normal 'A' and 'V' waves, expressed as a percentage of a known displacement (y). Any calibrated 'A' wave greater than 0.40 y is abnormal and in our experience was associated with right ventricular pressure load, pulmonary hypertension, or tricuspid stenosis. As illustrated in Fig. 7a, many patients with pulmonary hypertension had normal calibrated 'A' waves; therefore, this method is as sensitive as the right atrial pressure tracing in detecting resistance to right ventricular filling. There was no correlation between the degree of pulmonary hypertension and the magnitude of the calibrated 'A' wave. The largest 'A' waves were seen in conditions altering right ventricular compliance or obstructing inflow, i.e. tricuspid and pulmonary stenosis, cardiomyopathy, or idiopathic hypertrophic subaortic stenosis. Braunwald *et al.* (1964) have previously described large jugular 'A' waves in idiopathic hypertrophic subaortic stenosis and have attributed them to accompanying right ventricular disease.

Tricuspid regurgitation has been known to be associated with large 'V' waves which increase with inspiration or exercise (Müller and Shillingford, 1954). The venous congestion of heart failure gives rise to a high residual volume in the right atrium with an early appearing large 'V' wave (Tavel, 1972). Likewise, in atrial septal defect, a large 'V' wave may be seen relative to the 'A' wave (Reinhold, 1955; Haroutunian *et al.*, 1958; Tavel *et al.*, 1968). If associated pulmonary hypertension is present in tricuspid regurgitation or in atrial septal defect, as is commonly seen, the ratio A/V would lose its diagnostic potential. A similar problem might arise when tricuspid stenosis is associated with tricuspid regurgitation (Fig. 4). In atrial fibrillation, the characteristic changes associated with tricuspid regurgitation are subtle and those with atrial septal defect obliterated. In all but 3 instances, a calibrated 'V' wave greater than 0.40 y has separated patients with tricuspid regurgitation from normal and from other organic heart disease without tricuspid regurgitation, despite the presence of pulmonary hypertension or atrial fibrillation (Fig. 7b). As one might expect, many patients with clinically mild or unsuspected tricuspid regurgitation exhibited normal 'V' waves. Though there are currently no reliable, quantitative objective means to assess the exact amount of

tricuspid regurgitation, our overall impression was that jugular 'V' wave height did bear some relation to the degree of tricuspid regurgitation, as estimated not only from the height of the right atrial 'V' waves, but also from the clinical examination, that is character of the systolic murmur, mean venous pressure, degree of right ventricular enlargement, overall clinical status, etc. Large 'V' waves would also be expected in atrial septal defect and in constrictive pericardial disease (Lisa *et al.*, 1972) and, therefore, might not be separated by calibration from tricuspid regurgitation. Indeed, 2 of the 3 patients with large 'V' waves in the absence of tricuspid regurgitation had these conditions. The third had severe mitral regurgitation, and perhaps the giant left atrial 'V' wave was transmitted across the atrial septum and was seen in the jugular pulse.

In summary, we have shown the ability to obtain reproducible calibrated jugular pulse tracings, have validated them with intra-atrial pressure recordings, and clearly separated normal from abnormal venous waves. This simple method should expand the diagnostic potential obtainable from the jugular pulse.

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