and reverse flow. This directional capability may be of limited use in the pulmonary artery but should be particularly helpful when measuring blood flow in the aorta and inside the heart where both forward and backward flow might be expected. It is hoped that this approach will eventually allow the noninvasive measurement of volume blood flow in man.

References


Transcutaneous Doppler
Jugular Venous Flow Velocity Recording
Clinical and Hemodynamic Correlates

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SUMMARY Transcutaneous bidirectional Doppler jugular venous flow velocity patterns were classified and correlated in 82 patients with right heart hemodynamics. The normal forward flow pattern was biphasic with systolic flow (SF) greater than the diastolic flow (DF). With rare exceptions, flow patterns of SF = DF, SF < DF and DF alone indicated abnormal right heart hemodynamics. Abnormal flow patterns (SF = DF and SF < DF) seen in post cardiac surgery states, and in some rare patients with severe mitral regurgitation despite normal right-sided pressures, were probably secondary to postoperative change in right atrial compliance in the former and to a Bernheim effect in the latter. The most common cause of retrograde systolic flow in the absence of atrioventricular dissociation was tricuspid regurgitation. Persistent retrograde end-diastolic flow with normal forward flow was associated with high right atrial "a" wave pressures, indicating significant decrease in right ventricular compliance with a vigorous atrial contraction. The study clearly established that the jugular venous flow velocity pattern truly reflected derangements in the right heart hemodynamics, irrespective of the underlying etiology. The applicability to bedside evaluation of the jugular venous pulse and the right heart hemodynamics is emphasized.

MECHANISMS OF VENOUS RETURN AND RIGHT HEART FILLING have been of interest to clinicians since the days of Harvey and Purkinje.1,2 Many investigators have attempted to study variations in venae cavae/jugular flow in experimental animals3,4 and in both normal humans5,6 as well as in specific cardiac disorders.7,8-11 These measurements were carried out in some studies with differential manometers,3,5 bristle flow meters,6 or electromagnetic flow probes7,8,9-10,12,13,15-21 and implanted transistorsized pulsed ultrasonic flow meters.7,11

A new approach to study of flow velocities became available with the development of the Doppler ultrasonic flow meter.22-24 Although a number of studies have been carried out using Doppler flow velocity probes to determine mechanisms of right heart filling, the observations were confusing to the clinicians since many of the earlier studies utilized either unidirectional instruments, or bidirectional...
instruments, with unidirectional display.\textsuperscript{24-30} Further development of instrumentation has allowed accurate determination of flow direction.\textsuperscript{32} This has led to many interesting studies.\textsuperscript{32-40} Some of these studies have implied that certain patterns of the velocity recordings are diagnostic of specific cardiac lesions.\textsuperscript{34, 35, 37} Although correlation of jugular venous flow velocities with the right atrial pressure pulse contours have been reported, systematic correlation with the right heart hemodynamics has never been done.\textsuperscript{34-37} Furthermore, none of the previous studies have indicated the clinical applicability of these flow patterns to the bedside evaluation of the jugular venous pulse and right heart hemodynamics. This paper reviews our clinical experience with this noninvasive technique emphasizing the correlation of the venous flow patterns to right heart hemodynamics and the application of these observations to bedside care.

Methods and Materials

Jugular venous flow velocity recordings were made in 174 patients with cardiac conditions (table 1). Of these, 82 had hemodynamic measurements made during diagnostic heart catheterization. After careful examination of the jugular venous pulse contour with special attention to systolic and diastolic descents, flow velocity recordings were made using a Parks 800 bidirectional ultrasonic Doppler flow meter, and a 10 MHz probe. While this machine accurately records direction of flow, care must be taken to ensure proper balancing of the probe characteristics with the adjustments of the flow meter. In addition the accuracy of display of flow direction is checked against a radial arterial flow measurement. A special reversal switch added to our machine allowed representation of all flow toward the right atrium (away from the probe) as positive deflections and all flow away from the right atrium into the superior vena cava (toward the probe) as deflections below the zero line. The external probe was applied to the skin with gel at approximately 45° angle above the clavicle, between the two heads of sternomastoid attachments. For purposes of comparison, both external (jugular) as well as internal superior vena cava flow velocity recordings were made in a number of patients during heart catheterization. The latter was recorded using the catheter tip Doppler flow probe,\textsuperscript{41} introduced through the right antecubital vein. All recordings were made on a multichannel minograph 81 recorder at paper speed of 100 mm/sec. For timing phases of cardiac cycles, simultaneous recordings of an electrocardiogram, phonocardiogram and carotid pulse tracings were obtained, and where possible, a jugular venous pulse was also recorded.

In patients who had hemodynamic assessment, flow recordings were made within a day of cardiac catheterization. In those patients who had flow determination made during catheterization, simultaneous right atrial pressures were also recorded.

The variations in the jugular flow velocity pattern were identified by determining the following: a) monophasic or biphasic flow; b) systolic (SF) or diastolic (DF) flow; c) if flow is biphasic, dominance of the flow (SF > DF, SF = DF, or SF < DF); d) direction of flow — antegrade or retrograde; e) if retrograde, whether flow occurred during systole (Ret SF) or end-diastole, i.e., during atrial systole (Ret DF).

On the basis of these criteria, the variations in flow patterns obtained were correlated with the right heart hemodynamics.

| Table 1. Transcutaneous Jugular Flow Velocity Recordings: Diagnostic Categories |
|-----------------------------------|-------------------|
| Diagnosis                        | N     | Pts. with hemodynamic assessment |
| Ischemic heart disease           | 54    | 27                            |
| Valvular heart disease           | 68    | 30                            |
| Congenital heart disease         | 19    | 12                            |
| Cardiomyopathy                   | 17    | 7                             |
| Pericardial disease              | 8     | 3                             |
| Pulmonary hypertension           | 5     | 2                             |
| Myxoma                           | 3     | 1                             |
| Total                            | 174   | 82                            |

Results

Correlation of Flow Velocity Pattern with Right Heart Hemodynamics

Comparison of External Flow Velocity Recordings (Jugular) and Internal Flow Velocity Recordings (Superior Vena Caval)

In a number of individuals, internal superior vena caval flow velocity recordings were obtained in addition to external transcutaneous recording during heart catheterization. These allowed comparison of both flow patterns in the same patients. Tracings shown in figures 1A and B and 3A and B reveal very clearly that the externally recorded flow pattern truly reflected that recorded from the superior vena cava, except for slight transmission delay. Furthermore, these figures illustrate that antegrade flow toward the right atrium, although continuous, is also biphasic and occurs primarily during systole and diastole, corresponding each time to the fall in the right atrial pressure namely the x prime (x') and y descents of the right atrial pressure contour. Figure 6 shows a patient with a monophasic flow occurring primarily in diastole. When compared to the simultaneously recorded jugular venous pulse, it is seen to occur at the time of the y descent. Figure 1B shows the external systolic flow to reach its peak, near the time of the second heart sound (due to transmission delay from right atrium to the jugular vein). This complements the finding that the x' descent of the jugular venous pulse ends just before the second heart sound\textsuperscript{42} (fig. 2).

Correlation of Flow Patterns to Right Heart Hemodynamics (table 2)

Systolic Flow > Diastolic Flow (SF > DF)

This flow pattern (fig. 1A, 1B and fig. 2) was noted in 62 patients, of whom 29 had hemodynamic assessment. Right heart hemodynamics of all these 29 patients were completely normal, thus establishing the normality of this jugular flow velocity pattern. This correlated well with the normal jugular pulse tracings which always showed a larger x' descent than y descent (fig. 2). All flow patterns to be discussed below represented deviations from the normal as shown above.
Systolic Flow = Diastolic Flow (Sf = Df)

This flow pattern (figs. 3, 4) was recorded in 25 patients, 13 of whom had hemodynamic assessment. This correlated with equal x' and y descent in jugular pulse tracings. Four of these 13 had normal right heart hemodynamics (group A) and all four were post cardiac surgery patients (fig. 4). The remaining nine patients (group B) however, showed abnormal right heart hemodynamics with elevation in mean right atrial pressure (compared to those with Sf > Df, P < 0.025) as well as pulmonary artery pressure (compared to those with Sf > Df, P < 0.05).

Systolic Flow < Diastolic Flow (Sf < Df)

Figure 5 illustrates this flow pattern which was recorded in 46 patients of whom 23 had hemodynamic assessment. This was seen in jugular pulse tracings as a dominant y descent. The correlation with the right heart hemodynamics, as shown in table 2, divides these patients into two subgroups A and B. Group A with normal right heart hemodynamics included ten post cardiac surgery patients and two patients with severe mitral regurgitation. Group B patients however had abnormal right heart hemodynamics. When compared with patients who had Sf > Df, their mean right atrial pressure and pulmonary systolic pressures were significantly higher (P < 0.0025 and < 0.025 respectively). Three patients in this group had atrial fibrillation. Thus, both patterns Sf = Df and Sf < Df in unoperated patients represented an abnormal state of right heart hemodynamics except in rare instances of severe left ventricular volume overload.

Single Diastolic Flow (Df)

This pattern correlated with a jugular pulse tracing showing only a systolic wave and y descent. It was seen in 24 patients (fig. 6). All 11 who had hemodynamic assessment had abnormal right heart hemodynamics. Mean right atrial pressure, right atrial v wave, right ventricular end-diastolic pressure and pulmonary artery pressure, all differed significantly from patients with normal flow (P < 0.01,
P < 0.0005, P < 0.005, and P < 0.025 respectively). Eight were in atrial fibrillation. During systole no retrograde or significant antegrade flow was noted in these patients.

Retrograde Systolic Flow (Ret Sf)

Seventeen patients had retrograde systolic flow (Ret Sf). Six of these patients had hemodynamic assessment. While three of 17 patients had retrograde flow during cannon waves associated with atrioventricular dissociation (fig. 7), 11 had clinical evidence of significant tricuspid regurgitation. The forward flow in the latter group of patients occurred only in diastole as expected (fig. 8). In patients with atrioventricular dissociation, forward flow varied depending on the relationship of atrial and ventricular systole, i.e. P-R relationship (fig. 7). Separate flow in diastole, associated with the atrial relaxation (indicated by large arrows in fig. 7), can be seen clearly when P-R interval is long.

Retrograde Diastolic Flow During Atrial Systole (Ret Df)

Persistent retrograde flow during atrial systole was rarely seen in normal subjects either in the superior vena caval or jugular flow recordings. In an occasional subject, it occurred only during end expiration. However, it was constantly noted in 18 patients, nine of whom had hemodynamic assessment (fig. 9). The right heart hemodynamics of these patients are of interest in that they showed a high a wave

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**Figure 3.** A) The superior vena caval (SVC) flow velocity recorded through a catheter tip bidirectional Doppler flow probe in a patient with chronic constrictive pericarditis with simultaneous right atrial (RA) pressure. The systolic flow (Sf) and the diastolic flow (Df) are about equal and correspond to the equally dominant x' and y descents in the RA pressure pulse. The scale for the latter is in mm Hg. B) Externally recorded jugular venous flow velocity (V. Flow ext.) pattern from the same patient with the internal SVC flow shown in panel A demonstrates an equal systolic flow (Sf) and diastolic flow (Df) corresponding to the equally dominant x' and y descent of the right atrial (RA) pressure pulse, except for transmission delay. The similarity in contour between the external and internal recording is obvious.

**Figure 4.** A) Externally recorded jugular venous flow (V. Fl) velocity curve in a patient with coronary artery disease with normal right heart hemodynamics prior to aortocoronary bypass surgery, showing normal flow pattern with a dominant systolic flow (Sf), which is greater than the diastolic flow (Df). II = lead II ECG; CP = carotid pulse tracing; Ph = phonocardiogram. B) Jugular venous flow (V. Fl) velocity curve obtained from the same patient showed in panel A, after the aortocoronary bypass surgery shows the change in the venous flow velocity contour. The diastolic flow (Df) is equal to or slightly greater than the systolic flow (Sf).
pressure in the right atrium (mean 15 ± 3.0 mm Hg) with a mean right atrial pressure which was only mildly elevated (mean 7 ± 2.0 mm Hg) in the presence of pulmonary hypertension (systolic 66 ± 12.0 mm Hg/diastolic 31 ± 7.0 mm Hg). Of these 18 patients, 15 had normal forward flow pattern (SF > DF) while two had SF < DF, and one had SF = DF. The predominance of normal flow pattern in this group of patients with retrograde end-diastolic flow indicates a compensated right heart with strong right atrial contraction in the presence of decreased right ventricular compliance.

**Flow Patterns in Patients with Pulmonary Hypertension**

Thirty-five patients had significant pulmonary hypertension, (systolic 65 ± 4.0 mm Hg/diastolic 30 ± 2.0 mm Hg). Twenty-six of these had abnormal flow patterns (SF = DF in 4, SF < DF in 9, and single DF in 8 and Ret SF in 5 and retrograde DF during atrial systole in 1). Of the nine remaining patients with normal forward flow pattern (SF > DF), five had retrograde end DF and 4 did not. The right heart hemodynamics of these two groups of patients, although showing comparable degrees of pulmonary hypertension (table 3), showed the a wave in right atrial pressure to be significantly higher in the group with retrograde DF (P < 0.01). Thus, except in four patients, the altered hemodynamics of pulmonary hypertension were detectable.

**Table 2. Correlation of Jugular Flow Velocity Patterns to Right Heart Hemodynamics**

<table>
<thead>
<tr>
<th>Flow patterns</th>
<th>N</th>
<th>Pts with HA</th>
<th>a RAP (mm Hg)</th>
<th>v RAP (mm Hg)</th>
<th>mean RAP (mm Hg)</th>
<th>RVEDP (mm Hg)</th>
<th>PAP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SF &gt; DF</td>
<td>62</td>
<td>29</td>
<td>6.6</td>
<td>4.7</td>
<td>4.4</td>
<td>6.6</td>
<td>33.8</td>
</tr>
<tr>
<td>SF = DF</td>
<td>25</td>
<td>13</td>
<td>5.5</td>
<td>4.8</td>
<td>2.8</td>
<td>5.5</td>
<td>22.7</td>
</tr>
<tr>
<td>SF &lt; DF</td>
<td>46</td>
<td>23</td>
<td>4.9</td>
<td>3.8</td>
<td>2.6</td>
<td>4.9</td>
<td>27.0</td>
</tr>
<tr>
<td>Df</td>
<td>24</td>
<td>11</td>
<td>13.6</td>
<td>13.0</td>
<td>9.8</td>
<td>13.6</td>
<td>53.2</td>
</tr>
<tr>
<td>Ret. SF*</td>
<td>17</td>
<td>6</td>
<td>15.1</td>
<td>8.7</td>
<td>7.2</td>
<td>15.1</td>
<td>66.3</td>
</tr>
<tr>
<td>Ret. DF†</td>
<td>18</td>
<td>9</td>
<td>15.1</td>
<td>8.7</td>
<td>7.2</td>
<td>15.1</td>
<td>66.3</td>
</tr>
</tbody>
</table>

*Forward Flow Diastolic.
†Forward Flow: SF > DF in 15; SF < DF in 2; SF = DF in 1.

Abbreviations: DF = diastolic flow; HA = hemodynamic assessment; PAP = pulmonary artery pressure; RAP = right atrial pressure; Ret DF = retrograde end-diastolic flow; Ret SF = retrograde systolic flow; RVEDP = right ventricular end-diastolic pressure; SF = systolic flow.
in flow recordings. Lack of retrograde flow during atrial systole in these four patients despite significant pulmonary hypertension may indicate atrial disease and consequent lack of strong atrial contraction. This is supported by the fact that two of them had been in an atrial arrhythmia (fibrillation and flutter) requiring cardioversion.

**Flow Pattern in Patients with Atrial Fibrillation**

Of 27 patients with atrial fibrillation, nine had retrograde Sf, confirming clinical tricuspid regurgitation. Of the remaining 18 patients, 13 had monophasic forward diastolic flow and five had biphasic flows with the Sf < Df. The right heart hemodynamics, however, were abnormal in all 11 patients who had the assessment. This illustrates the well known fact that the lack of atrial contraction does not abolish forward systolic flow, i.e., an x' descent may still be preserved. It also points out that the abnormality in flow pattern in patients with atrial fibrillation may reflect the long standing nature of the underlying condition and the associated derangement in the right heart hemodynamics rather than an effect of the arrhythmia itself.

**Flow Pattern in Cardiac Tamponade**

In three patients with confirmed cardiac tamponade with classical clinical signs, recording jugular venous flow velocity was extremely difficult. In all of them only a low amplitude forward flow could be recorded during systole and this was confined to the peak of inspiration.

**Flow Patterns and Hemodynamic Possibilities**

The different flow patterns obtained are shown as a diagram in figure 10. The biphasic forward flow with dominant systolic flow (Sf > Df) is normal, while flow patterns of Sf = Df, Sf < Df, and single Df were abnormal. The abnormal flow patterns of Sf = Df and Sf < Df were however

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**Table 3. Pressure Parameters in Patients with Pulmonary Hypertension with Normal Forward Flow Pattern (Sf > Df)**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patients with</th>
<th>Patients without</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA a</td>
<td>20.0 ± 2.7</td>
<td>10.6 ± 1.5</td>
</tr>
<tr>
<td>RA Mean</td>
<td>10.8 ± 2.5</td>
<td>7.0 ± 1.0</td>
</tr>
<tr>
<td>PA Systolic</td>
<td>92.3 ± 11.6</td>
<td>79.6 ± 8.2</td>
</tr>
<tr>
<td>Diastolic</td>
<td>47.5 ± 10.3</td>
<td>29.6 ± 5.4</td>
</tr>
</tbody>
</table>

*All values are mean mm Hg ± SEM.*

Abbreviations: Df = diastolic flow; PA = pulmonary artery; RA = right atrial; Ret Df = retrograde end-diastolic flow; SEM = standard error of the mean; Sf = systolic flow.
postcardiac grade systolic underlying etiology. Certain pattern flow are variations seen only present study of diagnostic not noted in systematic attempt (Ret indicates high right atrial contraction. As expected there will be no controversy as to the mechanism of diastolic inflow which occurs with tricuspid valve opening, controversy and discussion have long centered around the cause of the more dominant systolic flow in normal subjects. Our observations support the concept that systolic flow is caused by two factors, namely atrial relaxation and descent of the base of the ventricle (tricuspid ring with a closed tricuspid valve) during active ventricular systole: the latter being dominant factor. Atrial relaxation flows have been shown by us as well as by others to occur during periods of atrioventricular dissociation or in patients with long P-R intervals (fig. 7). In the normal condition however, the atrial relaxation flow is only seen occasionally on Doppler tracings as a notch on the upstroke of the systolic flow wave (fig. 2), corresponding to the x descent, whereas the peak of the systolic flow as confirmed by internal pressure and flow recordings, always corresponds to the x' descent. The latter is due to the descent of the base during ventricular systole and must be distinguished from the x descent which is due to atrial relaxation. Preservation of systolic flow in patients with atrial fibrillation in this study as well as those of others further supports this concept. While some authors have suggested the lack of dominance of systolic flow in atrial fibrillation to be secondary to lack of atrial relaxation, it is probably related to a decreased Starling effect on the ventricle, due to loss of atrial systole, in turn leading to decreased ventricular contractility and diminished descent of the base.

Variations from the normal dominant systolic flow (namely, $Sf = Df$ and $Sf < Df$) are associated with abnormal right heart hemodynamics with elevation of right atrial pressures and right ventricular end-diastolic pressures (fig. 10 and table 2), except in post cardia surgery states and rarely in patients with severe left ventricular volume overload. In the post cardia surgery patients, the abnormality in flow patterns in the presence of normal right heart pressures is best explained by a decrease in the compliance of the right atrium induced iatrogenically. Both its contractility and relaxation may conceivably be affected, contributing to diminution of sstolic flow. The stiffness of the atrium may further help build up "v" wave pressures during systole and add to the diastolic flow velocity. Since the mean "v" wave pressures in the atrium in these patients are not significantly different from the normal, this suggests the former is probably more important. In patients with severe left ventricular volume overload, the reason for a change in flow pattern from the normal, despite the normal right heart hemodynamics, is perhaps explainable on the basis of a Bernheim effect on the atrial septum: the septum may bulge into the right atrium due to the high regurgitant volume and pressure build up in the left atrium during ventricular systole, causing restriction of flow into the right side. Both patients who had this feature had severe mitral regurgitation.

**Figure 10. Jugular venous flow patterns with electrocardiogram, carotid pulse tracing and phonocardiogram for time reference. Df = diastolic flow; ret. Sf = retrograde systolic flow; ret. Df = persistent retrograde end-diastolic flow; Sf = systolic flow. * Seen in postcardiac surgery patients and in some rare patients with severe mitral regurgitation despite normal right heart pressures. ** Indicates high right atrial a wave pressure with only mild elevation in mean pressure.**

noted in postcardiac surgery patients and in some rare patients with severe mitral regurgitation despite normal right heart pressures. Tricuspid regurgitation caused retrograde systolic flow. Persistent retrograde end-diastolic flow (Ret Df) in the absence of tricuspid obstruction was related to the high right atrial "a" wave, indicating significant decrease in right ventricular compliance with a vigorous atrial contraction. As expected a specific influence of the underlying etiology of the heart disease could not be directly related to any of these flow patterns.

**Discussion**

Although a few studies with bidirectional Doppler have emphasized certain variations in flow patterns to be diagnostic of specific cardiac disorders, none has attempted a classification of flow velocity patterns based on systematic correlation to right heart hemodynamics. The present study establishes clearly that flow patterns and their variations are closely related to right heart hemodynamics and truly reflect derangements in the latter, irrespective of underlying etiology.

Normal right heart hemodynamics were associated with a flow pattern of $Sf > Df$ (fig. 1, 10). Retrograde flow was seen only during atrial systole and in a small percentage (10%) of normal patients. In our studies it occurred only during end expiration, unlike that described by Kalmanson et al. Our findings however are very similar to those described by Froysaker, who measured flows with electromagnetic flow meters as well as those of Benchimol using bidirectional Doppler technique.

The biphasic flow pattern with dominant flow during systole is well recognized. While there is no controversy as to the mechanism of diastolic inflow which occurs with tricuspid valve opening, controversy and discussion have long centered around the cause of the more dominant systolic flow in normal subjects.

Our observations support the concept that systolic flow is caused by two factors, namely atrial relaxation and descent of the base of the ventricle (tricuspid ring with a closed tricuspid valve) during active ventricular systole: the latter being dominant factor. Atrial relaxation flows have been shown by us as well as by others to occur during periods of atrioventricular dissociation or in patients with long P-R intervals (fig. 7). In the normal condition however, the atrial relaxation flow is only seen occasionally on Doppler tracings as a notch on the upstroke of the systolic flow wave (fig. 2), corresponding to the x descent, whereas the peak of the systolic flow as confirmed by internal pressure and flow recordings, always corresponds to the x' descent. The latter is due to the descent of the base during ventricular systole and must be distinguished from the x descent which is due to atrial relaxation. Preservation of systolic flow in patients with atrial fibrillation in this study as well as those of others further supports this concept. While some authors have suggested the lack of dominance of systolic flow in atrial fibrillation to be secondary to lack of atrial relaxation, it is probably related to a decreased Starling effect on the ventricle, due to loss of atrial systole, in turn leading to decreased ventricular contractility and diminished descent of the base.
The other deviation from the normal flow pattern is that of a single Df. This is seen in subjects with a jugular pulse showing only a "v" wave and y descent (fig. 6). In these subjects, right ventricular end-diastolic pressures and right atrial pressures were invariably elevated, along with an elevation of pulmonary artery pressures in a number of these subjects (table 2). High "v" wave pressures in these patients with absent or insignificant forward flow during systole indicated not only decreased right atrial compliance but also decreased right ventricular function with poor descent of the base. This was further supported by the fact that the majority of patients were in atrial fibrillation with valvular heart disease and under treatment for congestive failure, which reflected the severity of the condition. The lack of atrial contraction further decreased the contractility of the right ventricle and helped to eliminate the descent of the base and systolic forward flow. Atrial fibrillation in these patients is not however the primary cause of this flow pattern. This flow pattern occurred in three patients who were in sinus rhythm with congestive cardiomyopathy (fig. 6).

Of 37 patients who had single Df, 14 were associated with tricuspid regurgitation, which were all confirmed by the presence of retrograde SF in the absence of atrioventricular dissociation (figs. 8 and 10). Since the right atrial "v" wave pressure of patients with and without tricuspid regurgitation were similar, its height was of little value in distinguishing between the two. Therefore, the jugular flow velocity tracing was extremely helpful. Reverse flow into the vena cava during systole has been shown to indicate tricuspid regurgitation by many previous investigators using a variety of techniques. We Doppler jugular flow velocity recordings is probably the best noninvasive tool available for clinical confirmation of this diagnosis. Clinically, the volume and the rate of rise of the "v" wave (jugular) is larger and faster in those with tricuspid regurgitation.

Of 18 patients with persistent retrograde flow during atrial systole (retrograde end-diastolic flow, Ret Df) not phasic with respiration, 15 were associated with a normal forward flow pattern (fig. 10). It was related to the very high mean right atrial "a" wave, indicating significant decrease in right ventricular compliance with a vigorous right atrial contraction (table 2). The preservation of normal flow (SF > Df) in these patients indicates a normally functioning right ventricle. On the other hand decumpensation of the right ventricle in the presence of pulmonary hypertension was often associated with abnormal flow patterns (31 of 40 patients) and only three of these 31 patients had retrograde flow during atrial systole (10%). However, five of the nine remaining patients with the decumpeated right ventricle and with normal systolic flow patterns had persistent retrograde diastolic flow during atrial systole. Thus, serial jugular flow velocity recordings may be of help in longitudinal follow-up and detection of early evidence of decumpeation in patients with pulmonary hypertension. Loss of retrograde flow during atrial contraction plus abnormal forward flow indicates advanced right ventricular failure.

Restriction to right ventricular filling may vary in degree and may be due to various causes (pericardial, myocardial and endocardial). If it is limited to late diastole, the only abnormality seen in the right heart hemodynamics will be elevation of the right atrial "a" wave pressure. When restriction to ventricular filling encroaches more and more into early diastole, elevation of both mean right atrial pressure and "v" wave pressure will result. This will lead to an augmented early diastolic inflow velocity due to a higher pressure head at the time of the tricuspid valve opening, i.e., during the rapid filling phase. As long as ventricular function is preserved, the increase in diastolic flow due to the restriction will equal systolic flow (fig. 3A). In contrast, patients with myocardial dysfunction and diminished contractility in the presence of restriction will be characterized by diminished systolic flow and augmented diastolic flow (fig. 5). When restriction is severe (as in tamponade) and occurs throughout diastole, no inflow can occur into the atrium during this phase. This totally abolishes diastolic flow into the atrium and therefore patients with tamponade exhibit inflow only during ventricular systole, when the ventricular size is least, allowing some expansion and filling of atrium within the pericardial sac. Jugular flow velocity recordings in patients with cardiac tamponade, which is not uncommon in the postoperative state, are extremely difficult to record due to diminished flow velocity. Thus, the only forward flow recognizable would occur in systole and only during inspiration. This was the case in all three patients in our study.

**Bedside Recognition**

Since systolic and diastolic jugular flows correspond to the x' and y descent of the jugular venous pulse, both of which have relatively equal transmission delay, the recognition of these at the bedside can be made easily by observing the descents of the jugular venous pulse and determining their relationship to the phases of the cardiac cycle. The latter is easily accomplished by timing the descent with the arterial pulse or the second heart sound. This recognition is made easier by the fact that the descents of the jugular pulse are generally rapid movements reflecting acceleration of flow velocity whereas ascents are often slow and difficult to appreciate. The x' descent corresponds to the systolic flow (SF) into the right atrium and in sinus rhythm follows the "a" wave. Due to transmission delay, this descent falls almost on to the second heart sound instead of landing in midsystole (fig. 2). It often coincides with the upstroke of the radial pulse. The diastolic y descent (which follows the peak of the "v" wave) reflects diastolic flow (DF) and falls well after the second heart sound is heard (about 150 msec). Thus, it should be easy to determine the flow pattern by observing the relative dominance of x' and y descents of the jugular venous pulse.

In normal subjects systolic flow is dominant. Therefore, the x' descent is the major descent appreciable at the bedside, and is often the only descent seen. Although the smaller diastolic flow is recordable, its corresponding y descent is often not seen. In fact, a prominent y descent probably represents an abnormal increase in diastolic flow velocity. If two descents are visible for each cardiac cycle, in the majority of instances it will turn out to be the x' vs y descents, unless the x descent is far separated from the x' descent, as with very long P-R intervals. In cases of double descents, the relative dominance of the x' vs y will easily help in identifying the flow pattern. On the contrary, the normal "a"
and “v” wave ascents representing the relatively slow slope of rise, are not as easily identifiable at the bedside. If the rise of the waves are as rapid as the descents and associated with an increased amplitude, then this indicates probable retrograde flow as seen in tricuspid regurgitation, cannon waves of atrioventricular dissociation, and in some cases of diminished right ventricular compliance with a strong atrial contraction (giant “a” waves). Rapid rise of a large amplitude jugular “a” wave would also be expected in patients with tricuspid stenosis, although we did not have a patient with this condition in our study.

Thus, an understanding of the jugular venous flow patterns makes examination of the jugular venous pulse contour meaningful and provides important information regarding right heart hemodynamics.

Clinical Value of Doppler Technique

The Doppler technique is noninvasive, accurate and easy to record in most patients, although care must be taken to avoid technical errors. Particular attention must be paid to proper angulation of the probe. Although an experienced observer can predict with relative accuracy flow patterns from the bedside assessment of the descents in the jugular venous pulse, the beginners and trainees often may find it difficult to determine dominance of systolic versus diastolic descents and therefore will be unable to predict flow patterns. Similar difficulty may be encountered even by the experienced observer in the presence of tachycardia, extremely low (secondary to diuretics) or high venous pressures. Since lack of or decrease in SF usually means decreased right ventricular function, it becomes important to make this assessment. Here the Doppler technique is not only of value in confirming clinical impression but also in documenting the flow patterns for follow-up purposes.

In all of our patients where both clinical assessment of the jugular descents and Doppler flow studies were possible, there was 100% correlation. The Doppler technique however proved to be more sensitive in approximately 5-10% of patients where for some of the various reasons cited the clinical assessment was difficult. Furthermore, Doppler recording is of particular value in the differentiation of the high “v” wave of heart failure and atrial fibrillation from that caused by tricuspid regurgitation. Demonstration of retrograde SF by Doppler is indispensible evidence for the presence of tricuspid regurgitation, while the “v” wave of heart failure is associated with forward flow during systole. Finally, serial Doppler recordings showing a progressive change in degree of descent of the base may be of value in early detection of depressed right ventricular function.

Acknowledgment

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Assessment of the Pulmonary Vascular Bed by Echocardiographic Right Ventricular Systolic Time Intervals

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SUMMARY Echocardiography was used to measure right ventricular systolic time intervals (RVSTI) in 85 normal children (group I) and in 229 patients undergoing cardiac catheterization (group II). Corrected right ventricular pre-ejection period (RPEP,) and right ventricular ejection time (RVET,) (based on regression analysis of group I) and RPEP/RVET were each correlated with pulmonary artery (PA) diastolic and mean pressures and pulmonary vascular resistance (PVR). The best correlation (0.83) was between a second degree polynomial of the RPEP/RVET and PA diastolic pressure. The RPEP/RVET allowed prediction of PA diastolic pressure within 10 mm Hg in 85% of the patients. The utility of RPEP/RVET was confirmed in sequential data of 22 patients, in whom alteration in RPEP/RVET accurately reflected the changing PA diastolic pressure. The RPEP/RVET could not be used to assess PA pressure in six patients with congestive cardiomyopathy nor in 18 patients with complete right bundle branch block (CRBBB).

THE ASSOCIATION BETWEEN ELEVATED PULMONARY ARTERY (PA) PRESSURE and characteristic alterations in the pulmonic valve echocardiogram has been established. Qualitative assessment of the pulmonic valve echogram in patients with pulmonary hypertension has included loss of the atrial systolic “a” dip and increased rate of cusp opening. Nanda et al. described lengthening of the right ventricular pre-ejection period (RPEP), indexed for heart rate (HR), in adult patients with elevated PA pressure. Hirschfeld et al. measured the RPEP and the right ventricular ejection time (RVET) in 64 pediatric patients undergoing cardiac catheterization and demonstrated a strong correlation between PA diastolic pressure and the ratio, RPEP/RVET (r = 0.72). Silverman et al. analyzed the same data and suggested that RPEP, corrected for HR, provided better correlation with pulmonary vascular resistance than did the RPEP/RVET. Recently, Johnson et al. measured both the RPEP and the interval from the Q wave of the electrocardiogram (ECG) to tricuspid valve closure (QT), in patients undergoing cardiac catheterization. The derived ratio, (RPEP – QT)/RVET, showed excellent correlation (r = 0.80) with PA diastolic pressure and could be applied in patients with complete right bundle branch block (CRBBB).

This study examined a larger series of patients undergoing cardiac catheterization to determine the sensitivity of each right ventricular systolic time interval (RVSTI) in the prediction of PA pressure and PVR. Since the RPEP is prolonged in patients with CRBBB or congestive cardiomyopathy, the evaluation of the pulmonary vascular bed in patients with these conditions has been a problem. A small number of children with CRBBB or cardiomyopathy were included in the study to determine whether RVSTIs could be used in the evaluation of their PA pressure and PVR.

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