Multilevel Atrioventricular Block

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SUMMARY Arrhythmias displaying conduction disturbances consistent with block at more than one level within the atrioventricular conduction system were seen in 36 patients during a two-year period in a community hospital. Two levels of block were postulated in each patient; one patient also demonstrated a third level of block.

In 24 patients (Type A), there was an integral conduction ratio at the upper level of block. This pattern was most frequently seen in atrial flutter (mean atrial rate 284 ± 35 beats/min) with 2:1 block at the upper level and Wenckebach at the lower level. Progression to 2:1 block at the lower level resulted in 4:1 block. Twelve patients (Type B) had a nonintegral conduction ratio of their block at the upper level with a mean atrial rate of 162 ± 62 beats/min. Their conduction patterns consisted of Wenckebach block at the upper level with either integral (2:1) or nonintegral (Wenckebach) block at the lower level.

The presence of multilevel block was not related to specific etiologic diagnoses, medications, or electrolyte patterns. It is suggested that multilevel block is a common, frequently transient, conduction pattern seen in a variety of clinical conditions. It is readily recognized from a standard electrocardiogram and, of itself, has no short-term detrimental prognostic implications.

RECENT EXPERIMENTAL AND CLINICAL STUDIES have pointed to the existence of block at more than one level within the atrioventricular (A-V) conduction system. Clinically, this has been exemplified primarily by an electrocardiographic rhythm displaying Wenckebach periodicity of alternate beats. An early example of this rhythm appeared in 1912 in Sir Thomas Lewis' classic report on auricular flutter. During the next 20 years, further examples were presented by Blackford et al., Parkinson et al., and MacMillan et al. In 1948, Langendorf emphasized the concept of "concealed conduction" as a tool in analyzing a variety of complex arrhythmias, that could not be readily explained by a single level of block in the A-V conduction system. Besoin-Santander and associates studied atrioventricular conduction patterns in atrial flutter and schematically displayed the occurrence of the Wenckebach phenomenon affecting alternate beats. They attributed this to a "double region of block ... and halving of the atrial rate at a higher level of the A-V junction and block with the Wenckebach phenomenon at the lower."

Halpern and associates recently reported five cases of Wenckebach periods of alternate beats in patients who had intraventricular conduction defects and who were prone to develop complete heart block. They concluded that alternating Wenckebach was most commonly due to disease below the A-V node and therefore was associated with a bad prognosis. Hartzler and Maloney demonstrated alternate-beat Wenckebach in the supr-His region during rapid atrial pacing in a patient with the sick sinus syndrome. Amat-y-Leon and associates induced alternating Wenckebach periodicity by means of rapidly pacing the atria in 13 patients. They demonstrated several types of two-level A-V block and postulated that such phenomena could occur spontaneously.

This report deals with the spontaneous occurrence of a group of arrhythmias which can be best explained by postulating the occurrence of block at more than one level within the atrioventricular conduction system. These rhythms were observed in 36 patients during a two-year period at St. Elizabeth's Hospital of Boston. Examples of several different arrhythmias are shown and their postulated mechanisms are explained utilizing the concept of multilevel A-V block. It is our contention that multilevel block is a common occurrence, which may be present in many patients with high degree A-V block.

Materials and Methods

The clinical material in this study was obtained from patients seen by the cardiology service of St. Elizabeth's Hospital of Boston in the course of routine consultative or hospital care. During a two-year period, in this 415 bed general community hospital, patients with arrhythmias displaying complex patterns of atrioventricular conduction were identified. Patients whose arrhythmias were interpreted as displaying multilevel A-V block constitute the material for this study.

In all instances, the arrhythmias were noted on routine electrocardiograms, rhythm strips placed in the hospital chart, or monitoring records obtained in the intensive care units. Clinical information, and the results of laboratory tests performed at about the time of the occurrence of the arrhythmia, were recorded for each patient.

One patient underwent further electrophysiologic evaluation after granting informed consent. A bipolar electrode catheter was introduced percutaneously via the right femoral vein and positioned in the region of the His bundle. Filtered (50 to 500 Hz) intracardiac electrograms as well as electrocardiographic leads I, aVF, and V5 were simultaneously recorded on a multichannel photographic recorder at a paper speed of 100 mm/sec.

Results

Multilevel atrioventricular block was demonstrated in 36 patients. Clinical, laboratory, and electrocardiographic data on these patients are summarized in table 1.

The patients were classified based on the nature of the conduction defect at their uppermost level of block. Twenty-four patients displayed an integral conduction ratio with a regular response (2:1 in all instances) at their upper level (Type A), whereas 12 patients had a nonintegral conduction ratio with an irregular response (e.g., 5:4, 4:3) at this level.
Table 1. Clinical and Laboratory Data on Patients with Multilevel A-V Block

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Type</th>
<th>Age/Sex</th>
<th>Diagnosis</th>
<th>Atrial rate (beats/min)</th>
<th>QRS duration (sec)</th>
<th>Conduction* pattern</th>
<th>Cardiac medication</th>
<th>Serum digoxin level (ng/ml)</th>
<th>Serum K+ mEq/L</th>
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<tr>
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<td>A</td>
<td>80/F</td>
<td>COPD, CAD</td>
<td>288</td>
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<td>2:1/W</td>
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<td>3.9</td>
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<td>A</td>
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<td>RHD</td>
<td>280</td>
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<tr>
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<td>3.8</td>
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<td>digoxin</td>
<td>8.0</td>
<td>3.2</td>
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</table>

Dig Tox

18 A 73/F CAD 300 0.09 2:1/W digoxin — 3.9
19 A 77/M B-TS 280 0.10 2:1/W propranolol — 3.0
20 A 63/M RHD, CAD 300 0.09 2:1/W digoxin 1.5 4.6
21 A 72/M CAD 167 0.10 2:1/W quinidine — 4.6
22 A 56/M CAD 330 0.08 2:1/W digoxin — 4.2
23 A 64/M COPD, CAD 288 0.08 2:1/W digoxin — 4.6
24 A 64/M CAD, AI 300 0.10 2:1/W digoxin 2.2 4.6
25 B 73/M COPD 220 0.09 W/2:1 propranolol — 3.8
26 B 86/F CAD, AMI 125 0.09 W/2:1 chlorothiazide 3.7
27 B 89/F CAD 130 0.08 W/2:1 — 3.4
28 B 54/M ABE 200 0.10 W/2:1 digoxin 3.8 4.5
29 B 60/M CAD 90 0.10 W/2:1 — 4.0
30 B 71/M CAD, HT 250 0.09 W/2:1 digoxin 1.6 3.3
31 B 80/M HT 300 0.10 W/2:1 digoxin 2.2 3.5
32 B 81/M HT 130 0.16 W/2:1 — 3.6
33 B 80/M CAD 170 0.08 W/2:1 — 4.6
34 B 71/F B-TS 170 0.08 W/2:1 — 3.1
35 B 83/F HT 95 0.08 W/2:1 chlorothiazide 2.5
36 B 78/M CAD 120 0.13 W/2:1 (3:1) digoxin 2.5 5.3

*Type of block at each level, separated by a slash. W = Wenckebach.
Abbreviations used: COPD = chronic obstructive pulmonary disease; CAD = coronary artery disease; Dx = diagnosis; RHD = rheumatic heart disease; HT = hypertension; CM = cardiomyopathy; PAF = paroxysmal atrial flutter; Dig Tox = digitalis toxicity; B-TS = bradyarrhythmia-tachyarrhythmia syndrome; AI = atrial insufficiency; AMI = acute myocardial infarction; ABE = acute bacterial endocarditis.

(Type B). Three patients displayed two different Type B conduction patterns.

Type A: Integral Conduction Ratio at Upper Level

The mean age of the 24 patients in this group was 66 years: there were 14 males and ten females. Eleven patients had coronary artery disease while four had chronic valvular disorders. Twenty-one patients were receiving digoxin therapy, one of whom had digitalis toxicity. Two other patients had slightly elevated serum digoxin levels but were not thought to be toxic clinically. The mean atrial rate during multilevel block was 284 ± 35 beats/min. Three patients had QRS durations of 0.12 sec, two with left bundle branch block, and one with an intraventricular conduction defect.

The arrhythmias in this group usually display classic Wenckebach periodicity of the ventricular beats. However, progressive prolongation of the P-R intervals relate to alternate P waves or flutter waves, and the Wenckebach periods terminate with three dropped atrial beats. Figure 1 depicts a representative example of Type A block. This electrocardiogram (patient 11) displays atrial flutter at an atrial rate of 276 beats/min with an irregular ventricular response of approximately 105 beats/min. Analysis of the ventricular rhythm reveals grouped beating with clusters of three or four beats, followed by pauses. The intervals from the onset of such a cluster to the beginning of the next cluster are mul-
minutes following the intravenous administration of digoxin, 0.25 mg, the ventricular rate slowed. At this time (panel B) the ECG revealed atrial flutter at 292 beats/min with coupled ventricular responses, progressive prolongation of alternate flutter to R intervals, and three consecutive dropped flutter waves. The ladder diagram depicts two level A-V block with 2:1 block above and 3:2 Wenckebach below. Panel C, obtained 15 min later, shows 4:1 A-V block at the same atrial rate. This is interpreted as progression of block at the lower level from 3:2 to 2:1 with a resultant combined 4:1 block.

Figure 4 shows the effect of carotid sinus massage on the rhythm of a 63-year-old woman with rheumatic heart disease (patient 2). The baseline rhythm is atrial flutter at 280 beats/min with apparent alternation of flutter-to-R intervals and dropped beats. It is not immediately obvious which flutter waves are conducted and which are blocked. During carotid sinus massage, the patient develops 4:1 A-V block at the same atrial rate. The ladder diagram demonstrates two level A-V block with 2:1 above and 3:2 Wenckebach below. The flutter-to-R interval preceding the QRS complex with a 3:1 A-V ratio is longer than the one with a 2:1 ratio. This suggests that the beats with shorter flutter to R intervals are not conducted. Carotid massage converts the 3:2 Wenckebach to 2:1, resulting in a combined 4:1 block.

Type B: Nonintegral Conduction Ratio at Upper Level

The mean age of the 12 patients in this group was 76 years; there were eight males and four females. Seven patients had isolated coronary artery disease. Four patients were receiving digitalis without overt evidence of toxicity, although two had elevated serum digoxin levels. In no instance was the occurrence of arrhythmia thought to be related to the administration of digitalis. The mean atrial rate during the 15 periods of arrhythmia was 162 ± 62 beats/min. Two patients had QRS durations greater than 0.12 sec: one with left bundle branch block and one with right bundle branch block, left axis deviation, and first degree block.

The arrhythmias in this group display a diversity of patterns dependent on the length of the Wenckebach cycle at the upper level, the type of block below it, and the relationship of the sequences of block at the two levels. Figure 5 shows an electrocardiogram obtained from an 86-year-old woman with an acute myocardial infarction (patient 26). The atrial rate is 102 beats/min with frequent nonconducted P waves and varying P-R intervals. One can
discern progressive prolongation of alternate P-R intervals; however, the cycles are terminated by a short, rather than long R-R interval, and there are no additional dropped P waves. This is consistent with Wenckebach periodicity at an upper level with 2:1 block at a lower level. In this instance, where there are an even number of atrial beats in each Wenckebach period (e.g., 4:3, 6:5), the beat blocked at the upper level is one that would have been dropped at the lower level had it been conducted that far. The next beat is the most rapidly conducted at the upper level and yet is able to traverse the lower level which has had ample time to recover, thus producing the shortest P-R and R-R intervals of the entire cycle. The shortest R-R intervals occur at the conclusion of the longest Wenckebach periods since the degree of R-R interval shortening depends on the difference between the shortest and the longest P-R intervals. This difference will tend to be greater in the longer Wenckebach cycles.

An 83-year-old woman with coronary artery disease (patient 35) displayed a similar conduction disturbance, consistent with 6:5 and 4:3 Wenckebach above with 2:1 block below. She also displayed a rhythm in which there are two P waves between each pair of QRS complexes, but with alternation in the P-R and R-R intervals (fig. 6). The ladder diagram depicts recurrent 4:3 Wenckebach above with 2:1 block below. The overall pattern is 2:1 block with alternating P-R intervals. As in the previous example, since there are an even number of atrial beats in each Wenckebach period, the R-R interval surrounding the P wave dropped at the upper level is the shortest of the entire cycle, and there are no additional dropped beats. This patient also had

**Figure 3.** Effect of digoxin on A-V conduction in atrial flutter. Panel A) Lead II, before therapy, atrial flutter with 2:1 block. Panel B) Monitor lead, 30 min after 0.25 mg digoxin intravenously. Pattern of Wenckebach of alternate flutter waves with Type A two-level block. Panel C) Monitor lead, 45 min after digoxin. There is 4:1 A-V block consisting of 2:1 block at both upper and lower levels.

**Figure 4.** Effect of carotid sinus massage (CSM) on Type A two-level block. Three-to-two Wenckebach at lower level is converted to 2:1 block by CSM, producing overall 4:1 block.
periods of junctional escape rhythm in which the R-R interval was longer than those seen in the rhythm noted above.

An 89-year-old woman (patient 27) with rhythms interpreted as two level block was noted to have a regular ventricular response with apparent 3:1 atrioventricular block (fig. 7). Since this was immediately preceded and followed by rhythms displaying Wenckebach above and 2:1 block below, it is likely that the rhythm depicted here is also attributable to two-level block. The ladder diagram demonstrates 3:2 Wenckebach above with 2:1 block below, resulting in a constant 3:1 A-V block. Two consecutive P waves are blocked in this instance when there is an odd number of atrial beats in each Wenckebach period.

Figure 8 was obtained from the same patient as in figure 5 above. In this strip there is a perfectly regular atrial rhythm at a rate of 120 beats/min with periods of 1:1 A-V conduction, progressive prolongation of P-R intervals, and occasional dropped beats. However, this is not simple Wenckebach periodicity since one can also identify two consecutive blocked P waves and, in addition, the P-R intervals following blocked beats are not necessarily the shortest in each cycle. The ladder diagram depicts Wenckebach block at two separate levels with 4:3 block below and either 5:4 or 6:5 block above. In the first sequence, the fourth impulse is blocked below, whereas the fifth is blocked above, resulting in two consecutive blocked P waves, culminated by a short P-R interval. The fourth P wave in the next sequence is also blocked below, however the fifth P is conducted above in a delayed fashion as part of a Wenckebach pattern. Thus, this P-R interval is long, even though it follows a dropped beat, and only a single dropped beat is seen. The sixth P wave of the sequence is then blocked above, allowing recovery of the entire conduction system and resulting in a subsequent short P-R interval at the beginning of the next cycle.

Figure 9 is a rhythm strip from a patient (31) whose rhythm displays a varying conduction pattern with prolongation of alternate P-R intervals, but with four consecutive blocked P waves. The ladder diagram reveals that the initial eight P waves follow a pattern of Wenckebach block at a first level with 2:1 block at a second, lower level. The ninth P wave is blocked at the first level immediately following a beat that is conducted to the ventricle. The tenth P wave is then conducted with the shortest A-V conduction time, but is blocked at a still lower level, which has not yet recovered from the previous depolarization. The eleventh beat is blocked in a 2:1 fashion at the second level, while the twelfth beat is blocked at the first level. This sequence of four consecutive dropped P waves repeats whenever a P wave is blocked at the highest level immediately following a beat which is conducted to the ventricle.

**Discussion**

It has been generally accepted that atrial impulses can partially penetrate the atrioventricular junctional tissues and thereby produce complex patterns of conduction. Langendorf and associates used the term "concealed conduction" to refer to this phenomenon and displayed clinical examples of Wenckebach periods of alternate beats, 2:1 block with alternation of the P-R intervals of the conducted beats, and 3:1 A-V block. However, spontaneous arrhythmias of this
type have been thought to be very uncommon clinically, and in some instances said to augur poorly for the patient's prognosis.7

During a two year period in a general community hospital, 36 patients were noted to have conduction defects which could be best explained by implicating multiple levels of A-V block. These cases were identified prospectively by examining routine standard electrocardiograms and intensive care monitoring records. His bundle recordings were obtained in only one patient since the arrhythmias were often transient and the clinical circumstances did not usually require this information for patient management.

Multilevel block was noted in a variety of clinical circumstances. Such patterns were chronic in some patients but were usually transitory, especially in patients with acute myocardial infarction or those being treated for rapid atrial flutter. No correlation with digitalis toxicity or electrolyte imbalance was noted. There were no instances of progression to complete heart block during the hospital course of these patients. Patients in group A had a significantly higher mean atrial rate than those in group B (284 ± 35 vs. 162 ± 62, P < 0.001). Two-to-one block at the highest level was generally observed at the more rapid atrial rates and was seen only once at an atrial rate lower than 230 beats/min.

The mechanisms of A-V block postulated in this report are theoretical and are probably oversimplified. Nevertheless, by supposing discrete levels of blockade, one can facilitate analysis of otherwise obscure conduction disturbances. Arrhythmias that defy such simple analyses may represent more complex and variable degrees of partial penetration of the conduction system.

The precise levels at which blocks occur cannot be identified in the majority of our patients. In the first example shown, the block is proximal to the His bundle as determined by electrophysiological study. The fact that the QRS duration is less than 0.12 sec in 31 of 36 cases suggests that the block is supraventricular in most instances.10 This is consistent with the studies of Amat-y-Leon and associates9 who found alternating Wenckebach periods to occur within the A-V node in 12 of their 13 cases. Schuilenburg and Durrer11 reported a case of alternating Wenckebach periods within the His bundle and Halpern and associates12 demonstrated block in the Purkinje system in their cases of alternating Wenckebach. Castillo and associates13 demonstrated antegrade and retrograde two-level block during atrial and ventricular pacing in which there was a Wenckebach phenomenon in the A-V node and Mobitz type II block below the His bundle. Since Wenckebach block is most common in the A-V node, especially in the presence of a normal QRS duration,10 patients with Type A block who have Wenckebach periodicity at their lowest level most likely have defects limited to the supraventricular portion of the conduction system. Patients with Type B block will tend to have Wenckebach block in the A-V node but can have further block in the Purkinje system, His bundle, or within the A-V node itself. His bundle recordings are necessary to clarify the precise location of this form of block.

Type A multilevel block is readily recognized by the typical Wenckebach periodicity of the ventricular response. However, in this instance, there are three dropped atrial beats instead of the single dropped beat in one level block. Stable 4:1 A-V block in atrial flutter probably represents two levels of 2:1 block. The transition from 2:1 block, to
two-level block with Wenckebach, finally resulting in 4:1 block, was documented in figure 3. This formulation would be consistent with Sir Thomas Lewis' well-confirmed observation that "whereas 2:1 and 4:1 degrees of block are often stabilized in flutter, persistent 3:1 block is exceptional." Type B block can have a multiplicity of ventricular response patterns and may display one, two, or more dropped P waves in succession. When the dropped beat of a Wenckebach cycle at the upper level follows a beat that was conducted in a 2:1 fashion at a lower level, the dropped beats at the two levels coincide and there is only one nonconducted P wave. Furthermore, since both levels are able to recover fully during this period, the subsequent P-R interval is the shortest of the entire cycle, resulting in a paradoxical narrowing of the R-R interval surrounding the dropped P wave (fig. 5). This is seen when there are an even number of atrial beats in the Wenckebach cycle. Two consecutive nonconducted P waves will occur when the dropped beat at the upper level follows a nonconducted beat at a lower level (fig. 7). This happens when the number of atrial beats in the Wenckebach cycle is odd. A recurring rhythm pattern displaying more than three consecutive dropped P waves can occur in the presence of three-level block (fig. 9).

Since Wenckebach cycles can be demonstrated at either an upper or lower level in the conduction system, it is reasonable to assume that Wenckebach block can occur simultaneously at both levels. In this circumstance, if the upper level displays a greater degree of block than the lower, the rhythm would be that of simple one-level Wenckebach as the lower level conduction disturbance would not be manifested on the standard ECG. His bundle electrograms could identify such a mechanism if the lower level of block is within or below the His bundle. When the upper level displays a lesser degree of block than the lower, a variety of conduction patterns can ensue. The primary ventricular rhythm will be Wenckebach periodicity. When block occurs at the lower level, there will be a dropped atrial beat but the subsequent P-R interval will remain relatively prolonged since it reflects slow conduction at the upper level. In contrast, the P-R interval following a beat dropped at the upper level will be short, since the entire conduction system will have had a chance to recover. When there is a dropped beat at the lower level immediately followed by a similar occurrence at the upper level, the ECG will show two successive nonconducted P waves followed by a short P-R interval (fig. 8). To our knowledge, this formulation of conduction patterns has not been reported previously, although Langendorf's case of 4:2 block probably represents this phenomenon (ref. 5, fig. 2). Since there can be two levels of block above the His bundle, it is conceivable that a third level can exist within the His-Purkinje system.

In the absence of electrophysiologic studies, one cannot be certain of the exact conduction mechanisms in these arrhythmias. Similar disturbances have been explained by some investigators utilizing the concepts of dual conduction pathways and complex block patterns at a single level. Rhythms displaying 2:1 A-V block with alternating P-R intervals could represent high degree block with periodic nodal escape beats, and 3:1 block could reflect a very prolonged refractory period at a single level. Consecutive dropped P waves could reflect the occurrence of concealed re-entry. However, several of the rhythms displayed here are identical to those reported in perfused rabbit hearts by Watanabe and Dreifus, in which electrophysiologic studies revealed alternating deeper penetration of blocked atrial impulses. In Halpern and associates' report of alternating Wenckebach they presented rhythms in which the atrial rates were below 130 beats/minute and in which there were two consecutive dropped P waves. They postulated the presence of a markedly prolonged absolute refractory period during which two subsequent beats could be blocked. However, in their patients with intraventricular conduction defects, the rhythm more likely represents Wenckebach block at an upper level with 2:1 block below it, as in figure 5. Similarly, Hartzler and Maloney's case of alternate beat Wenckebach with two dropped P waves probably represents Wenckebach above and 2:1 below, rather than 2:1 above and Wenckebach below, as they had postulated.

In conclusion, a variety of rhythm disorders have been presented which can be explained utilizing the concept of
multilevel atrioventricular block. These interpretations can be made from the clinical electrocardiogram and do not require complex electrophysiologic studies. This phenomenon is not rare and occurs under a variety of clinical circumstances. It is likely that multilevel block is far more common than previously suspected, since stable 4:1 block in atrial flutter probably represents two levels of 2:1 block. The more complex patterns are often transient, or may simply not be recognized. The patterns presented in this series are not exhaustive of the permutations of multilevel block that can occur. Recognition that block can occur at more than one level in the A-V conduction system may facilitate the interpretation of hitherto obscure rhythm disturbances and thereby aid in the management of the patient with arrhythmia.

References
1. Lewis T: Auricular flutter. Heart 4: 171, 1912

Echocardiographic Evaluation of the Valsalva Maneuver in Healthy Subjects and Patients with and without Heart Failure

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SUMMARY The Valsalva maneuver was evaluated by echocardiography in three groups: A) 10 normal volunteers, B) 10 patients with no history of heart failure and normal ejection fractions, and C) 10 patients with heart failure and depressed ejection fractions.

Groups A and B had a significant fall in left ventricular internal dimensions and calculated stroke volume by end strain which returned rapidly to baseline in recovery without significant overshoot. Arterial pressure showed a sigmoidal strain pattern with a normal overshoot in early recovery in all group B patients. In group C ventricular dimensions did not diminish during strain; arterial pressures showed a "square wave" pressure elevation during strain without an overshoot in recovery. Echocardiography allows a new approach to evaluate further the left ventricular response to the Valsalva maneuver. Patients with severely depressed ejection fractions, unlike those with normal ventricular function, are unable to alter stroke output in response to acutely increased intrathoracic pressure. A square wave pressure response is a likely consequence of a fixed stroke output during the strain maneuver.

THE VALSALVA MANEUVER is commonly employed clinically and far more often practiced unwittingly in the course of biological and occupational activities. The blood pressure response to the Valsalva maneuver has been extensively studied.1,2 Individuals without heart failure have a sigmoidal strain blood pressure response, i.e., a rise above resting pressures during early strain followed by a progressive fall to below control pressure in late strain. At release blood pressure overshoots baseline measurements. In patients with heart failure there is a square wave response, i.e., blood pressure rises above control during the entire strain phase and falls to resting blood pressure levels at release.

There has been little opportunity to examine the effects of acutely raising intrathoracic pressure on left ventricular chamber dimensions and beat-to-beat stroke volume. With the advent of echocardiography it has become possible to assess rapidly and continuously the left ventricular response to this maneuver. Accordingly this study was designed to further evaluate with echocardiography the effect of the Valsalva maneuver on the left ventricular dimensions of normal subjects as compared to those of patients with and without heart failure. A report of our initial findings has already been made.4

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