Mechanism Underlying Kussmaul's Sign in Chronic Constrictive Pericarditis

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In 1873 Kussmaul[1] reported the observation that, in 2 patients with constrictive pericarditis, the expected inspiratory collapse of neck veins was replaced by increased inspiratory distension. The association of Kussmaul's sign, as it subsequently became called, with constrictive pericarditis is now widely accepted. At the same time it is also recognized that it is not specific to this pathology and can be observed in congestive heart failure,[2-4] restrictive cardiomyopathy,[5] right ventricular infarction[6] and acute cor pulmonale.[7] As stressed by Spodick,[8] it is not a feature of pericardial tamponade and is of value in distinguishing tamponade from pericardial constriction. In spite of its clinical value and wide use, the underlying mechanism is poorly understood.[9] We report our observations based on 6 patients with chronic constrictive pericarditis in an attempt to clarify the mechanism of this sign.

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Six patients, in whom the diagnosis of severe constrictive pericarditis was based on clinical, radiologic

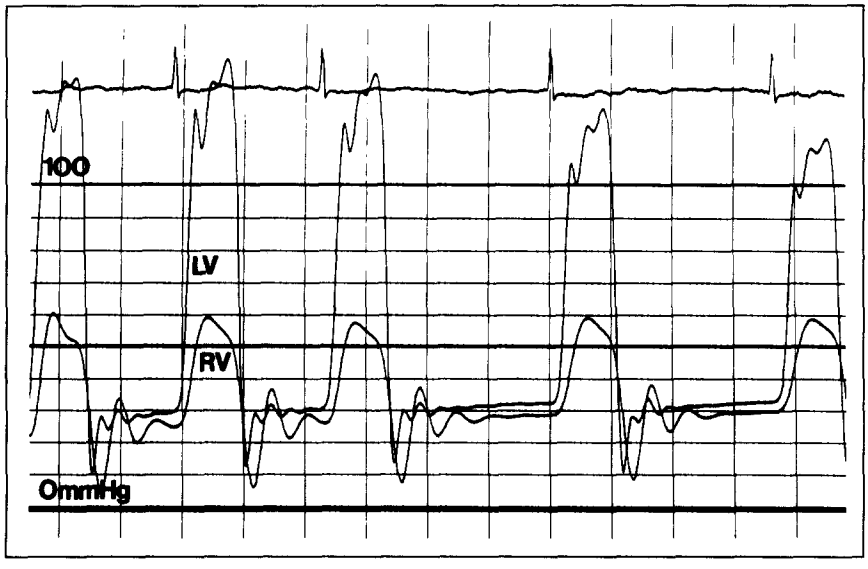

FIGURE 1. Simultaneous pressure recording of the right ventricle (RV) and left ventricle (LV) in patient 6. Note the early diastolic dip and subsequent "plateau," with virtual equilibration of pressures throughout the rest of diastole. (Paper speed 50 mm/s in this and subsequent recordings.)

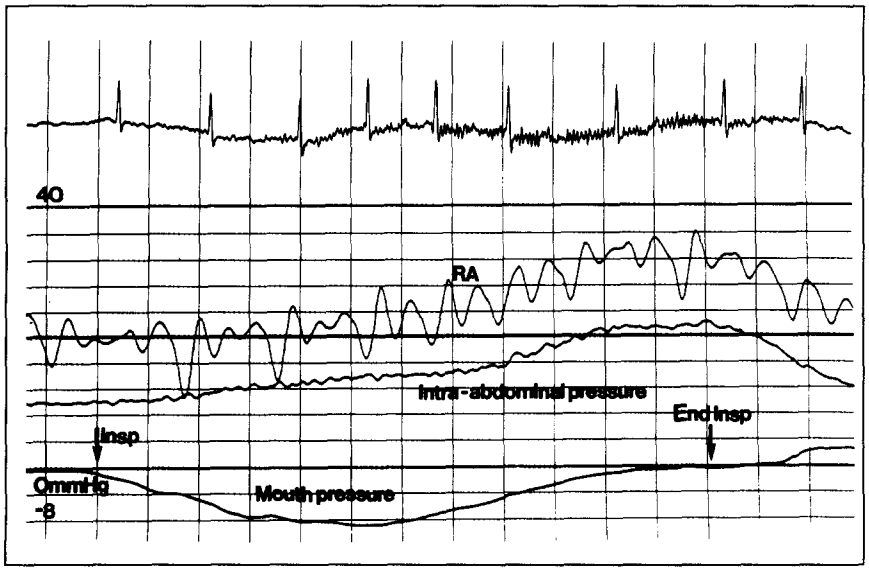

FIGURE 2. Simultaneous recording of right atrial (RA), intraabdominal and mouth pressures in patient 4. There is a progressive increase in RA pressure from the onset of inspiration (Insp) to peak at end-inspiration (End Insp). This was paralleled by a concomitant increase in intraabdominal pressure. Note the slight increase in the right atrial *y* descent during the early part of inspiration.

TABLE I Clinical, Hemodynamic and Pathologic Findings in Six Patients with Constrictive Pericarditis

Pt	Age (yrs), Sex	Ascites	RAP (mm Hg)	CI (liters/min/m²)	Histology
1	33, M	+	20	1.7	Tuberculous pericarditis
2	35, M	++	31	1.3	Tuberculous pericarditis
3	58, M	+	23	1.5	Tuberculous pericarditis
4	31, M	+	18	1.9	Nonspecific pericarditis
5	20, M	+	18	1.7	—
6	60, F	+	24	1.4	—

CI = cardiac index; RAP = mean right atrial pressure; + = present; ++ = marked.

and echocardiographic features (Table I), were studied during combined right- and left-sided cardiac catheterization. Pressures, electrocardiogram and respiration were recorded simultaneously, the latter by recording the pressure developed at the mouth through a rubber mouthpiece. A Hewlett-Packard 1290 A unit was used for pressure recording. Intraabdominal pressures were recorded via a 7Fr pigtail catheter introduced through the lateral abdominal wall. The zero reference point for the cardiac and intraperitoneal pressures was the midchest.

Pressures were recorded during normal and deep inspiration, and during gentle manual compression of the abdomen in held midexpiration. In patient 2, the aforementioned recordings were made before and after drainage of 4.25 liters of ascitic fluid, and in patient 4 before and after the application of an abdominal corset.

The cardiac output was low and the mean right atrial pressure was considerably elevated in all 6 patients (Table I). All patients showed the typical diastolic equilibration of pressures associated with constrictive pericarditis (Figure 1).

During ordinary quiet breathing, the inferior vena cava, right atrial and right and left ventricular end-diastolic pressures, as well as the intraabdominal pressure, showed little or no variation. A slight decrease in both pulmonary artery and aortic pressures was noted during inspiration.

During slow, deep inspiration, the right atrial pressure increased progressively from the onset of inspiration to a maximal level at end-inspiration. This was paralleled by a comparable increase in intraabdominal pressure (Figure 2). A slight increase in the depth of the right atrial y descent was observed in most patients during the early part of deep inspiration (Figure 2). Right

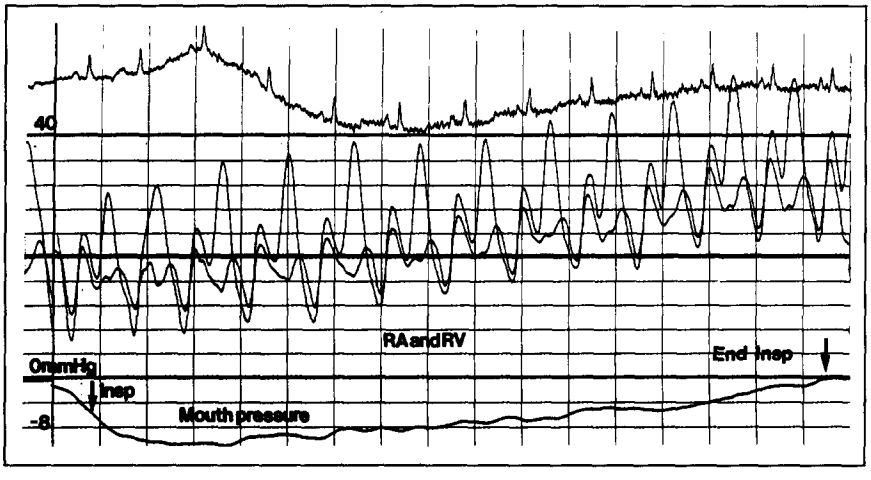

FIGURE 3. Simultaneous recording of the right ventricle (RV), right atrium (RA) and mouth pressures in patient 3. There is a progressive increase in RA and RV pressures from the onset of inspiration (Insp) to peak at end-inspiration (End Insp).

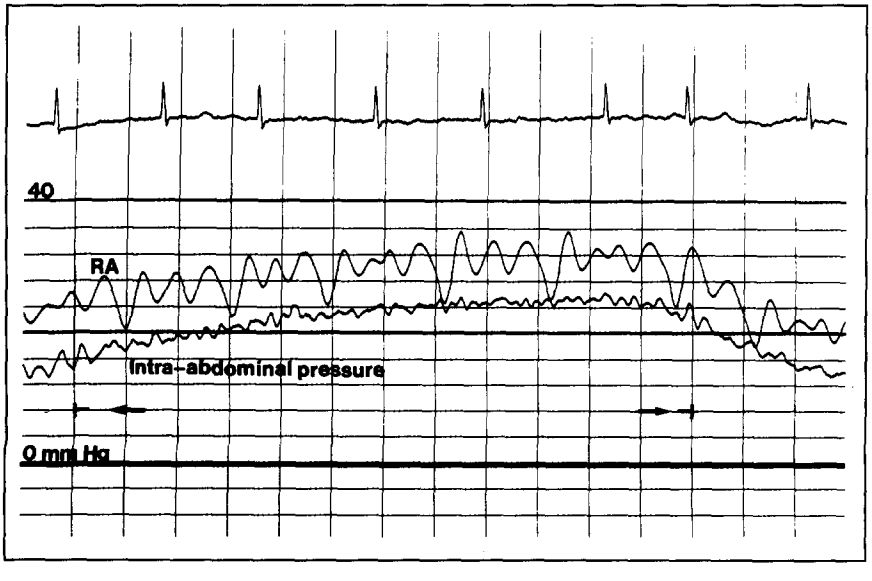

FIGURE 4. Simultaneous recording of right atrium (RA) and intra-abdominal pressures in patient 2 during gentle abdominal compression (*arrows* indicate period of compression). Note the parallel increase in RA and intraabdominal pressures during abdominal compression.

ventricular systolic and diastolic pressures (Figure 3) also progressively increased to peak at end-inspiration.

Gentle abdominal compression resulted in a simultaneous and parallel increase in intraabdominal, inferior vena cava, right atrial (Figure 4), and right and left ventricular systolic and diastolic pressures.

Drainage of 4.25 liters of ascitic fluid in patient 2 resulted in a decrease of both end-expiratory and end-inspiratory right atrial and intraabdominal pressures (Figure 5).

The application of an abdominal corset in patient 1 resulted in an increase of both the end-expiratory and end-inspiratory right atrial pressures.

In constrictive pericarditis the heart is encased in a thick pericardium that is virtually inelastic. When not fully filled, in systole and early diastole, changes in pleural pressure are transmitted to its contents. However, once filled in early diastole, such a heart cannot be filled further. Thus, throughout the greater part of diastole, the heart is incapable of further distension in response to the increased negative intrapleural pressure of inspiration.[10] While this adequately explains the absence of a normal inspiratory decrease in venous pressure in constrictive pericarditis, it does not account for the increase of venous pressure during inspiration observed by Kussmaul.[1]

In 1942, Hitzig[3] examined the effect of abdominal compression on the venous pressure in the neck in patients with constrictive pericarditis, right-sided cardiac failure, cardiac tamponade and superior vena caval obstruction. He concluded that the increase in venous pressure resulting from abdominal compression was due to an "augmented venous return" to an "insufficient or compressed right heart" that was "overloaded beyond its functional capacity." [3]

Our observations are fully consistent with the hypothesis that the inspiratory increase in venous pressure in constrictive pericarditis appears to be the direct consequence of an inspiratory increase in abdominal pressure transmitted through an already distended venous system to a heart that is incapable of greater diastolic distension, the "inspiratory hepatojugular reflux phenomenon" suggested by Hitzig.[3] Thus, the lack of variation in venous pressure during quiet breathing in our patients was associated with little or no change in abdominal pressure, whereas during maneuvers that increased abdominal pressure such as deep inspiration (Figure 2) or manual abdominal compression (Figure 4), the increase in venous pressure was paralleled by the increase in intraabdominal pressure. The dependence of the venous pressure on changes in intraabdominal pressure is confirmed by the higher right atrial pressures generated by an abdominal corset and the parallel decrease in both right atrial and abdominal pressures (Figure 5) after drainage of tense ascites.

Other pathogenetic mechanisms may be operative during inspiration in constrictive pericarditis. It has been proposed[11] that, with pleuropericardial-diaphragmatic adhesions, descent of the diaphragm in inspiration may

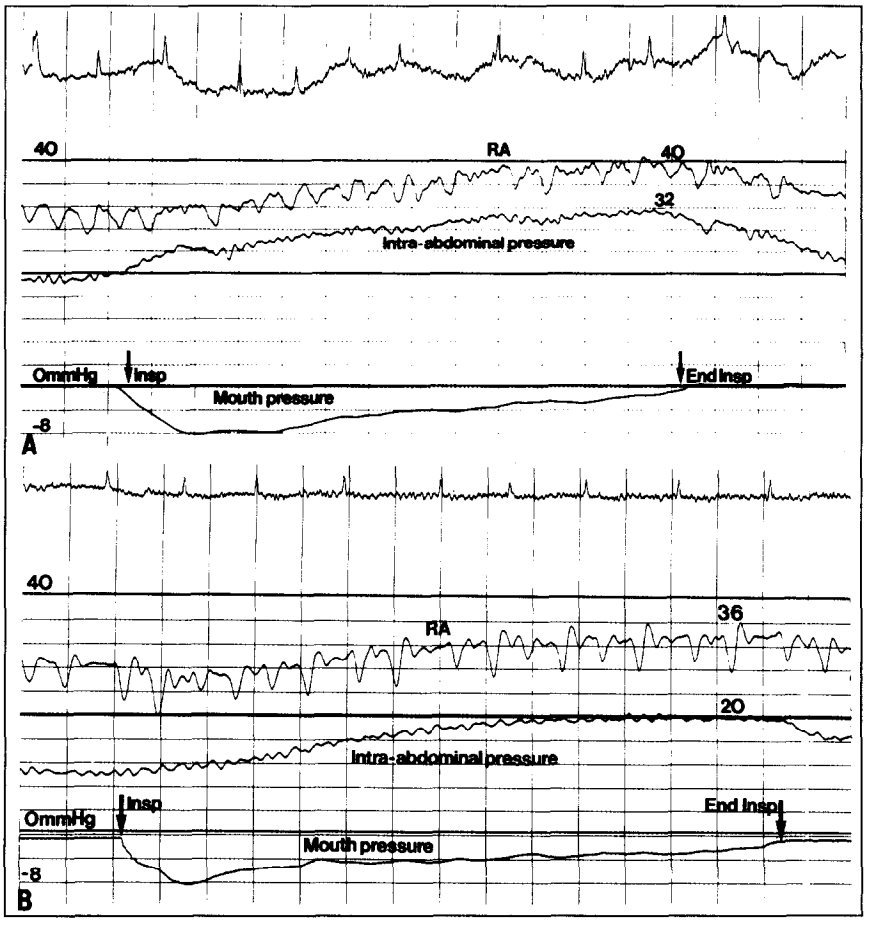

FIGURE 5. Simultaneous recording of right atrium (RA), intra-abdominal and mouth pressures during deep inspiration in patient 2 before (*A*) and after (*B*) drainage of 4.25 liters of ascitic fluid. Note the reduction in both end-expiratory and end-inspiratory RA and intra-abdominal pressures in *B*.

stretch the heart in a caudocranial dimension and thus effectively reduce its diastolic compliance. If this mechanism were operative, diastolic compliance would be further reduced by maneuvers that cause the downward displacement of the diaphragm and lessened by maneuvers with the opposite action. The fact that drainage of ascitic fluid reduced the inspiratory right atrial pressure, whereas application of an abdominal corset increased it, suggests that this mechanism did not significantly contribute to the inspiratory increase of venous pressure.

Another possibility is that the inspiratory increase in right atrial pressures seen in these patients is due in part to an increased left ventricular afterload, a possibility that cannot be excluded on the basis of these data. However, increased left ventricular afterload has not, to our knowledge, been documented in constriction. Furthermore, the effect of an increased transmural pressure during inspiration on left ventricular wall stress or afterload may theoretically be attenuated by an effective increase in total wall thickness resulting from the myopericardial inflammatory process.

In conclusion, the inspiratory increase in venous pressure seen in chronic constrictive pericarditis would seem to depend on 2 factors, that is, an inelastic pericardium or heart in which diastolic stiffness is greatly increased and a distended venous system that is capable of transmitting the increased abdominal pressure of inspiration to the intrathoracic and superior vena cava systems. It is probable that the same 2 factors operate in conditions such as acute cor pulmonale,[7] right ventricular infarction[6] or congestive cardiac failure,[3,4] in which Kussmaul's sign may be present. In all these situations there is acute distension of the right ventricle and pericardium, with resultant increase in "stiffness" of the heart, and thus in restrictive hemodynamics.

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Left Ventricular Structure and Function in Professional Basketball Players

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The noninvasive nature of echocardiographic/Doppler methods has allowed detailed assessment of cardiac anatomy and physiology in normal subjects and athletes, as well as accurate determination of left ventricular (LV) wall thickness and mass.[1,2] Recently, interest has developed in the use of Doppler recordings of mitral inflow to assess LV filling patterns in normal subjects, in conditioned athletes, and in various disease states associated with LV hypertrophy.[3–10] Several studies[6–9] have suggested that an increase in LV mass may be seen in certain highly trained athletes without any abnormalities in Doppler-derived LV filling parameters. This finding has engendered the term "physiologic" hypertrophy. However, little data are available on hearts of large body size athletes in whom LV mass is increased. In the present study highly trained aerobic athletes (professional basketball players) and nontrained normal subjects with large body size were evaluated by echocardiographic/Doppler techniques to determine whether increased LV mass and cavity size per se affect diastolic function when systolic function is normal.

The study group consisted of 12 professional male basketball players with a mean age 26 ± 3 years, who at the time of the study were at the end of their preseason training program and were participating in national competition. Eleven healthy, nonsedentary male subjects formed an age-matched control group (mean age 30 ± 5 years), who were not involved in a routine training program and were recruited on the basis of being above normal for height, weight and body surface area. Both groups were normotensive, free of known cardiovascular disease and in normal sinus rhythm during the study.

All subjects had technically adequate M-mode and 2-dimensional echocardiograms performed by the same technician on an Interspec XL unit using a 3.5-MHz transducer and standard acquisition techniques. After completion of the study, echocardiographic analysis was performed by the same observer. M-mode measurements were made according to the leading edge to leading edge recommendations of the American Society of Echocar-

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