Pathophysiology of Cardiac Tamponade*

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Cardiac tamponade is always life threatening and nearly always requires urgent and precise therapeutic intervention. It is perhaps unique in that appreciation of its pathophysiologic state is essential to precise diagnosis and rational treatment. Since the 19th century, investigations in experimental animals provided a basic understanding that has been continually refined by recent investigators to the point where an integrated picture of its macrophysiologic condition emerges. This review covers the resultant concepts. Procedural details of investigations can be found in the references (thus, for example, the text omits the relative merits of catheters vs flat balloons for intrapericardial pressure determination).

GENERAL CONSIDERATIONS

Cardiac tamponade is defined as significant compression of the heart by accumulating pericardial contents, including effusion fluids, blood, clots, pus, and gas, singly or in combinations. “Significant compression” depends on whether tamponade is approached from a purely physiologic or clinical standpoint. Since tamponade is a pathophysiologic continuum, hearts can be said to be mildly to floridly tamponaded, the latter being a life-threatening emergency and the former a stage that can progress in that direction. “Life threatening” is a result of progressive, ultimately critical, reduction in cardiac output that can occur from as little as 150 mL of blood flooding the pericardium after a cardiac wound to much more than 1 L of fluid in slowly accumulating pericardial effusions. The “continuum” concept, formulated by Reddy et al,1 involves stages seen in intact, unanesthetized animals, fully recovered from operations to implant instrumentation (with responses quantitatively different from open chest, anesthetized animals undergoing short-term experiments2,3) and also in human tamponade as measured by cardiac catheterization.1 However, the continuum begins before catheterization is clinically justified when small, medium, and large asymptomatic, “nontamponading” effusions couple the parietal pericardium to the heart, thereby exaggerating ventricular interaction and the consequent respiratory responses, as measured by sensitive noninvasive techniques in patients without pulsus paradoxus, the classic respiratory response of critical cardiac tamponade.4

INTERACTION BETWEEN THE PERICARDIUM AND ITS CONTENTS

Clinically significant cardiac compression by pericardial fluids depends on three interrelated conditions. The pericardial contents must do the following: (1) fill the relatively small pericardial reserve volume (Fig 1)—the volume which, added to the normal 15 to 35 mL of pericardial fluid, will just distend the parietal pericardium by filling its numerous recesses and sinuses;5 (2) thereafter increase at a rate exceeding the stretch rate of the parietal pericardium;6 and (3) exceed the rate at which venous blood volume expands to support the small normal pressure gradient for right heart filling.1,6 Because at any instant the pericardium is relatively inextensible, the heart and the pericardial contents compete continuously for the relatively fixed intrapericardial volume. Therefore, unless the abnormal pericardial contents increase very slowly, permitting progressive “give” of the parietal pericardium, their increase is at the expense of cardiac chamber volume. The key operational defect is reduced cardiac inflow due to compression of all cardiac chambers, progressively reducing their diastolic compliance and ultimately equalizing the mean diastolic pressures in each of them.

Pericardial stiffness determines the fluid increments causing tamponade. Despite elastic tissue and initially wavy collagen, the initial elements of pericardial relaxation,7 even the normal parietal pericardium is relatively noncompliant8 so that after the initial give, the pericardial pressure-volume curve angulates and becomes vertical5 (Fig 1). Thus, patients with critical tamponade function on the steep vertical portion of the pericardial pressure-volume curve with progressively smaller fluid increments.

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provoking progressively large pressure increments. If unchecked by compensatory mechanisms (Fig 2) or effective treatment, the heart becomes critically compressed, because cardiac filling, ie, maximum diastolic volume, competes unsuccessfully with pathologic pericardial filling for the relatively fixed space within the parietal pericardium. This results in progressive reduction in cardiac chamber volumes.

**CARDIAC RESPONSES TO SIGNIFICANT PERICARDIAL COMPRESSION**

Figure 2 schematizes the major hemodynamic events and compensatory mechanisms in uncomplicated cardiac tamponade.

When increasing pericardial contents put the intrapericardial pressure on the steep portion of its J-shaped pressure-volume curve (Fig 1), the cardiac chambers must operate on parallel steep pressure-volume curves, a form of diastolic dysfunction where, at any diastolic volume, there is necessarily excessive intracardiac pressure. Thus, progressive compression of the atria and ventricles progressively resists cardiac filling, progressively reducing ventricular stroke volume. In intact, unanesthetized, long-term instrumented animals compensatory mechanisms maintain arterial pressure until relatively sudden decompensation, as in comparably intact human patients. This is a “last straw”—literally “last drop”—phenomenon due to the final increment of pericardial contents. This situation is demonstrated by the reciprocal effect of therapeutic pericardial fluid drainage—the initial decrement usually produces the largest relative hemodynamic improvement by rapidly shifting the stretched pericardium back toward the “flat” portion of its pressure-volume curve with parallel improvement in intracardiac pressure-volume relations and arterial pressures.

Although the key chambers maintaining cardiac output are the right and left ventricles, within the pathophysiologic continuum of tamponade, the earliest targets of significant compression are the thinner chambers: the right atrium and ventricle, diastolic pressure in which equilibrates with rising pericardial pressure before the left atrial and ventricular pressures do. That right atrial and ventricular pressures are normally somewhat lower than in the corresponding left chambers contributes to their earlier entrainment by inexorably rising pericardial pressure.

**Significance of Transmural Pressures and Respiratory Reciprocation**

For each cardiac chamber, its transmural pressure—intracardiac pressure minus pericardial pressure—is a principal determinant of its filling. (Transmural pressure is a true filling [distending] pressure that contributes to ventricular preload.) Normal pericardial pressure is lower than the right atrial mean and right ventricular diastolic pressures so that right atrial transmural pressure (right atrial pressure minus pericardial pressure) is normally higher than its cavitary pressure. In tamponade, rising pericardial pressure progressively reduces—and ultimately can make phasically negative—the average transmural pressure of first the right and subsequently the left cardiac chambers. Survival necessitates the ensuing parallel rise in diastolic pressures, first in the right side of the heart and later the left side of the heart, critically reducing all chamber transmural pressures, and with them all filling. Like most tamponade-induced abnormalities of pressure and flow, transmural pressures are reciprocally reduced and increased during the respiratory phases for the left vs the right heart. Thus, inspiration increases right heart filling at the expense of the left heart with reversal in expiration. In critical tamponade, when cardiac output usually has fallen by at least 30%, transmural pressures are, on average, zero (typically between 15 and 30 mm Hg within the pericardium and between 15 and 30 mm Hg within the heart in euvolemic patients) so that respiratory reciprocation becomes a principal physi-
ologic mechanism contributing at some level to cardiac input and output. A significant component of respiratory reciprocation is the marked shift of the ventricular septum into the left ventricle when inspiration fills the right heart at the expense of the left with reversal on expiration. Clinically, respiratory reciprocation is expressed as pulsus paradoxus.

VASCULAR AND CARDIAC PRESSURE RESPONSES

Since the systemic and pulmonary venous beds must generate sufficient pressure to fill both sides of the heart, cardiac filling is supported by a parallel rise in systemic and pulmonary venous pressures which, in tamponade, are determined primarily by pericardial rather than myocardial compliance.1 Secondarily, of course, cardiac chamber compliance is reduced by pericardial compression and this produces the immediate force progressively resisting filling. In response, blood volume expands (Fig 2). Indeed, part of the ability to tolerate increasing cardiac compression is the rate of venous volume expansion. This, like pericardial stretch, requires time since it occurs by fluid transfer from the tissues to the venous side of the cardiovascular system. In contrast, in rapid, hemorrhagic tamponade, eg, due to cardiac wounds, venous volume expansion is virtually inoperative. Finally, venous filling may be augmented at very low ventricular volumes by diastolic suction, the relative effectiveness of which is undetermined.6

Eventually, diastolic pressure in both ventricles and the pulmonary artery equilibrate with mean right and left atrial pressures at approximately intrapericardial pressure. Conventionally, "equilibrate" represents diastolic pressures differing by no more than 5 mm Hg. Clinically, this corresponds to "florid" tamponade, when most patients will have frank exaggeration of the respiratory fluctuation in arterial BP—pulsus paradoxus—conventionally a 10 mm Hg or greater inspiratory fall that reflects the exaggerated respiratory pressure and volume reciprocation between the right and left heart chambers. For example, during peak inspiration, left heart filling is minimal and its pressure differences from pericardial pressure are least, nil or even negative, with reversed, ie, negative, transmural pressure.3,6 In such severe tamponade, the mitral valve may only open when atrial systole occurs during expiration.11 With inspiration, pulmonary wedge pressure falls below pericardial pressure. In contrast, right atrial pressure, which tends to "track" pericardial pressure12 (Fig 3) also falls, but not below pericardial pressure, enhancing inspiratory filling of the right ventricle.

Figure 2. Cardiac tamponade: relationships among major hemodynamic events and major compensatory mechanisms (see text). Simple arrows = tamponade sequences; pointed arrowheads = stimulatory compensatory actions; blunt arrowheads = oppositional compensatory actions. Modified from Spodick DH. In: Spodick DH, ed. The pericardium: a comprehensive textbook. New York: Dekker, 1997: 182 (with permission).
Figure 3. Cardiac tamponade. Simultaneous right atrial (RA) and pericardial (Pm) pressure curves; 

Insp = beginning of inspiration. Patient with atrial flutter. Top: prepericardicentesis: high atrial pressure (mean, 25 mm Hg) tracks high pericardial pressure; in early inspiration, pericardial pressure transiently falls below RA pressure. Note QRS electric alternation and atrial flutter. Bottom: postpericardicentesis: mean RA pressure has fallen to 15 mm Hg with little respiratory variation (pericardial restraint removed). Pericardial pressure has fallen below RA pressure with marked respiratory fluctuations: expiratory level, 8 mm Hg; inspiratory level, approximately zero. Note disappearance of electric alternation; flutter waves on atrial pressure tracing and ECG.

(Tracking is perhaps best demonstrated in experimental animals by intrapericardial pressure measurement with a flat balloon.)

Tamponading pericardial fluids compress the heart throughout systole and diastole. Although the atria fill continuously, blood mainly enters the heart when blood is leaving it during the right and left ventricular ejection periods, since ventricular ejection expels blood, reducing ventricular volumes. Ejection thus transiently reduces pericardial pressure, transiently increasing transmural pressure. Ejection simultaneously aids atrial filling through enlarging the atria by pulling their “floors” (valve levels) toward the ventricular apices; this produces the normal descent in atrial pressure curves. Pericardial volume and pressure thus vary continuously during the cardiac cycle reflecting the variations in cardiac chamber volumes. Ventricular pressure is high throughout diastole resisting filling. In early diastole, the normally rapid peak ventricular
filling rate becomes radically reduced, along with the filling fraction; this increases the relative contribution to ventricular filling of end-diastolic atrial contraction. At end-diastole, the compressed ventricles are thus maximally expanded by filling, raising intrapericardial pressure to its maximum. Although continuous atrial filling tends to expand the atria, which also tends to raise pericardial pressure, at end-systole, ventricular ejection (emptying) is complete so that the ventricles are at minimal volume permitting intrapericardial pressure to fall. In tamponade, when ventricular filling is "maximal," the ventricles remain critically underfilled relative to their normal capacity ("underpreloaded"). They therefore operate at the low end of their Frank-Starling curves while ejecting the reduced stroke volume.

**Intracardiac Pressure Curves**

In full-blown tamponade, ventricular pressure curves remain flat throughout diastole at their high equilibrated level. During systole and ventricular ejection, the atrial cavity pressure drop preserves the normal x descent in atrial pressure curves. However, continuously high ventricular diastolic pressures resist rapid ventricular filling so that atrial emptying in early diastole is resisted and ultimately aborted. Consequently the y descent of atrial pressure following arterioventricular valve opening is progressively amputated and ultimately eliminated. Moreover, high ventricular diastolic pressure favors premature mitral and tricuspid valve closure, further resisting filling with further reduction of ventricular preload reflected in the shorter fiber length of the compressed, underfilled chambers. This reduces the ventricular ejection rate which, with the progressive underfilling, progressively reduces stroke volume. In inspiration, because the right atrium and ventricle expand at the expense of the left (due to both septal shift and elevation of pericardial pressure; Fig 2), inspiratory left ventricular chamber compliance is minimal. Ultimately the mitral and aortic valves may open during expiration only with atrial systole.11

**Influence of State of Hydration**

The progressively increasing diastolic chamber pressures ultimately equilibrate (averaged over the respiratory cycle) throughout the heart. The typical range of 15 to 30 mm Hg applies mainly to euvolemic patients and animals. All physiologic phenomena in tamponade occur earlier and at lower pressures in hypovolemic patients and later and at higher pressures in hypervolemic than in euvolemic patients. Indeed, significantly hypovolemic patients can have tamponade at average diastolic pressures as low as 6 mm Hg, a form of low-pressure tamponade.2,4,14 (This can be difficult to recognize clinically15 particularly in patients treated with diuretics.)

**Coronary Blood Flow**

With normal coronary arteries, coronary blood flow is reduced but remains adequate to support aerobic metabolism.16 This is due to a proportionate reduction in cardiac work, because the ventricles are underloaded—"underafterloaded" as well as "underpreloaded."6,15 Even coronary vasodilatory reserve, capacitance, and resistance are not reduced sufficiently to add a measurable ischemic burden.16

**Myocardial Status**

In otherwise normal hearts, gross systolic function remains intact17 and the ventricles support stroke volume by an increased ejection fraction (often visibly striking during echocardiography). In contrast, previously diseased or injured hearts may not maintain cardiac output and arterial pressure as well,18 and tamponade decompensates earlier.

**Compensatory Responses: Decompensated Tamponade**

In addition to time-dependent blood volume expansion, pericardial stretch, and increased ejection fraction, compensatory mechanisms for tamponade include tachycardia and peripheral vasconstriction due to intense adrenergic stimulation evoked by the falling cardiac output.19 Rising right atrial pressure reflexly contributes to the tachycardia that enhances the minute cardiac output (stroke volume×heart rate) in the face of the falling stroke volume (Fig 2).

Adrenergic stimulation, both alpha and beta, and including increased serum catecholamines,19 is a principal compensatory response to the reduced cardiac output (Fig 2) with four major effects: (1) β-adrenergic contribution to heart rate increases; (2) β-adrenergic-dependent augmentation of diastolic relaxation; (3) α-adrenergically increased peripheral resistance to maintain central blood pressure and support the gradient for coronary flow; and (4) increased inotropy to minimize ventricular end-systolic volume via increased ejection fraction which, as already noted, is normal to high in tamponade in the absence of cardiac disease.3,6,12,15

Despite progressively falling cardiac output, increased peripheral resistance supports arterial BP until relatively late, partly by an α-adrenergic mechanism. Thus, increased peripheral resistance is not
affected by β-blockade, but is opposed by α-blockade.²⁰ Ultimately BP tends to decline precipitously—the “last drop” phenomenon. (In experimental animals that are anesthetized or not recovered from surgery, BP falls relatively slowly in contrast to intact animals that more closely resemble human responses.²³) The critical BP fall is also influenced by an opioid-dependent mechanism, demonstrated by naloxone-induced BP increase during tamponade.²⁰ This increase occurs without increasing cardiac output, so that this mechanism must act through increased systemic vascular resistance. (Further evidence that systolic function does not limit cardiac output in uncomplicated tamponade.⁹) Naloxone-induced BP increase further suggests a “peripheral resistance reserve,” despite the already high peripheral resistance that is a principal compensatory response (Fig 2).

FURTHER NEUROHORMONAL ACTIVATION

BP is augmented by further neurohormonal activation associated with arterial and atrial baroreceptor unloading,²¹ resembling neurohormonal activation in cardiac failure (but much less investigated). The renin-angiotensin-aldosterone system contributes renin, angiotensin II, arginine vasopressin, and aldosterone, with consequent sympathetic nervous system stimulation, vasoconstriction, decreased urine flow, decreased renal sodium and potassium excretion, and water retention. These effects are mainly relatively late, during decompensating tamponade, after aortic BP decreases by about 30%.²² and are followed by increased production of adrenocorticotrophic hormone. They are preceded by neurogenic reduction in renal sodium output.²³ However, unlike cardiac failure at comparably high central pressures, serum atrial natriuretic factor does not increase, because tamponade prevents myocardial stretch.²³-²⁵ This is associated with the external compression of the heart and the critically reduced transmural filling pressures of tamponade that prevent atrial distention. (In contrast, transmural pressures are preserved in cardiac failure, permitting myocardial stretch with consequent atrial natriuretic factor production). Thus, tamponade prevents this mechanism for increasing renal natriuresis. This, with the neurogenic renal sodium retention,²³ contributes to increased blood volume, tending to support the compensatorily increased venous pressures (Fig 2).

TAMPOONADE IN SUMMARY

Figure 2 summarizes the principal elements of tamponade dynamics and the main compensatory mechanisms. The discussion emphasizes the complexity of each of these, adding the important neurohormonal elements that resemble those of congestive heart failure. The pathophysiologic continuum of the tamponade state—“not all or none”³—is fundamental to its appreciation.

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