**Case report**

**English**

45 years old Caucasian woman with history of breast cancer (diagnosed 3 years ago). She is in radiotherapic and chemotherapic treatment. She complained of progressive dyspnea for two weeks, but it got worse in the last 2 hours (at rest). Additionally, she refers weight loss, fatigue, anorexia, and positional chest pain.

**Physical**

Dyspnea at rest, tachycardic, hypotension (BP = 90/50 mmHg), respiratory rate of 20/minute and temperature of 37.5°C. Paradoxical pulse with increased jugular venous pressure (presence of Kussmaul sign), dysphoria, and hypophonesis of the heart sounds.

Questions:

1. Which is the ECG diagnosis and why?
2. Which is the most probable clinical diagnosis?

We are waiting for your valuable opinions.

Portuguese

Mulher branca de 45 anos com história de tratamento com radioterapia e quimioterapia de câncer de mama há 3 anos. Queixa de dispneia progressiva há 2 semanas, com piora marcada nas últimas 2 horas, além da perda de peso, fadiga, anorexia e dor precordial posicional.

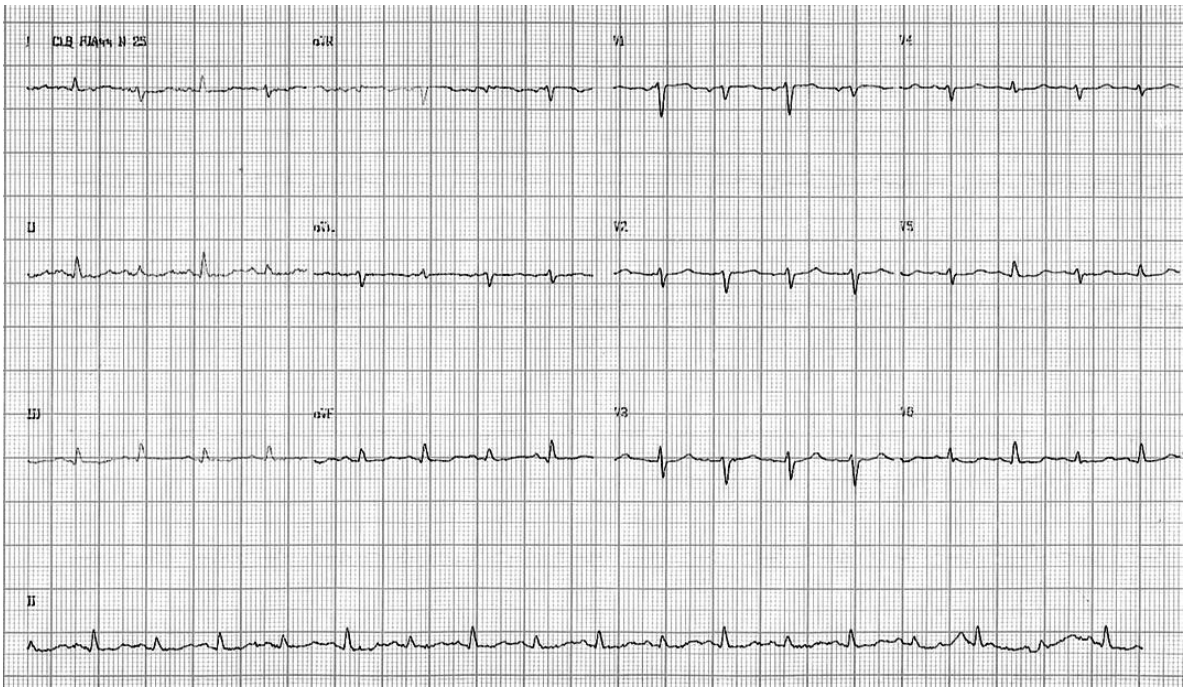
**Exame físico**: dispneica em repouso, taquicárdica e hipotensa (PA 90/50 mmHg), taquipneica (FR 20/min) e com febre moderada (37,5°C). Pulso paradóxico com aumento do pulso venoso jugular (presença de sinal de Kussmaul), disforia e hipofonese dos ruídos cardíacos.

Perguntas:

1. Qual é o diagnóstico ECG e por quê?
2. Qual é o mais provável diagnóstico clínico?

Esperamos pelas suas valiosas opiniões.

**Andrés Ricardo Pérez-Riera, MD, PhD**



**Colleague's opinions**

Dear Andrés,

Sinus tachycardia, unusual QRS electrical alternans, right bundle branch block morphology in some leads, micro voltage, slightly enhanced AV conduction.

I would say tamponade plus a myocardial infiltrative process or an associated pulmonary embolism.

Best,

Philippe **Chevalier** MD PhD. Lion France Claude Bernard University Lyon 1 Service de Rythmologie Villeurbanne, France

Looks like cardiac tamponade

**Melvin Scheinman MD**

**Cardiac Electrophysiology and Arrhythmia Service**
400 Parnassus Ave., Floor B1, Room 094
San Francisco, CA, USA

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**Portuguese**
**Maestro,minha opinião:**

**1-ECG mostra baixa voltagem com alternância elétrica do QRS**
**2-Diag clínico: tamponamento cardíaco**
**3-Invasão/metástase pericárdica**

**English**

**Master, my opinion:**

**1-ECG shows low QRS voltage with electric alternance**
**Clinical Diagnosis: cardiac tamponade**
**Invasion / pericardial metastasis**

Raimundo **Barbosa-Barros**, MD Fortaleza Ceará Brazil

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Dear Andrés

The ECG shows sinus rhythm, low QRS voltage, and clear electrical alternans and is consistent with the clinical diagnosis of cardiac tamponade that you describe.

In this case, there is most likely metastatic involvement of the pericardium with a large pericardial effusion (frequently hemorrhagic) resulting in the clinical presentation that you describe. An echocardiogram will probably show beat to beat swinging of the heart.

The patient needs to have the pericardial fluid drained as soon as possible.

Best regards,

**Mario Gonzalez M.D. Cardiovascular Disease Electrophysiology.**

Penn State Hershey Heart and Vascular Institute
500 University Drive
Hershey, PA 17033 Tel: 800-243-1455Fax: 717-531-4077

http://www.pennstatehershey.org/heartandvascular

**Portugues**

1) **ECG: belo exemplo de tamponamento cardíaco, com os 2 sinais clássicos - importante baixa voltagem e alternância elétrica. Ótimo para ilustrar aulas.**
2) **Derrame pericárdico - uma possibilidade: metástase no pericárdio. Pode ser um hemopericárdio (?).**

**Numa época na qual muitos médicos estão, primeiramente, pedindo o ECOCG, para depois examinar o paciente (se o ECO for anormal!), felicito o Andrés pela valorização da clínica (propedêutica): lembrou-nos que o pulso arterial paradoxal e o pulso venoso de Kussmaul não "morreram". Basta procurá-los. Peço permissão para mostrar este caso aos alunos. Cordial abraço.**

**Paulo Roberto P Toscano (Belém, Pará, Brasil)**

***Resposta de Andrés para Paulo Roberto***

Prezado Paulo Roberto da linda Belém do Pará Este caso não é de minha propriedade deve usar para uma causa nobre o ensinamento aos alunos. Peço-te que esperes uns dias para os comentários finais.

Apenas faço uma observação sobre o diagnóstico de alternância elétrica. Esta é definida como uma variação batimento a batimento (AB-AB-AB) da morfologia o configuração, amplitude e/ou polaridade (mudança do eixo) das ondas P, complexos QRS, segmentos ST, ondas T ou ondas U isoladas ou da combinação delas, considerando que se originam em um marcapasso. Quando a P, QRS, e T estão envolvidas é denominada alternância total. A alternância do segmento ST associada ou não a alternância do QRS se há descrito no padrão Brugada, durante a isquemia miocárdica transmural (1) e recentemente na intervenção percutânea coronariana "percutaneous coronary intervention (PCI)" (2)

O tipo de alternância mais frequente é aquele que afeta apenas ao complexo QRS. Quando inclui as três principais ondas do ECG com ou sem o segmento ST (P, QRS e T) denomina-se alternância total a qual é observada em casos de tamponamento cardíaco. Este tipo de alternância na realidade é uma pseudo-alternância ou alternância aparente porque resulta de uma mudança rotacional batimento a batimento sequencial do coração suspenso dentro do saco pericárdico cheio de líquido (3). É o que se há denominado muito propriamente "the dancing heart"(4),isto é, consequência de um movimento oscilante do coração na cavidade pericárdica batimento a batimento com mudanças na amplitude e no eixo dos complexos QRS. Nela não existe alternância intrínseca eletrofisiológica por tanto não haverá ou terá menor risco de eventos taquiarrítmicos. Existe uma alternância do QRS durante as taquicardias de QRS estreito como na taquicardia ortrodrómica, atrial, e atrioventricular

AV reentrante de QRS estreito as quais são devidas a um fenômeno dependente da frequência por abrupto aumento para uma frequência crítica independente do mecanismo da taquicardia. Nestes casos, durante a estimulação atrial a frequência maior resultou em alternância do QRS nos pacientes que tiveram alternância durante a taquicardia A presença de alternância no estava relacionada com o mecanismo da taquicardia o período refratário relativo o funcional do sistema His Purkinje e sim depende de um aumento abrupto da FC para uma taxa crítica e é independente do mecanismo de taquicardia.(5)

Em 1978, Klein, Segni e Kaplinsky cunharam o termo pseudo-alternância elétrica "pseudoelectrical alternans" em um relato de caso de bloqueio divisional antero-superior esquerdo com mudança do eixo para a esquerda, presumivelmente relacionada a terapia com procainamida que ocasionou uma sequencia Wenckebach 2:1 (6). Neste conceito alternância pseudo-electrica deve ser considerada. aos eventos que alteram a condução em forma intermitente e não a orientação física do coração dentro do saco pericárdico como ocorre no tamponamento cardíaco, nestes casos se observa no ECG variações batimento a batimento tanto da amplitude quanto do eixo dos complexos QRS (7).

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Andrés

**Spanish**
**Estimado Dr. Perez Riera**

**Siguiendo sus instrucciones considero:**

**Questions:**
**1. Cual es el diagnostic electrocardigráfico y porque?**

**El trazado electrocardiográfico tiene varias características que avalan la posibilidad diagnostica de derrame pericárdico importante con criterios de taponamiento, estos son:**

- **Taquicardia sinusal (con una frecuencia de descarta de 111 LPM aproximadamente)**
- **Criterios electrocardiográficos de bajo voltaje ( uno de los criterios descrito es que la suma de los QRS en las derivaciones DI-DII-DIII no suman mas de 15mm)**
- **Alternancia eléctrica del QRS**

**2. Cula es el diagnostic clinic mas probable?**

**En el contexto clínico que se describe reúne la descripción clínica de UN TAPONAMIENTO CARDIACO. (hipotensión, disnea, polipnea, pulso paradójico, y ruidos cardiacos hipofonéticos). Por sus antecedentes la primera opciones diagnostica y mas probable sería: DERRAME PERICARDICO IMPORTANTE CON CRITERIOS DE TAPONAMIENTO DE ETIOLOGIA NEOPLASICA**

**Gracias.**
Solon Nabarrete

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**Spanish**

**Para mi son extrasístoles atriales bigeminadas, ya que las ondas P de los complejos más pequeños, podría ser que esta paciente sufre de cardiomiopatía dilatada, post debido a la destrucción de miocitos ya sea por irradiación o tratamiento irradiación Segunda posibilidad ,podria considerse una pericardiopatia constrictiva oncológico Según mi esperiencia las extrasistoles atriales bigeminadas ,persistente , indica una Pobre mujer, lesión severa atrial ,que termina frecuentemente en fibrilación auricular tan joven con pronóstico severo Un fraternal abrazo**

**Samuel Sclarovsky Israel**

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Spanish

Estimado Andrés: Creo que como se ha expresado anteriormente el cuadro clínico y ECG corresponde a un derrame pericárdico con taponamiento cardíaco, todos los signos que presenta la paciente son de bajo gasto cardíaco y congestión venosa: disnea, presión venosa elevada, signo de Kussmaul positivo, pulso paradojal, hipotensión arterial; unido a un ECG que muestra un ritmo sinusal con pseudoalternancia tanto del complejo QRS como de la onda P y la onda T, con complejos de bajo voltaje lo cual apoyaría dicho diagnóstico. Lo que observo además es un agradamiento y/o trastorno de conducción de AI y un segmento ST rectificado en las derivaciones izquierdas con una onda T que tiende a ser negativa. Como bien ya lo has expuesto esta no es un alternancia eléctrica verdadera ya que es ocasionada por el movimiento cardíaco y es debida a cambios de la posición cardiaca en relación al registro de los electrodos exploradores y es efectivamente un artefacto. Otra patología que hemos visto que puede ocasionar estos cambios es la miocardiopatía hipertrófica; por supuesto con otras características del ECG basal. Si bien el cáncer de mama puede presentar metástasis que comprometen el pericardio produciendo derrame y taponamiento cardíaco; no nos debemos olvidar de las miocardiopatía por cardiotoxidad del tratamiento que en más de una oportunidad nos produce insuficiencia cardiaca congestiva. En esta paciente el gran compromiso hemodinámico obliga a maniobras de descompresión rápida y después vendrán las especulaciones etiológicas.

Atte. Isabel Konopka Buenos Aires Argentina

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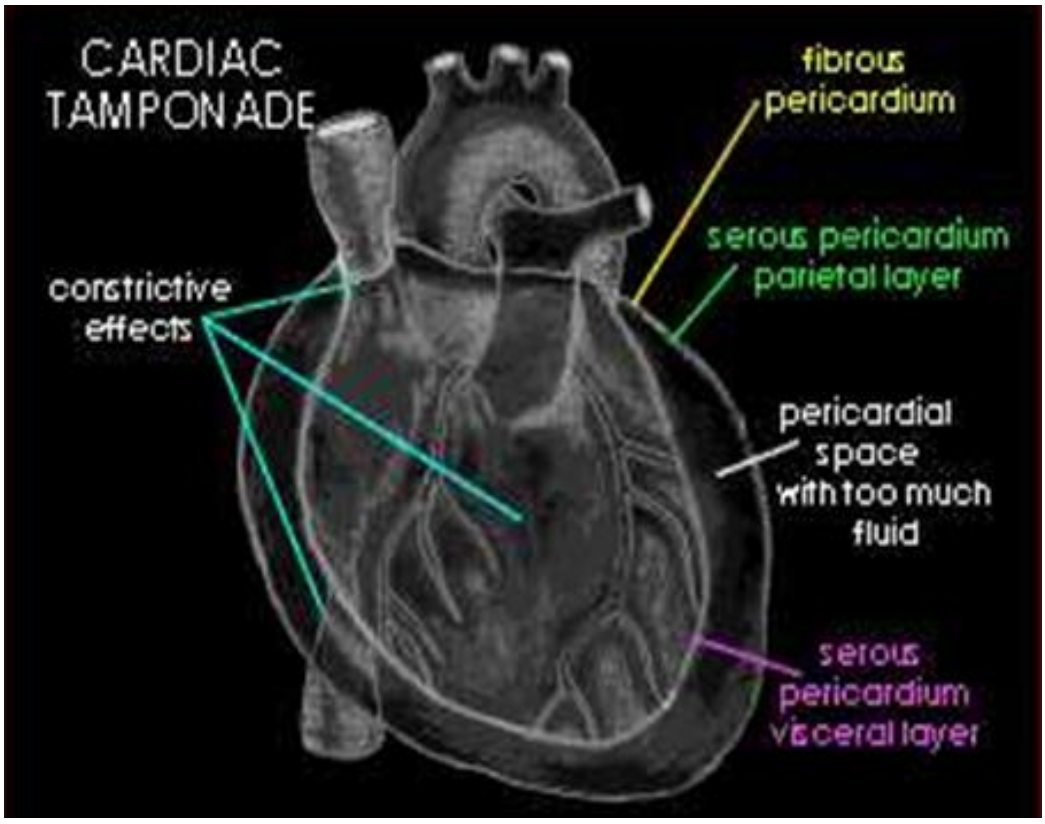
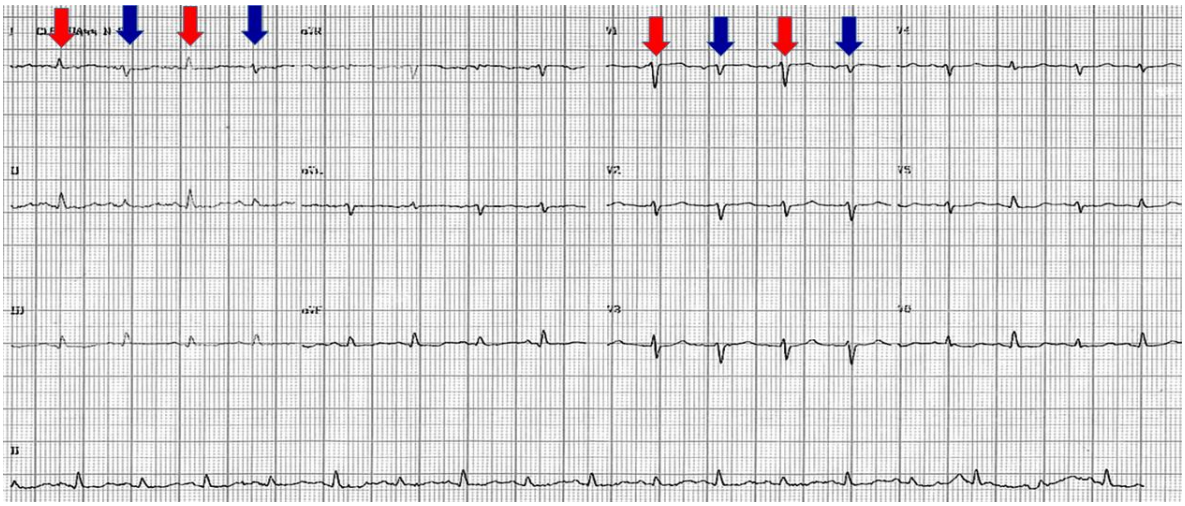
**Final comments By Andrés Ricardo Pérez-Riera M.D. Ph.D.**

**Clinical diagnosis:** Cardiac tamponade**.** Pericardial effusion with the heart "swinging" in it and changing its location and proximity to the chest wall (where the electrodes are) from beat to beat. Neoplastic pericarditis with massive pericardial effusion (metastasis of breast cancer). The pressure prevents complete filling of the heart before the next heartbeat. This lessens the amount of blood that can be pumped by the heart. Severe pericardial fluid pressure can cause a drop in blood pressure, shock, abnormal heart rhythms, and death. Basically, there is just too much fluid accumulating too fast in the pericardial space!!!

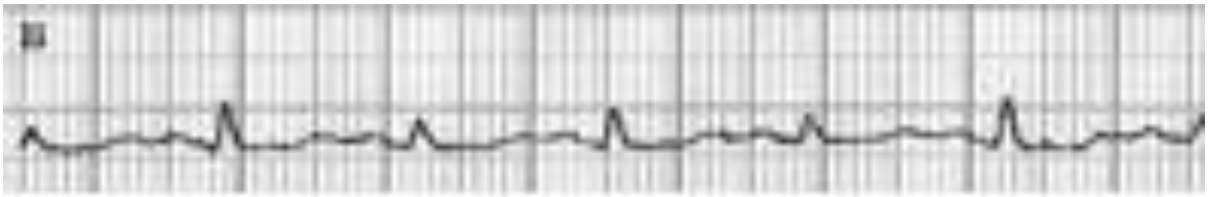
The fluid in cardiac tamponade can be blood, purulence, or effusion fluid (serum leaking out of blood vessels)...it doesn't matter. It just has to accumulate in that space surrounding the heart, causing increased pressure on the heart, so that the ventricles and atria cannot fill during diastole. With rapid accumulation of fluid, 100-200 cc may be enough to cause death. If the fluid accumulates slowly, the pericardium will expand, and perhaps 1 liter would be necessary to cause death



**ECG analysis**



**ECG diagnosis:** Regular sinus tachycardia, (HR 115 bpm) PR interval 200 ms, low P voltage preceding each QRS complex of normal duration of low QRS voltage. The QRS complexes in both frontal plane (≤ 5mm or 1 large square or 5 small squares) and horizontal plane (no wave exceeds ≤ 10 mm) and beat to beat alternating amplitudes and axis, of the QRS complexes and the configuration of QRS (electrical alternance)



Low voltage of the P wave and T-wave changes. These ECG features are more frequent than low QRS voltage. (**Habashy 2004**).

**Clinical diagnosis Cardiac tamponade**

**Concept** Cardiac tamponade is a clinical syndrome characterized by pressure on the heart that occurs when blood or fluid builds up in the space between the heart muscle (myocardium) and the outer covering sac of the heart (pericardium) resulting in reduced ventricular filling and subsequent hemodynamic compromise. The condition is a medical emergency, the complications of which include pulmonary edema, shock, and death.

Main causes of pericardial effusion are numerous and precipitating factors causing cardiac tamponade are

Likely to progress to cardiac tamponade

**Neoplastic diseases:** Pericardial neoplastic disease usually is metastatic, and by the time of detection it frequently has a previously diagnosed primary tumor. Metastatic malignancies

affecting the pericardium are associated with terminal cancer patients, with the vast majority of cases not being detected premortem due to a classic absence of clinical manifestations (**Lentoja 2007**). Pericardium metastatic involvement can occur through three main routes: hematogenous, lymphatic, and local extension (**Barbetakis 2003**) For many years, the hematogenous pathway has been considered the main spread route by some authors, although others, like Warren and Kline, consider the lymphatic system as the most common metastatic pathway.(**Kline 1972**)

**Infectious agents:** these include a number of viral, bacterial, fungal and parasitic agents. (i.e. viral: EBV, CMV enteroviruses Human immunodeficiency virus, bacterial, especially tuberculosis). Human immunodeficiency virus–associated pericardial tuberculosis complicated by cardiac tamponade is another combined etiology.(**Heller 2010**)

**Iatrogenic hemopericardium:** In a contemporary cohort, complications of percutaneous cardiac intervention replaced malignant diseases as the leading cause of cardiac tamponade. Nevertheless, these iatrogenic complications were associated with relatively favorable outcome compared to tamponade induced by complications of myocardial infarction, coagulation abnormalities and malignant diseases. (**Orbach 2015**).

**Post-traumatic pericardial effusion:** The term "post-cardiac injury syndromes" includes post-myocardial infarction pericarditis, post-pericardiotomy syndrome, and post-traumatic pericarditis (iatrogenic, i.e. after percutaneous coronary or intracardiac interventions, such as pacemaker lead insertion, radiofrequency ablation, or non-iatrogenic, i.e. following blunt or penetrating trauma). All these conditions represent different clinical conditions characterized by an initial cardiac injury involving the pericardium/myocardium and/or pleura and the subsequent inflammatory syndrome ranging from simple, uncomplicated pericarditis to more complicated cases with pleuropericarditis, cardiac tamponade or massive pleural effusion. The etiopathogenesis is presumed to be immune-mediated in predisposed individuals that develop autoreactive reactions following the initial traumatic event.(**Imazio 2013**).

**Post-pericardiotomy syndrome:** this syndrome may be associated with tamponade and pericardial constriction that may require procedural intervention. Younger age, early-onset Postpericardiotomy syndrome, and postoperative constrictive physiology are associated with the need for procedural intervention in patients with Postpericardiotomy syndrome, whereas colchicine is associated with reduced odds of adverse events and procedural intervention (**Alraies 2014**).

**Hemopericardium in acute type A aortic dissection:** Cardiac tamponade has been reported in 18.7% of patients with acute type A aortic dissection and its presence is associated with worse outcomes. Emergency aortic repair together with intra-

operative pericardial drainage is the recommended treatment approach. However, controversy surrounds how to manage patients with haemopericardium and cardiac tamponade who cannot survive until surgery. Preoperative controlled pericardiocentesis can be lifesaving when managing patients with critical cardiac tamponade (pulseless electrical activity or refractory hypotension) complicating acute type A aortic dissection, namely when cardiac surgery is not immediately available. **(Cruz 2014)**

**Renal failure(Adams 2013)**

**Associated with Rivaroxaban(Boone 2015)**

Rarely progressing to cardiac tamponade

**Systemic autoimmune disease** collagen vascular disease **or autoimmune inflammatory disorders**: like acute rheumatic fever, juvenile rheumatoid arthritis, rheumatoid arthritis, Scleroderma, and Systemic Lupus Eritematous(**Maharaj 2015**).

**Autoreactive pericardial effusions:** The role of inflammatory and angiogenic cytokines such as Vascular endothelial growth factor (VEGF), basic fibroblast growth factor, (bFGF), Interleukin-1 beta(IL-1*β)* and tumor necrosis factor alpha, (TNFα) cachexin, or cachectin) in patients with inflammatory pericardial effusion still remains uncertain. VEGF and bFGF levels in pericardial effusion are elevated in patients with inflammatory pericardial effusions. It is thus possible that VEGF and bFGF participate in the pathogenesis of inflammatory pericardial disease.( **Karatolios 2012**)

**Hypotiroid tamponade ( hypothyroidism)**: For patients diagnosed with cardiac tamponade without sinus tachycardia, hypothyroidism should be highly suspected. Although emergent pericardiocentesis should be performed in clinical cardiac tamponade, patients with echocardiographic tamponade signs without a paradoxical pulse should be treated with thyroxine initially. ( **Wang 2010**)-

Hyperthyreosis Graves' thyrotoxicosis (**Teague 2009**)

**Early and late pericarditis (Dressler's syndrome) in acute myocardial infarction(Paelinck2003)**

**Any other aetiology of pericardial disease (i.e. cholesterol pericarditis(Raposo 2005), idiopathic chylopericardium(Mehrotra 2006)**

**Never progressing to cardiac tamponade**

**Pericardial transudates caused by heart failure or pulmonary hypertension**

**Pericardial transudates in the last trimester of normal pregnancy**

**Precipitating factors**

**Drugs**

**Antihypertensive medications**

**Anticoagulants, thrombolytics, etc.**

**Injury**

**Complex Percutaneous coronary intervention (PCI)**

**Pacemaker implantation** (**Navarrete 2005**)

**Left ventricle (LVEMB) and right ventricle (RVEMB) Endomyocardial biopsy**

**Complications of LVEMB and RVEMB (Chimenti 2013)**

| | LVEMB (n=3549), n (%) | RVEMB (n=3068), n (%) | *P*Value |
|---|---|---|---|
| Major complication | | | |
| Perforation with cardiac tamponade | 3 (0.08) | 9 (0.29) | 0.033 |

**Recent cardiac surgery**

**Indwelling instrumentation**

**Blunt chest trauma**

**Septicemia**

**Dehydration, diuretics (reduced circulating volume)**

**After percutaneous catheter ablation (Goossens 2012)**

## Clinical picture

Symptoms vary with the acuteness and underlying cause of the tamponade. Patients with acute tamponade may present with dyspnea, tachycardia, and tachypnea. Cold and clammy extremities from hypoperfusion are also observed in some patients. A comprehensive review of the patient's history usually helps in identifying the probable etiology of a pericardial effusion. The following may be noted: Patients with systemic or malignant disease present with weight loss, fatigue, or anorexia, chest pain may be the presenting symptom in patients with pericarditis or myocardial infarction, musculoskeletal pain or fever may be present in patients with an underlying colagenopathies, a history of renal failure can lead to a consideration of uremia as the cause of pericardial effusion, careful review of a patient's medications may indicate that drug-related lupus caused the pericardial effusion, recent cardiovascular surgery, coronary intervention, or trauma can lead to the rapid accumulation of pericardial fluid and tamponade (**Rylski 2010**), a recent pacemaker lead implantation or central venous catheter insertion can lead to the rapid accumulation of pericardial fluid and tamponade (**Lee 2009**), consider HIV-related pericardial effusion and tamponade if the patient has a history of IV drug abuse or opportunistic infections, inquire about chest wall radiation, for lung, mediastinal, or esophageal cancer and about symptoms of night sweats, fever, and weight loss, which may be indicative of tuberculosis.

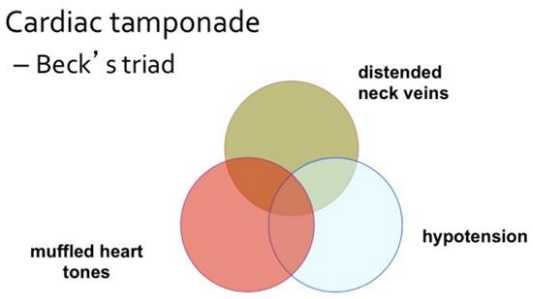
## Physical Examination

In a retrospective study of patients with cardiac tamponade, the most common symptoms noted by Roy et al were dyspnea, tachycardia, and elevated jugular venous pressure (**Roy 2007**). Evidence of chest wall injury may be present in trauma patients.

Tachycardia, tachypnea, and hepatomegaly are observed in more than 50% of patients, and diminished heart sounds and a pericardial friction rub are present in approximately 30% of patients. Some patients may present with dizziness, drowsiness, or palpitations. Cold, clammy skin and a weak pulse due to hypotension are also observed in patients with tamponade.

## Acute Cardiac compression triad, “Beck triad”

1. Increased venous pressure
2. Decreased arterial pressure
3. Muffled heart sounds

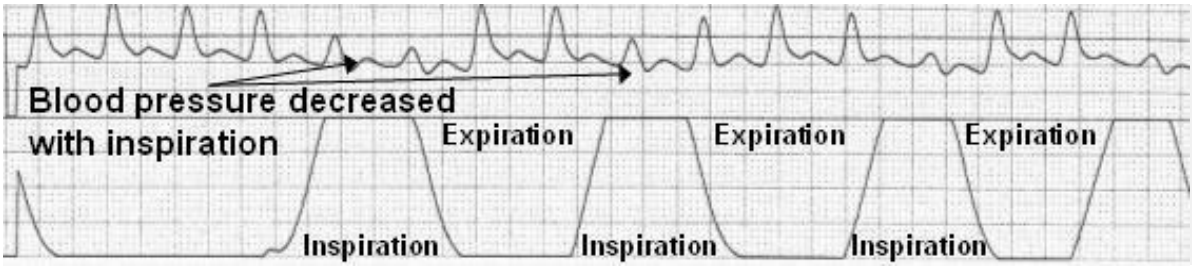


| Grade | Pericardial Volume (mL) | Cardiac Index | MAP | CVP | HR | Beck's Triad |
|---|---|---|---|---|---|---|
| **I** | < 200 | Normal or ↑ | Normal | ↑ | ↑ | Usually not present |
| **II** | ≥ 200 | ↓ | Normal or ↓ | ↑ (≥12 cm $H_2O$) | ↑ | May or may not be present |
| **III** | > 200 | ↓↓ | ↓↓ | ↑↑ (≤30–40 cm $H_2O$) | ↓ | Usually present |

Described in 1935 (**Sternbach 1988**), this complex of physical findings, also called the acute cardiac compression triad, refers to increased jugular venous pressure, hypotension, and diminished heart sounds. Claude Beck described two triads of clinical findings which he found constituted the essential components of acute and chronic cardiac tamponade. The first of these triads consisted of hypotension, an increased venous pressure, and a quiet heart. It has come to be recognized as "Beck's triad," a collection of findings most commonly produced by acute intrapericardial hemorrhage. Subsequent studies have shown that these classic findings are observed in only a minority of patients with cardiac tamponade. However, Beck deserves credit for presenting a physiologic basis for the signs of cardiac compression. These findings result from a rapid accumulation of pericardial fluid. This classic triad is usually observed in patients with acute cardiac tamponade.

**Pulsus paradoxus or paradoxical** pulse is an exaggeration (>12 mm Hg or 9%) of the normal inspiratory decrease in systemic blood pressure. To measure the pulsus paradoxus, patients are often placed in a semirecumbent position; respirations should be normal. The blood pressure cuff is inflated to at least 20mm Hg above the systolic pressure and slowly deflated until the first Korotkoff sounds are heard only during expiration. At this pressure reading, if the cuff is not further deflated and a pulsus paradoxus is present, the first Korotkoff sound is not audible during inspiration. As the cuff is further deflated, the point at which the first Korotkoff sound is audible during both inspiration and expiration is recorded.

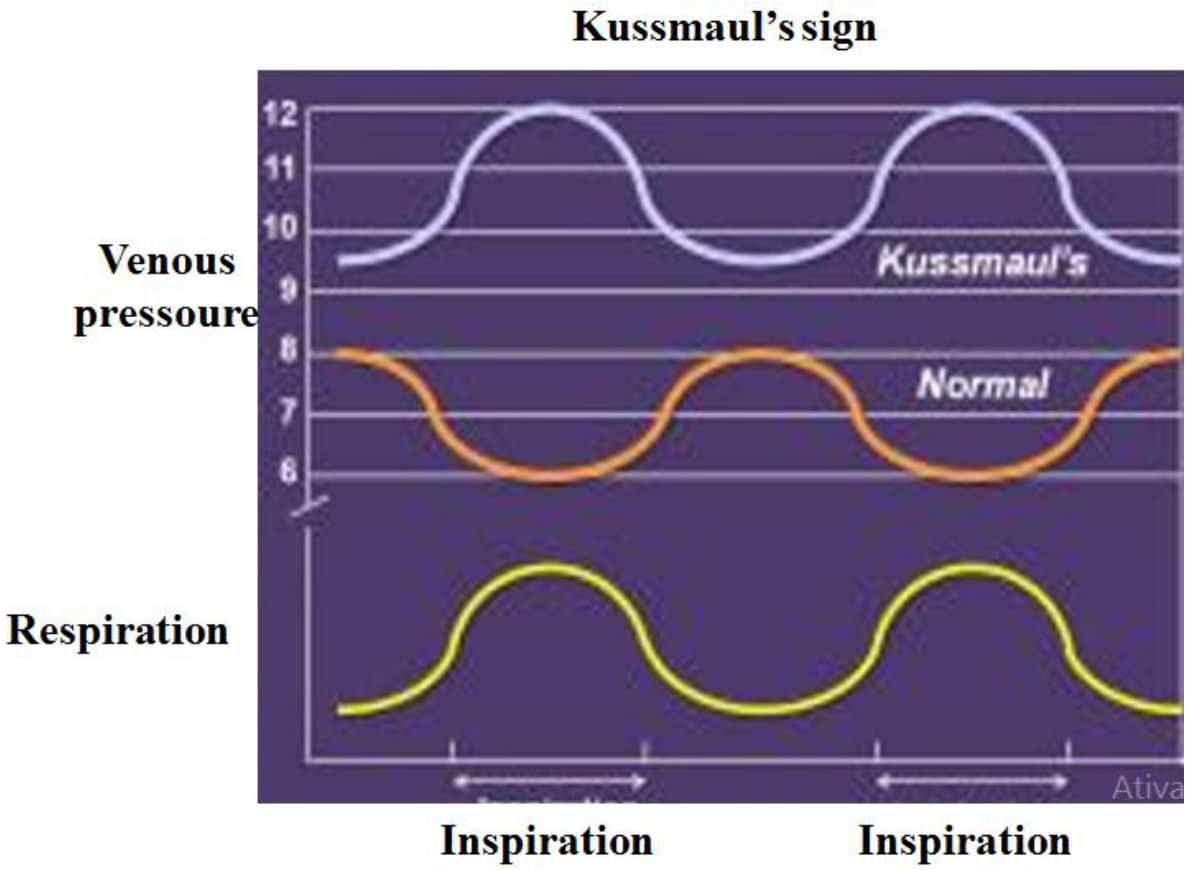
If the difference between the first and second measurement is greater than 12 mm Hg, an abnormal pulsus paradoxus is present.



The paradox is that while listening to the heart sounds during inspiration, the pulse weakens or may not be palpated with certain heartbeats, while $S_1$ is heard with all heartbeats.

A pulsus paradoxus can be observed in patients with other conditions, such as constrictive pericarditis, asthma, severe obstructive pulmonary disease, restrictive cardiomyopathy, pulmonary embolism, rapid and labored breathing, and right ventricular infarction with shock. A pulsus paradoxus may be absent in patients with markedly elevated LV diastolic pressures, atrial septal defect, pulmonary hypertension, aortic regurgitation, low-pressure tamponade, or right heart tamponade.

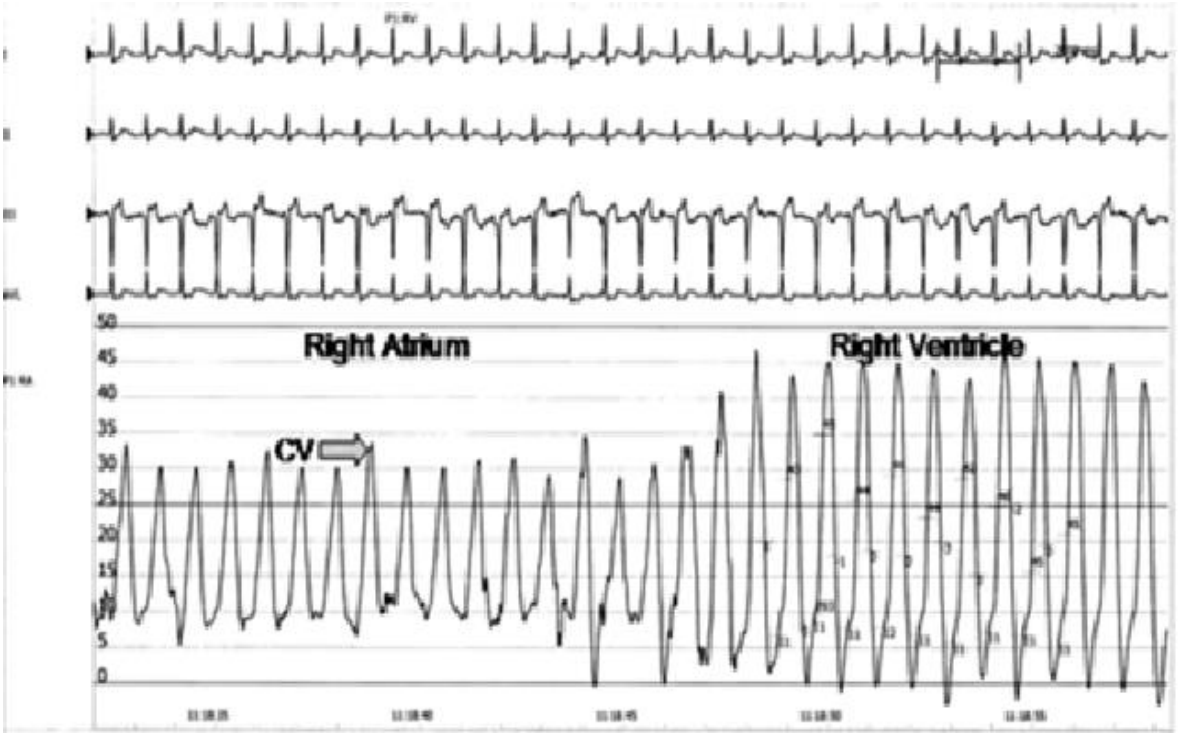
**Kussmaul's sign (Mansoor 2015)** It is an increase in jugular venous pressure on inspiration instead of the normal decrease in jugular venous pressure. Describe what is happening during inspiration that would lead to the Kussmaul's sign? During inspiration there will be a negative intra-thoracic pressure that's not transmitted to the heart. This will lead to impaired filling of the right ventricle. Now because of the impaired filling of the right ventricle, blood backs up into the vena cava. That means there's gonna be a lot more blood in the vena cava which would lead to that jugular venous distention. What are some of the conditions in which you would see the Kussmaul's sign? some of the conditions in which you would see the Kussmaul's sign are **constrictive pericarditis, restrictive cardiomyopathy, right atrial or ventricular tumors and cardiac tamponade**. This was described by Carl Philipp Adolf Konrad Kussmaul as a paradoxical increase in venous distention and pressure during inspiration.



**Bamberger-Pins-Ewart sign, Ewart sign (Ewart 1896) or** Pins' syndrome: this is observed in patients with large pericardial effusions. It is described as an area of dullness, with bronchial breath sounds and bronchophony below the angle of the left scapula. Typically localized pulmonary auscultation- and percussion phenomenon in large pericardial effusion, with dullness to percussion, increased fremitus and bronchial breathing. Between the vertebral column and the scapula, a blowing sound can be heard. This is generally heard on the left side, but can also be heard on the right. Due to compressive atelectasis by the large pericardial sac. Bamberger is probably the German internist Heinrich von Bamberger (1822-1888), but we are not certain about this.

### The y descent

The *y* descent is abolished in the jugular venous or right atrial waveform. This is due to an increase in intrapericardial pressure, preventing diastolic filling of the ventricles.




Hemodynamic trace obtained by right heart catheterization, demonstrating right atrial and Right ventricular pressures CV indicates cannon V waves

### Dysphoria

Behavioral traits such as restless body movements, unusual facial expressions, restlessness, and a sense of impending death were reported by Ikematsu in about 26% patients with cardiac tamponade (**Ikematsu 2007**).

### Low-pressure tamponade

In severely hypovolemic patients, classical physical findings such as tachycardia, pulsus paradoxus, and jugular venous distention were infrequent. Sagristà-Sauleda et al identified

low-pressure tamponade in 20% of patients with cardiac tamponade (**Sagristà-Sauleda 2006**). They also reported low-pressure tamponade in 10% of large pericardial effusions.

**Diagnostic Considerations**

Early diagnosis with a high index of suspicion is necessary to minimize morbidity and mortality from tamponade.

**Large pleural effusion**

Cases of cardiac tamponade have been reported with large pleural effusions. The increased intrapleural pressure resulting from large pleural effusions can be transmitted to the pericardial space and impair ventricular filling, thus producing the hemodynamic equivalent of cardiac tamponade.

**Tension pneumopericardium**

The hemodynamic changes in tension pneumopericardium simulate acute cardiac tamponade. Clinically, distant heart sounds, bradycardia, and shifting tympany occur over the precordium, and a characteristic murmur, termed bruit de la roue de moulin, is heard. This is usually observed in infants with mechanical ventilation but is also seen after sternal bone marrow aspiration, penetrating chest wall injury, esophageal rupture, and bronchopericardial fistula.

**Rapid and labored breathing**

Large decreases in intrathoracic pressure with deep inspirations, often observed during respiratory failure, can accentuate pulsus paradoxus, simulating pericardial tamponade.

Cardiac tamponade is responsible for an obstructive shock.

**Electrocardiographic features in pericardial tamponade**

The features are not diagnostic for, pericardial tamponade but suggestive. Low voltage of the P wave, QRS complex and T waves, QRS alternance, and PR segment depression are suggestive ECG signs, but not diagnostic, of pericardial effusion and cardiac tamponade (**Eisenberg 1996**). Because these ECG findings cannot reliably identify these conditions, we conclude that 12-lead ECG is poorly diagnostic of pericardial effusion and cardiac tamponade.

**I)** **Heart rate:** Sinus tachycardia is the rule. In absence suspect myxedema.

**II)** **Low voltage of the P wave:** Low voltage of the P wave and T-wave changes were more frequent than low QRS voltage (**Habashy 2004**).

**III)** **Low QRS voltage:** It is a feature of cardiac tamponade but not of pericardial effusion per se. The presence and severity of cardiac tamponade, in addition to inflammatory mechanisms, may contribute to the development of low QRS voltage in patients with large pericardial effusions (**Bruch 2001**). Several mechanisms have been proposed to explain the association between pericardial effusion and low QRS voltage, namely:

- Internal short circuiting of the electrical currents by the accumulated fluid within the pericardial space (**Toney 1966**)
- Changes beat to beat in the position of the heart (Toney 1966) "the dancing heart"( Richardson 2014) ,
- Increasing distance from the current generator to the recording electrodes (**Gonzalez 1996**)
- Decrease in cardiac chamber size and volume (**Karatay 1993**),
- Changes in the generation and propagation of electrical currents in the myocardium (**Karatay 1993**),
- Inflammation as well as changes in the propagation and generation of electrical currents in the myocardium. QRS amplitude normalization few days after initiation of anti-inflammatory treatment, suggests a potential role of inflammation,
- Brody's hypothesis, which claims that alterations in end-diastolic blood volume will change the magnitude of cardiac electrical potentials recorded at the body surface, changing the QRS voltage (**Brody 1956**).

Karatay et al. (**Karatay 1993**) produced cardiac tamponade in closed-chest pigs by the introduction of saline, blood and plasma into the pericardial space. Consequently, mean limb and precordial lead QRS voltage fell significantly, with no significant difference among these fluids. ECG recordings from a unipolar electrode catheter in the RV showed an increase in R wave voltage, whereas body surface recordings of stimuli introduced into the RA via a bipolar electrode catheter showed no amplitude change. The authors attributed their results to a reduction of cardiac volume and size during cardiac tamponade.

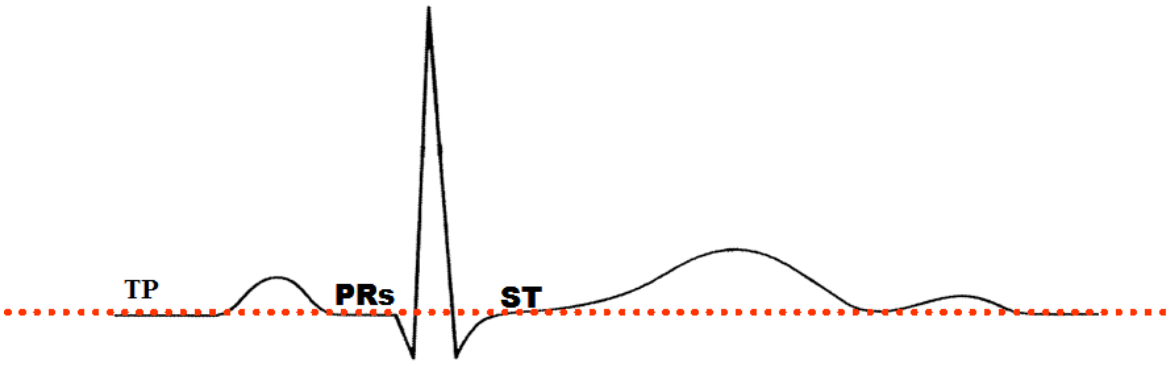
**IV)** **Electrical alternans:** The combination of QRS-amplitude alternans and QRS-vector alternans (namely, beat-to-beat variation of the QRS vector) on a 12-lead electrocardiogram is very highly specific for cardiac tamponade.QRS-vector alternans is most commonly appreciated in only 1 or 2 precordial leads. One must recognize that the sensitivity of vector alternans is low; however, when present, it is almost a give-away to thediagnosis. Total electrical alternans is yet another highly specific finding for the diagnosis of tamponade, which involves electrical alternation of P wave, QRS complex, ST segment, and T wave. Again, this finding is present in only in about 5% to 10% of cardiac tamponade patients, but itspresence is virtually quasispecific for the diagnosis (**Khalid 2015**). Electrical alternans is also observed in patients with myocardial transmural ischemia, (**Ortega Carnicer 2007**), acute pulmonary embolism, and tachyarrhythmias (**Morady 1987**) Pseudo electrical alternans is observed in procainamide-induced left anterior fascicular block of the 2:1 Mobitz type. (**Klein 1978**). Finally, a PR segment depression is observed sometimes.

Friedman et al (**Friedman 1977**) studied the ECG features of experimental cardiac tamponade in closed-chest dogs. In their study, acute cardiac tamponade produced by rapid saline infusion into the pericardium induced reduction of the QRS amplitude and left-axis deviation of the QRS complex, whereas the amplitude of the P wave remained essentially unchanged. Interestingly, in agreement with the findings of Karatay et al, the amplitude of bipolar ventricular ECG increased during tamponade, further supporting Brody's concepts. However, the authors of these animal studies focused on the impact of a rapidly produced

cardiac tamponade but not on a gradual intrapericardial fluid accumulation, which more closely reflects the situation in a daily clinical setting. Different mechanisms may interplay to explain low QRS voltage in patients with pericardial effusion and cardiac tamponade. The presence and severity of cardiac tamponade seem to be crucial factors because low QRS amplitude was observed only in subjects with clinical evidence of tamponade but not in patients with pericarditis and large effusions who were clinically stable. However, in patients with cardiac tamponade, no changes in the maximum QRS amplitude were observed immediately after pericardiocentesis. Following Brody's hypothesis, successful removal of intrapericardial fluid during pericardiocentesis should have increased the end-diastolic blood volume. Thus, the QRS amplitude should have increased after the procedure. Previously, it has been demonstrated that the epicardial circumference shortens proportionally less than the endocardial circumference, resulting in different tension distributions (**Rusmer 1970**). Schlant and Hurst (**Schlant and Hurst 1976**) have shown the strong impact of these electrical inhomogeneities on the surface ECG. Friedman et al have demonstrated that both intrapericardial and end-diastolic intraventricular pressures rise during cardiac tamponade, leading to compression of the myocardium in between. This increase of end-diastolic ventricular pressure contributes to the approximation of the surfaces of the myocardium. As a consequence, the mechanicoelectrical conditions of the endocardial and the epicardial myocardium become more alike, offering an explanation for the changes present in the surface ECG (**Karatay 1993**). Low QRS voltage is observed in the majority of subjects with cardiac tamponade but not in patients with pericarditis and large pericardial effusions. After pericardiocentesis and after initiation of anti-inflammatory treatment, QRS amplitude normalizes within one week. The presence and severity of cardiac tamponade, but also inflammatory mechanisms, may contribute to the development of low QRS voltage in patients with pericardial effusion.

## V) PR segment depression

The PR segment is the flat, usually isoelectric segment between the end of the P wave and the start of the QRS complex. Regarding the level of PRs, in normal conditions is at the same level as ST segment (isoelectric) and TP segment.




*PR segment changes are relative to the baseline formed by the* T-P segment. PR-segment depression is a relatively common ECG sign associated with clinically silent pericardial effusion, and it is an ECG indicator of inflammatory pericardial involvement. PR-segment depression is considered present when at least 0.5 mm of PR-segment depression from the TP

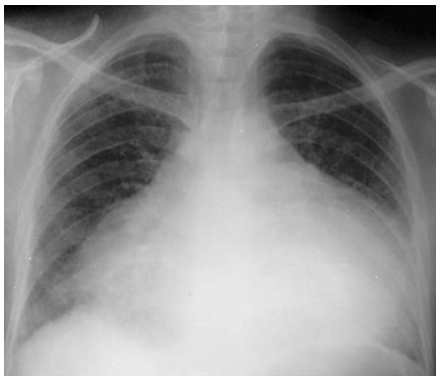
segment in both the limb (more than two leads in leads I, II, aVL and aVF) and precordial leads (more than two leads in leads $V_3$ through $V_6$). PR-segment depression is a reflection of underlying inflammatory conditions of the heart, for example, acute pericarditis, pericardial effusion, or atrial ischemia. Acute pericarditis or Dressler's syndrome is a late complication after acute MI and unlikely to be the cause, as all ECG changes were detected very early after onset of symptoms. Early pericardial effusion preceding rupture could be the other explanation, but in most of the patients with pericardial effusion (64.8%) PR-segment depression was < 0.5 mm. On the other hand, there was no pericardial effusion in more than half of PR-segment depressions ≥1.2 mm. Thus, pericardial effusion could possibly contribute, but not be the sole cause of profound PR-segment depression. Profound PR-segment depression probably represented a subgroup of patients who had extensive atrial ischemia or infarction in addition to acute inferior MI.

The diagnosis of acute pericarditis requires typical widespread J-ST–segment elevation (stage I ECG changes), but stage I ECG changes are frequently not recorded, and nearly half of the patients with acute pericarditis of mixed causes have a variation in the typical ECG evolution of acute pericarditis (**Spodik 1997; Bruce 1980**). PR-segment depression is also known to be one of the typical ECG changes associated with acute pericarditis (**Spodik 1973**) and (**Spodik 1974**). Pericarditis, sympathetic stimulation and left atrial overloading due to LV failure are some of the geneses of PR-segment depression (**Tranchesi 1960**) and (**Silvertssen 1973**). The ECG changes produced by hypersympathetic activity are characterized by concordant PR- and ST-segment depressions (the PR and ST-segments are in the arcs of circumference with the same radius). PR-segment depression frequently reflects abnormal atrial repolarization due to atrial inflammation.

In malignant pericardial effusion, combination of ECG abnormalities can supplement clinical examination in the diagnosis of echocardiographic cardiac tamponade. Due to its low negative predictive value, 12-lead ECG cannot be used as a screening tool to exclude cardiac tamponade with malignant pericardial effusion (**Argula 2015**).

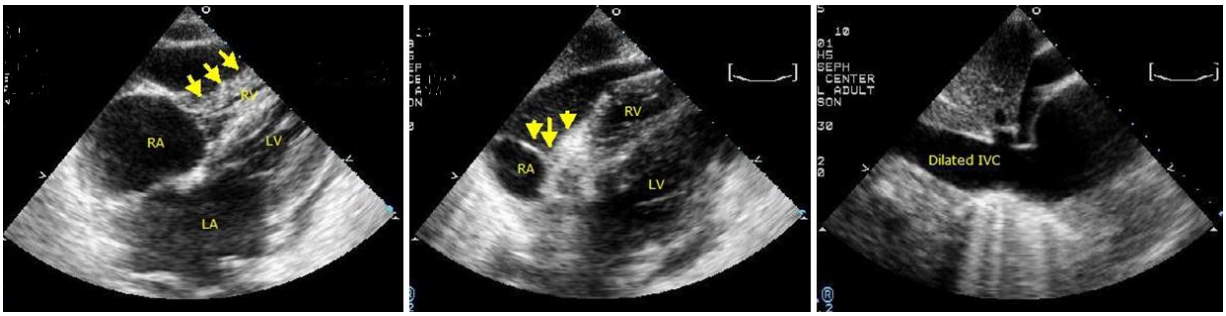
**Chest radiography**

Chest radiography findings may show cardiomegaly, a water bottle–shaped heart, pericardial calcifications, or evidence of chest wall trauma.



Chest X Ray show a massive bottle shaped cardiac silhouette

Echocardiography




| | | |
|---|---|---|
| Early diastolic collapse of right ventricular free wall (parasternal short-axis view at aortic valve). | Late diastolic collapse of right atrium (subcostal view). | Dilated inferior vena cava. |

Although echocardiography provides useful information, cardiac tamponade is a clinical diagnosis. The following may be observed with 2-dimensional (2-D) echocardiography: An echo-free space posterior and anterior to the left ventricle and behind the left atrium - After cardiac surgery, a localized, posterior fluid collection without significant anterior effusion may occur and may readily compromise cardiac output, Early diastolic collapse of the right ventricular free wall, swinging of the heart in its sac, LV pseudohypertrophy and inferior vena cava plethora with minimal or no collapse with inspiration.

**Pulse Oximetry**

Respiratory variability in pulse-oximetry waveform is noted in patients with pulsus paradoxus. In a small group of patients with tamponade, Stone et al noted increased respiratory variability in pulse-oximetry waveform in all patients (**Stone 2006**). This finding should raise the suspicion for hemodynamic compromise. In patients with atrial fibrillation, pulse-oximetry may aid in detecting the presence of pulsus paradoxus.

Before or after insertion of the Swan-Ganz catheter, the system must be zeroed after positioning the transducer at the midpoint of the left atrium. Then calibrate the monitoring system. Prior to insertion, test the balloon and flush all of the ports. Then insert the catheter into one of the major veins.

At a depth of 20 cm, inflate the balloon and slowly advance the catheter, while continuously monitoring the pressure from the distal lumen. Always deflate the balloon before withdrawing the Swan-Ganz catheter. The waveforms help to indicate the position of the catheter tip if fluoroscopy is not readily accessible.

At approximately the 40-50 cm mark, the wedge pressure is usually recorded. Secure the catheter position, and obtain a chest radiograph to confirm the position.

In tamponade, near equalization (within 5 mm Hg) of the right atrial, right ventricular diastolic, pulmonary arterial diastolic, and pulmonary capillary wedge pressure (reflecting left

atrial pressure) occurs. The right atrial pressure tracings display a prominent systolic *x* descent and abolished systolic *y* descent.

Boltwood et al (**Boltwood 1984**) described the diastolic equalization of pulmonary capillary and right atrial pressures as predominantly inspiratory; this is known as the inspiratory traction sign.It results from inspiratory traction of the taut pericardium by the diaphragm.

**Histologic finding**

Occasionally, a pericardial biopsy is performed when the etiology of the pericardial effusion that caused the tamponade is unclear. This is especially useful in cases of tuberculous pericardial effusions, because cultures of the pericardial fluid in these cases rarely yield a positive result for mycobacteria. However, granulomas seen on pericardial biopsy specimens are often seen in patients with tuberculous pericarditis. In general, cytopathologic findings from pericardial fluid and histologic findings from pericardial biopsy specimens depend on the underlying pathology. Cytologic examination identifies the etiopathologic cause of tamponade in about 75% of cases.

**Diagnosis of cardiac tamponade**

| Clinical presentation | **Elevated systemic pressure, tachycardia, pulsus paradoxus, hypotension, dyspnea or tachypnea with clear lungs** |
|---|---|
| **Precipitating factors** | Drugs (cyclosporine, anticoagulants, thrombolytic, etc.), recent cardiac surgery, indwelling instrumentation, blunt chest trauma, malignancies, connective tissue disease, renal failure, septicemia |
| **ECG** | Can be normal or non-specifically changed (ST-T wave), low P and QRS voltage, PR depression, electrical alternans (QRS, rarely T), bradycardia (end-stage), Electromechanical dissociation (agonal phase) |
| **Chest X-ray** | Enlarged cardiac silhouette with clear lungs |
| **M mode/2D echocardiogram** | Diastolic collapse of the anterior RV-free wall f RA collapse, LA and very rarely LV collapse, increased LV diastolic wall thickness “pseudohypertrophy”, VCI dilatation (no collapse in imperium), “swinging heart”. |
| **Doppler** | Tricuspid flow increases and mitral flow decreases during inspiration (reverse in expiration) Systolic and diastolic flows are reduced in systemic veins in expirium and reverse flow with atrial contraction is increased. |
| **M-mode color Doppler** | Large respiratory fluctuations in mitral/tricuspid flows |
| **Cardiac catheterization** | Confirmation of the diagnosis and quantification of the haemodynamic compromise. RA pressure is elevated (preserved systolic x descent and absent or diminished diastolic y descent) Intrapericardial pressure is also elevated and virtually identical to |

| | |
|---|---|
| | RA pressure (both pressures fall in inspiration) RV mid-diastolic pressure elevated and equal to the RA and pericardial pressures (no dip-and plateau configuration) Pulmonary artery diastolic pressure is slightly elevated and may correspond to the RV pressure Pulmonary capillary wedge pressure is also elevated and nearly equal to intrapericardial and right atrial pressure LV systolic and aortic pressures may be normal or reduced. Documenting that pericardial aspiration is followed by hemodynamic improvement. Detection of the coexisting hemodynamic abnormalities (LV failure, constriction, pulmonary hypertension). Detection of associated cardiovascular diseases (cardiomyopathy, coronary artery disease). |
| **RV/LV** | Atrial collapse and small hyperactive ventricular chambers. |
| **Coronary angiography** | Coronary compression in diastole |
| **Computer tomography** | No visualization of subepicardial fat along both ventricles, which show tube-like configuration and anteriorly drawn atria. |

### Differential diagnosis

- Cardiogenic Shock
- Constrictive Pericarditis
- Effusive-Constrictive Pericarditis
- Pneumothorax
- Pulmonary Embolism

### Approach Considerations

As previously stated, prompt diagnosis is key to reducing the mortality risk for patients with cardiac tamponade. Although cardiac tamponade is a clinical diagnosis, further assessment of the patient's condition and diagnosis of the underlying cause of the tamponade can be obtained through lab studies, imaging studies, and electrocardiography.

Echocardiography, for example, can be used to visualize ventricular and atrial compression abnormalities as blood cycles through the heart, while lab studies can demonstrate signs of myocardial infarction, cardiac trauma, and infectious disease.

The European Society of Cardiology (ESC) Working Group on Myocardial and Pericardial Diseases released a stepwise scoring system for treating patients with cardiac tamponade. The

system is used to identify patients who need immediate pericardiocentesis and patients who can safely be transferred to a specialized institution (Ristik 2014). According to the guidelines, patients with suspected cardiac tamponade should undergo echocardiography without delay. After diagnosis, patients are scored according to disease etiology, clinical presentation, and imaging findings. A score of 6 or more requires the patient to undergo immediate pericardial drainage. A lower score indicates that drainage can be postponed for up to 12 to 48 hours.

Cardiac tamponade is a medical emergency. Preferably, patients should be monitored in an intensive care unit. All patients should receive the following:

- Oxygen
- Volume expansion with blood, plasma, dextran, or isotonic sodium chloride solution, as necessary, to maintain adequate intravascular volume - Sagristà-Sauleda et al noted significant increase in cardiac output after volume expansion (**Sagristà-Sauleda 2008**).
- Bed rest with leg elevation - This may help increase venous return
- Inotropic drugs (eg, dobutamine) - These can be useful because they increase cardiac output without increasing systemic vascular resistance

Positive-pressure mechanical ventilation should be avoided because it may decrease venous return and aggravate signs and symptoms of tamponade.

### Inpatient care

After pericardiocentesis, leave the intrapericardial catheter in place after securing it to the skin using sterile procedure and attaching it to a closed drainage system via a 3-way stopcock. Periodically check for reaccumulation of fluid, and drain as needed.

The catheter can be left in place for 1-2 days and can be used for pericardiocentesis. Serial fluid cell counts can be useful for helping to discover an impending bacterial catheter infection, which could be catastrophic. If the white blood cell (WBC) count rises significantly, the pericardial catheter must be removed immediately.

A Swan-Ganz catheter can be left in place for continuous monitoring of hemodynamics and to assess the effect of reaccumulation of pericardial fluid. A repeat echocardiogram and a repeat chest radiograph should be performed within 24 hours.

Consultations associated with cardiac tamponade can include the following:

- Hemodynamically stable patients - Cardiologist
- Hemodynamically unstable patients - Cardiologist, cardiothoracic surgeon

### Activity

Initially, the patient should be on bed rest with leg elevation to increase the venous return. Once the signs and symptoms of tamponade resolve, activity can be increased as tolerated.

### Follow-up

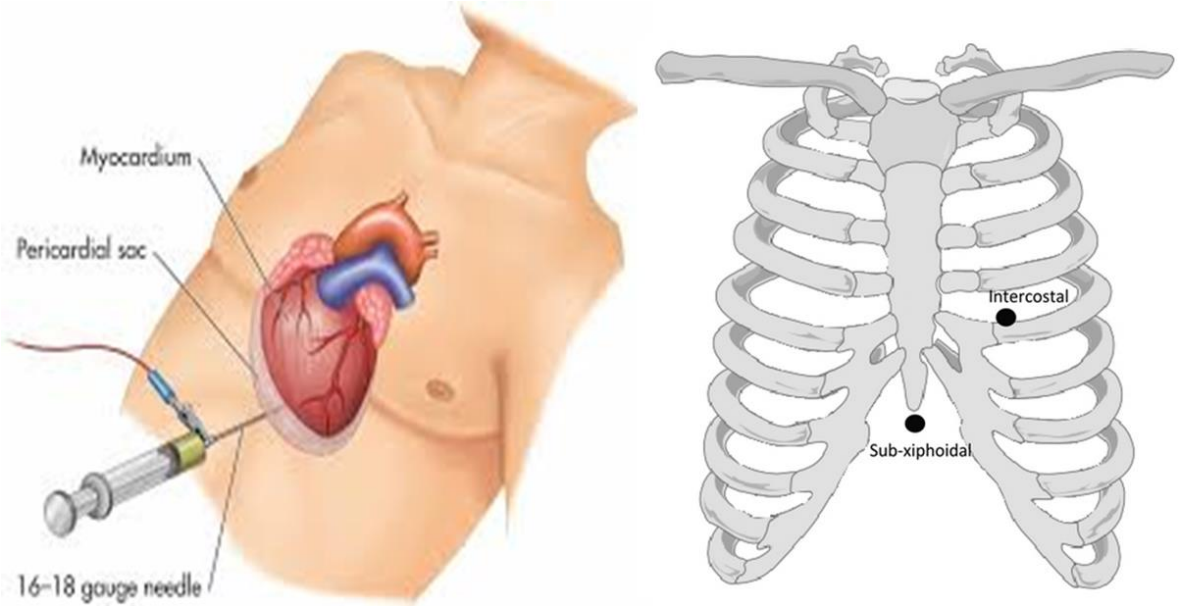
A follow-up echocardiogram and chest radiograph should be performed at a monthly follow-up examination to check for recurrent fluid accumulation.

**Pericardiocentesis and Pericardiotomy**

Removal of pericardial fluid is the definitive therapy for tamponade and can be done using the following 3 methods.

## I. Emergency subxiphoid percutaneous drainage

This is a life-saving bedside procedure. The subxiphoid approach is extrapleural; hence, it is the safest for blind pericardiocentesis. A 16- or 18-gauge needle is inserted at an angle of 30-45° to the skin, near the left xiphocostal angle, aiming towards the left shoulder. When performed emergently, this procedure is associated with a reported mortality rate of approximately 4% and a complication rate of 17%.




Sites for pericardiocentesis. **A**, Standard sub-xiphoidal approach. **B**, Intercostal approach used with ultrasound guidance in this case.

Echocardiographically guided pericardiocentesis Pericardiocentesis with echocardiography guidance was feasible in 96% of loculated pericardial effusions after cardiac surgery. A rescue pericardiocentesis guided by echocardiography, successfully relieved tamponade after cardiac perforation in 99% of 88 patients, and was the definitive therapy in 82% of the cases. The most serious complications of pericardiocentesis are laceration and perforation of the myocardium and the coronary vessels. In addition, patients can experience air embolism, pneumothorax, arrhythmias (usually vasovagal bradycardia), and puncture of the peritoneal cavity or abdominal viscera. Internal mammary artery fistulas, acute pulmonary o edema, and purulent pericarditis were rarely reported. The safety was improved with echocardiographic or fluoroscopic guidance. Recent large echocardiographic series reported an incidence of major complications of 1.3–1.6%. In a large series of fluoroscopy-guided percutaneous pericardiocenteses cardiac perforations occurred in 0.9%, serious arrhythmias in 0.6%, arterial bleeding in 1.1%, pneumothorax in 0.6%, infection in 0.3%, and a major vagal reaction in 0.3%. Incidence of major complications was further

significantly reduced by utilizing the epicardial halo phenomenon for fluoroscopic guidance.

## II. Percutaneous balloon pericardiotomy

This can be performed using an approach similar to that for echo-guided pericardiocentesis, with the balloon being used to create a pericardial window.

## III. Surgical Care in Hemodynamically Unstable Patients

For a hemodynamically unstable patient or one with recurrent tamponade, provide care as described below.

### Surgical creation of a pericardial window

This involves the surgical opening of a communication between the pericardial space and the intrapleural space. This is usually a subxiphoidian approach, with resection of the xiphoid. However, a left paraxiphoidian approach with preservation of the xiphoid has been described (**Motas 2010**).

Open thoracotomy and/or pericardiotomy (**Rylski 2010**) may be required in some cases, and these should be performed by an experienced surgeon.

### Recurrent cardiac tamponade or pericardial effusion

#### *Sclerosing the pericardium*

This is a therapeutic option for patients with recurrent pericardial effusion or tamponade. Through the intrapericardial catheter, corticosteroids, tetracycline, or antineoplastic drugs (eg, anthracyclines, bleomycin) can be instilled into the pericardial space.

#### *Pericardio-peritoneal shunt*

In some patients with malignant recurrent pericardial effusions, the creation of a pericardio-peritoneal shunt helps to prevent recurrent tamponade.

#### *Pericardiectomy*

Resection of the pericardium (pericardiectomy) through a median sternotomy or left thoracotomy is rarely required to prevent recurrent pericardial effusion and tamponade.

### Video-Assisted Thorascopic Procedure

In a study of 15 patients with cardiac tamponade, Monaco et al found that a modified, video-assisted thoracoscopic procedure seemed to be a feasible treatment for the condition (**Monaco 2009**).

Using a right hemithoracic approach, the investigators employed a 15mm trocar on the fourth right intercostal space on the anterior axillary and a 10mm trocar on the seventh right intercostal space on the median axillary line.

Utilization of a 5mm optic allowed 2 instruments, for the optic and for the endoscopic forceps, to be employed simultaneously using 1 trocar; this left the second trocar available

for dissecting scissors. All patients underwent a pericardial resection equal to that achievable via an anterolateral thoracotomy.

The pericardial effusion was effectively drained in all patients, with no intraoperative mortality or perioperative morbidity encountered.

The most common causes of cardiac tamponade is idiopathic and cancer. Echo-guided pericardiocentesis is safe, and the rate of complications has remained stable despite the fact that a greater number of procedures have been performed on an emergency basis. Surgery might be preferred in purulent, recurrent, and/or malign effusions and if pericardial biopsy is required for diagnosis. In addition, immediate surgical approach should be performed for traumatic hemopericardium, otherwise, less invasive procedure echo-guided pericardiocentesis might be the first choice especially in idiopathic cases and in patients with hemodynamic instability.

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