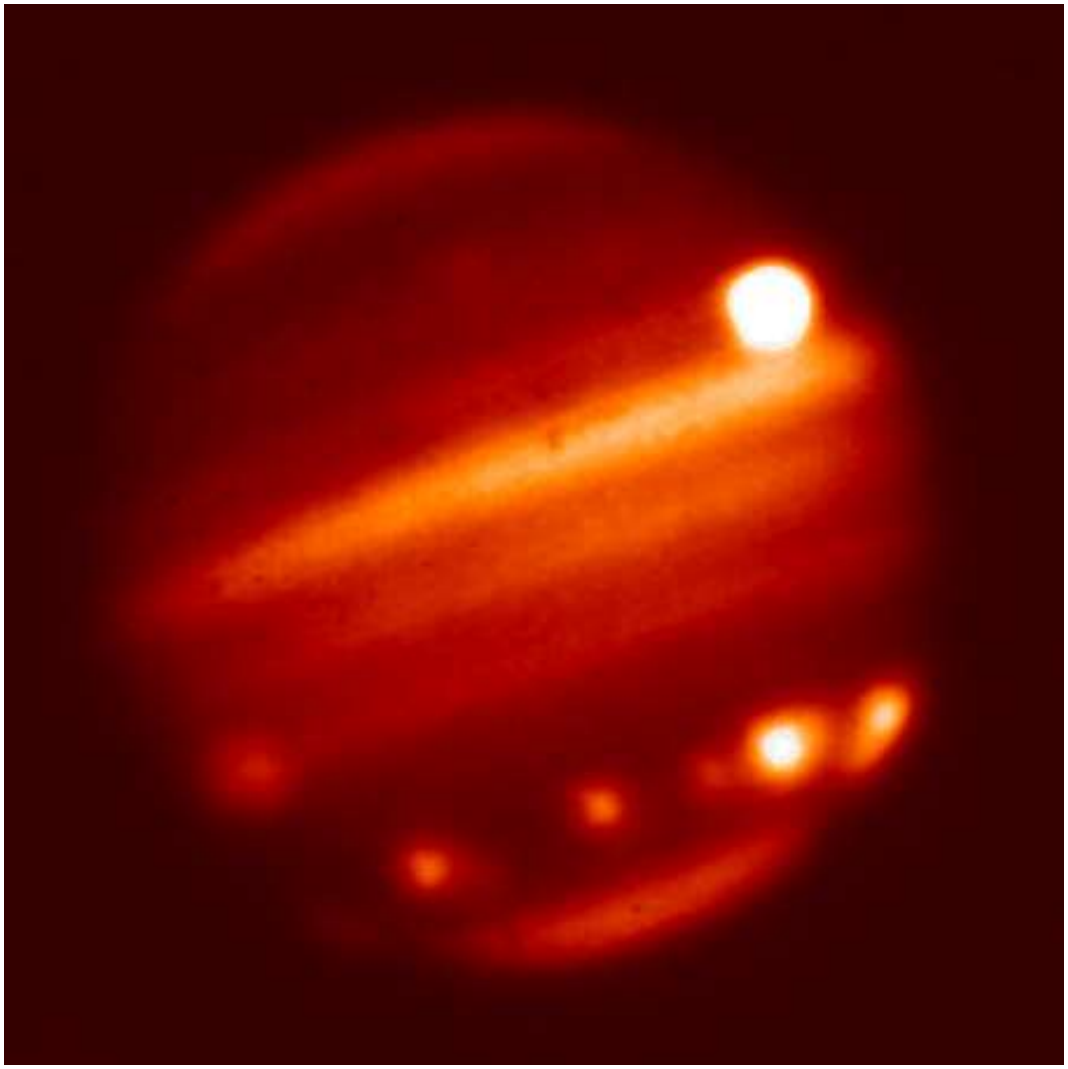
THE NORTHERN HOSPITAL

CARDIAC TAMPONADE (NON TRAUMATIC)

"Noah's Ark", oil on canvas, c.1620, Roelant Savery of Utrecht.

"…all the fountains of the great deep burst open, and the floodgates of the sky were opened. And the rain fell upon the land for forty days and forty nights…and living things that stirred on earth perished; birds, cattle, wild animals, all the creatures swarming over the Earth…and all human beings"

Genesis 7: 11-21



Above is an infrared image of Jupiter taken by the NASA Infrared Telescope Facility at Mauna Kea, Hawaii, at 08:54 on July 21, 1994. It shows the dramatic moment of impact of a number of fragments of the comet Shoemaker-Levy 9 with the planet Jupiter. Io, the closest of the Jovian moons, can be seen crossing the planet at the top right. The Great Red Spot is visible in the lower left of the planet. At the collision latitudes, the impact due to Fragment Q is just setting on right side. Just to the left of it, the R Fragment impact site shows up very brightly. Another four impact sites form a chain of flashes behind the R fragment.

One of the great scientific intellectual leaps of the Nineteenth century in the field of geology was the concept of "gradualism" which essentially argued that currently observable modern forces operating at the same rates that where presently observed was sufficient to explain the full spectrum of geological history. Entire mountains, even continents were shaped over untold eons of time to give the results that were observed around us. The greatest exponent of this world view, Charles Lyell, the father of modern geology, allocated the far greater part of the structure of the observable Earth to the effects of gradualism. Lyell postulated that the world could be hundreds of thousands, even millions of years old. In an age where the majority of the people firmly believed by religious dogma that it was no more than 6 thousand years old, Lyell's idea was ground shakingly radical, even many believed "heretical". Rapidly increasing knowledge of geological processes all pointed to the validity

of Lyell's revolutionary idea. It is difficult not to underestimate the enormous contribution this new theory had on the impact of Western science. It had a major influence along with the mathematical concepts of supply and demand espoused by Thomas Malthus, on the development of an even more momentous idea by an obscure English amateur naturalist named Charles Darwin. Darwin's even more radical ideas on evolution (rocks were one thing, human beings quite another) were founded to a large degree on the new concepts of gradualism.

Opposing these new world views were the traditional views of "catastrophism". This doctrine argued that most geological change occurred in rare episodes of truly global paroxysm, such as earthquakes and volcanos and in particular floods of unimaginable scale. The theory of catastrophism had powerful allies in the Church of England whose literal interpretation of the Bible pointed to the story of Noah and the flood in the book of Genesis as the undeniable truth of the place of catastrophism as the dominant force of the shaping of the world. Throughout the early Nineteenth century the battle thus raged between the new scientific gradualism and the catastrophism backed by the established religious order of the day. Not only the religious establishment however were convinced of the role of catastrophism. The great French naturalist Baron Cuvier was a firm believer in catastrophism. Though not a believer in the biblical flood he nonetheless wrote that "…the surface of the globe has been overturned by successive revolutions and diverse catastrophes"

By the mid Nineteenth century the gradualists had won some ground with many catastrophists conceding at least that the world must be far older than anyone had imagined, though they would still hold that the major part of the world had nonetheless been shaped by periodic cataclysmic events such as the biblical flood. Charles Lyell would ultimately argue that if global catastrophes forge most of geological history, how could a workable science of geology ever be achieved, for humanity had not ever witnessed such an event, (biblical flood aside) and so the whole argument was moot. With his theory of gradualism the present known and observable forces of nature were the key to the past and the Earth's entire history could thus be opened up to scientific enquiry.

There the argument stayed for over a century until two momentous breakthroughs came in the late 20th century that again shook the foundation of conventional thinking. The apparently clear cut boundaries between the arguments for catastrophism and for gradualism were suddenly blurred by these breakthroughs, the first occurring in 1980 and the second in the most stunning imaginable way in 1994. In 1980 Walter Alvarez put forward the extremely controversial theory that the reign of the dinosaurs was ended as the result of a cataclysmic impact of a Mount Everest sized comet that hit the Earth 65 million years ago. The energy of the resulting explosion would have far exceeded the simultaneous detonation of the world's entire nuclear arsenal at the height of the "cold war". Alvarez had accumulated very strong geological evidence for his theory, but many remained sceptical until another discovery was made in 1990. This was the recognition of a giant and ancient impact crater situated on the coast and adjacent seabed of the Yucatan peninsular of Mexico. Exacting scientific studies were made of the crater, the comet or asteroid that would have caused it was estimated to be about the size of Mount Everest and its date of impact…65 million years ago! Since 1990 further evidence from around the entire globe, in the form of the iridium rich layer within the geological strata that marks the end of the Cretaceous period, has turned Alvarez's theory into an almost overwhelming certainty. The second discovery was made by one of the greatest geologists and astronomers of the 20th century,

Eugene Shoemaker. He was a renowned comet hunter. He along with his colleague David Levy, discovered the comet Shoemaker-Levy 9 on 24th March 1993. By computer modelling he was able to predict that the comet was on a direct collision course for the planet Jupiter. Cosmic impacts of comets had long been assumed to occur, however was this thought unlikely to ever be directly witnessed by humans because of the relative rarity of the event when compared to the scale of a human life span, in fact even when compared to the span of the whole of recorded human history. Every astronomical device on planet Earth and in space prepared for the event, which happed exactly as predicted on 16th July 1994.

The Alvarez discovery has forced a rethink on the role of admittedly rare, but nonetheless stupendous cosmic events that can permanently change the face of the Earth in the literal blink of an eye. The stunning direct witness by humans of just such an event has resulted in a sobering reassessment of our tenuous place in the universe. If the Shoemaker-Levy comet had hit the Earth, this would have been a life extinguishing event for the planet. An event worthy of the biblical flood of Genesis. Both Lyell and Darwin would have had great cause to reflect on this fact. Lyell would have had his "witness of catastrophe". The Baron Cuvier would have marvelled at the cosmic event. So as in most things the truth of the matter lies somewhere between two extremes, whilst it is true that much of the Earth's shaping is due to gradualist processes nonetheless sudden catastrophe has indeed played a very real and significant role.

The signs and symptoms of cardiac tamponade are classically described in all medical texts, however confusingly these signs and symptoms are often not seen. We must recall the lessons of the gradualist and catastrophic debate and realise that both processes can shape the condition and therefore one set of clinical features does not necessarily apply in all cases. In the gradualist scenario of the slow accumulation of pericardial fluid due to the processes of chronic pericarditis of whatever cause the classical features are likely to be seen, yet this will only tell part of the story. The rest may be told by the sudden catastrophic scenarios of events such as penetrating traumatic impact or the sudden "bursting open of the floodgates" of a myocardial rupture where these signs may be far less obvious.

CARDIAC TAMPONADE (NON TRAUMATIC)

Introduction

Cardiac tamponade is an accumulation of intra-pericardial fluid which eventually leads to reduced cardiac chamber filling which in turn leads to a diminishing cardiac output.

Left unchecked this process leads to clinical shock and eventually pulseless electrical activity and death.

The early stages of this process can be detected by echocardiography, before clinical signs can be readily seen. [1]

The latter stages of the process become obvious on clinical signs with a picture of shock.

The best terminology is probably **compensated** (**or echocardiographic**) and **decompensated (or clinical)** cardiac tamponade. [2]

The method of drainage will depend on the etiology, how unstable the patient is, as well as the local resources and expertise that is available.

See also separate document on Sonography of Cardiac Tamponade and Pericardiocentesis (in Radiology folder).

Pathophysiology

Pericardial effusion is the accumulation of fluid (exudate, transudate, blood or chylus) within the pericardial cavity.

Normally this cavity contains up to 35mL of fluid. More than this can be accommodated in the short term, up to about 200 mls. In the longer term, if fluid accumulates more slowly, up to 2 liters can be accommodated with little clinical consequence. However, above these values the process of cardiac tamponade will occur, with lethal consequences if unrecognized.

Causes:

The best classification is in terms of non-traumatic (these guidelines) and traumatic as not only the cause, but the clinical course and approach to treatment are different.

Causes of acute onset non-traumatic cardiac tamponade:

1. Acute myocardial infarction with free wall rupture.
2. Stanford A type aortic thoracic dissection.
3. Coagulation disorders.

Causes of gradual onset non-traumatic cardiac tamponade:

1. This can be due to any of the recognized causes of pericarditis.

Causes of traumatic cardiac tamponade:

2. These can be penetrating or blunt. In both cases the onset can be acute or delayed.

Clinical Features

The signs and symptoms of cardiac tamponade are variable and non-specific, and a high index of suspicion must therefore be maintained for this condition.

Important points of history:

1. The commonest symptom is dyspnea.

Further symptoms generally relate to:

2. Those due to depressed cardiac output.

3. Those due to the underlying disease process.

Important points of examination:

In the early stages of the process, there may be very little in the way of examination findings.

1. Tachycardia

2. Tachypnea

3. Beck's triad:

    - Hypotension (late, initially with compensatory mechanisms may even have hypertension).

    - Diminished heart sounds - this however is a very insensitive (may be seen in obesity or COPD) and subjective sign.

    - Elevated JVP (however in cases of co-existing dimished intra-vascular volume, the JVP may not be elevated even in the presence of a significant tamponade).

4. Pulsus paradoxus, but this sign is difficult to appreciate in hypotensive patients. It is more readily appreciated on invasive arterial pressure monitoring.

5. There may be a degree of peripheral cyanosis (typically of the head, neck, chest, upper limbs).

Associated findings may also include:

6. Relatively **clear** lung fields, (in association with cardiomegaly)

7. Pleural effusions (in subacute / chronic settings)

8. Signs of the causative pathology (eg. fever, pericardial rub).

Differential Diagnosis

These include:

1. Massive pulmonary embolism

2. Tension pneumothorax

3. Superior vena cava obstruction

4. Chronic constrictive pericarditis

5. Air embolism

6. Right ventricular infarct

7. Severe congestive cardiac failure/cardiogenic shock

8. Extra-pericardial compression e.g.due to haematoma or tumor

Investigations

It should be noted that cardiac tamponade is most often diagnosed when the clinical suspicion is high, ie the diagnosis is thought of and "chased", rather than being determined on clinical signs. Alternatively it may be suspected as an incidental finding on imaging such as CT scan.

CXR

The cardiac silhouette may be normal in cases of acute tamponade, (at least 250 mls of intra-pericardial fluid is needed before an increase in cardiac silhouette can be appreciated).

In subacute or chronic cases cardiomegaly is seen:

Classically the cardiac silhouette is described as "globular" or "waterbottle" in shape. This however is a very non-specific (and very subjective) finding and is consequently of little practical use.

The best indicators of *possible* effusion include :

- Cardiomegaly, when there is a relatively acute increase in size

See appendix 2 below.

- The larger the silhouette the greater should be the index of suspicion
- The presence of *clear lung fields* is also suggestive, of pericardial effusion.

A CXR will also help rule out other diagnoses and may give clues to the causative pathology e.g. lung malignancy.

ECG

Findings are non-specific but may include:

- Small voltages are suggestive of large effusion (but not necessarily tamponade).
- Electrical alternans (especially total) again reflects a large effusion. This may be more specifically associated with tamponade (but evidence for this is tenuous).
- Other changes include sinus tachycardia, non-specific ST changes or the changes of pericarditis, although these do not of themselves indicate tamponade.

FAST Scan

Bedside FAST Scan **ultrasound** is an excellent screening test if the condition is suspected.

Even though tamponade cannot be proven, it may pick up pericardial fluid (especially in the larger subacute / chronic effusions) and thus provide strong supportive evidence

Echocardiography

Note that acute cardiac tamponade may have little in the way of clinical signs initially and that CXR and ECG will not be helpful. Ultimately if there is a clinical suspicion of cardiac tamponade then imaging with an echocardiogram must be done.

TTE

Transthoracic echocardiography is the current gold standard investigation as:

- It is the most sensitive method of detecting pericardial fluid.
- It is the most specific investigation.
- It is non invasive and can be done at the bedside.
- It can also assess cardiac function and degree of compromise.
- **It can detect cardiac tamponade, before clinical signs become apparent.**

TOE

- If TTE is inconclusive then a TOE (trans-esophageal echocardiogram) may be done. This can also detect occult loculated effusions.

- It can help rule out some of the differential diagnoses e.g. large PE.

- It can look for causative pathology e.g. proximal aortic dissections and myocardial wall ruptures.

CT Scan/ CT Angiogram:

Detects fluid readily but is less reliable than an echocardiogram in determining tamponade.

Note that this can be an extremely useful imaging modality when echocardiography is not readily available and cardiac tamponade is suspected. Pericardial fluid is readily detected, but early tamponade cannot be reliably detected.

It also has the added advantage of imaging the chest for possible causative pathology, such as malignant masses.

CT angiogram should be done when aortic dissection with complicating pericardial bleeding is suspected. This is an important diagnosis to make, as initial attempts at pericardial drainage may be detrimental in this setting, and where definitive treatment of the dissection itself is required.

MRI

Again this will readily detect fluid but not tamponade.

Both CT and MRI are problematic (and not as good as an echocardiogram) in the critically ill patient.

Hemodynamic Monitoring

In the ICU setting, monitoring with a pulmonary artery catheter can provide clues to a tamponade.

- The classical finding is elevation of pressures to around 25 mmHg and equalization of right atrial, right ventricular end diastolic, pulmonary artery diastolic and pulmonary artery wedge pressures.

- Pre-terminally, however, right sided pressures may be greatly elevated whilst left sided pressures become greatly depressed.

Management

Needle pericardiocentesis is best reserved as a procedure of last resort in the setting of the ED.

The preferred methods are ultrasound guided drainage performed in the cardiac catheter laboratory, or more ideally subxiphoid pericardiotomy in the operating theater if the clinical situation allows for this.

In practice, available resources, and expertise, urgency of the clinical situation as well as the exact etiology will determine which procedure is done. In cases of myocardial rupture and aortic dissection, thoracotomy with drainage and repair is the definitive management, rather than pericardiocentesis which can only be a temporizing measure, and may in some cases be harmful.

Modalities include:

1. Fluid therapy:

    - This is only a temporizing measure at best.

2. Inotropic support:

    - Again this will be a temporizing measure only, and is not usually effective.

3. Intubation and ventilation:

    - Although these may be unavoidable in the critically ill/ arrested patient institution of mechanical ventilation may cause a sudden drop in blood pressure as the positive intrathoracic pressure further impairs cardiac filling.

4. Drainage procedures:

    Sub-xiphoid peri-cardiotomy:

    - Sub-xiphoid peri-cardiotomy is used for purulent or recurrent effusions or when tissue is required for diagnostic purposes.

      This may be done under local anesthetic in theater.

      A drainage catheter is placed under direct vision following careful dissection into the pericardial space from the sub-xiphoid region.

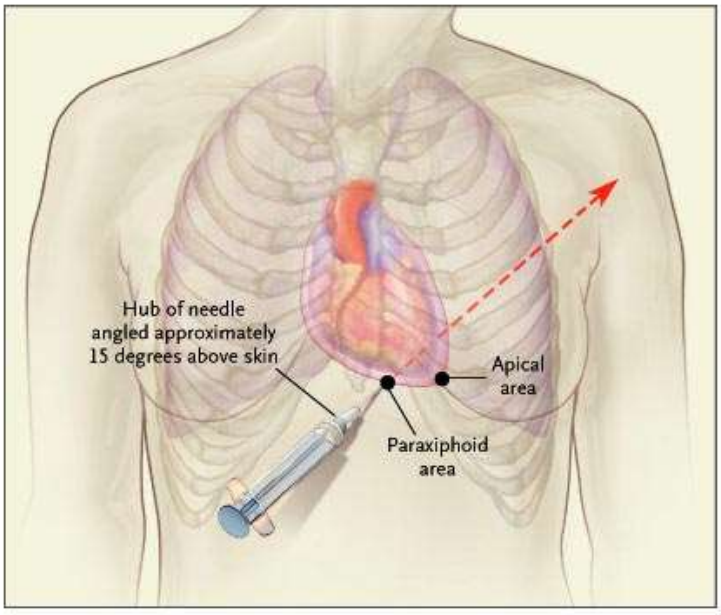
    Needle peri-cardiocentesis:

    - This is the best done in the cardiac catheter laboratory under fluoroscopic guidance.

    - In more urgent situations it may be done under ultrasound guidance in the ED

      See also separate document on Sonography of Cardiac Tamponade and Pericardiocentesis (in Radiology folder).

- Blind needle peri-cardiocentesis is best considered as a method of last resort in arrest or pre-arrest situations, when ultrasound is not available.



(from NEJM August 14 2003)

A central line needle (or similar) is inserted para-xiphisternally i.e. between the xiphoid process and the left costal margin at an angle of 15 degrees and aimed toward the left shoulder. The needle is then advanced slowly, with regular aspirations.

The patient should be on cardiac monitoring whilst the procedure is done. Attempts at attaching a lead to the needle are not necessary and may provide misleading results.

Once fluid has been successfully drained a seldinger wire may be introduced into the pericardial space followed by a central line or "pigtail" catheter. This can remain in situ, should further aspirations be necessary.

5. Thoracotomy:

- Thoracotomy rather than attempts at needle drainage will be necessary when definitive surgical repair of the causative pathology is necessary; eg. myocardial free wall rupture, proximal aortic dissections, and cases of trauma.

6. Treatment of the underlying condition:

- Note that in the life-threatening case of **bacterial pericarditis** causing a pericardial effusion, antibiotics are critical as is surgical drainage, independent of whether or not the patient has signs of cardiac tamponade.

- IV hydrocortisone will also be a useful *adjunct* in cases of auto-immune induced pericardial effusions.

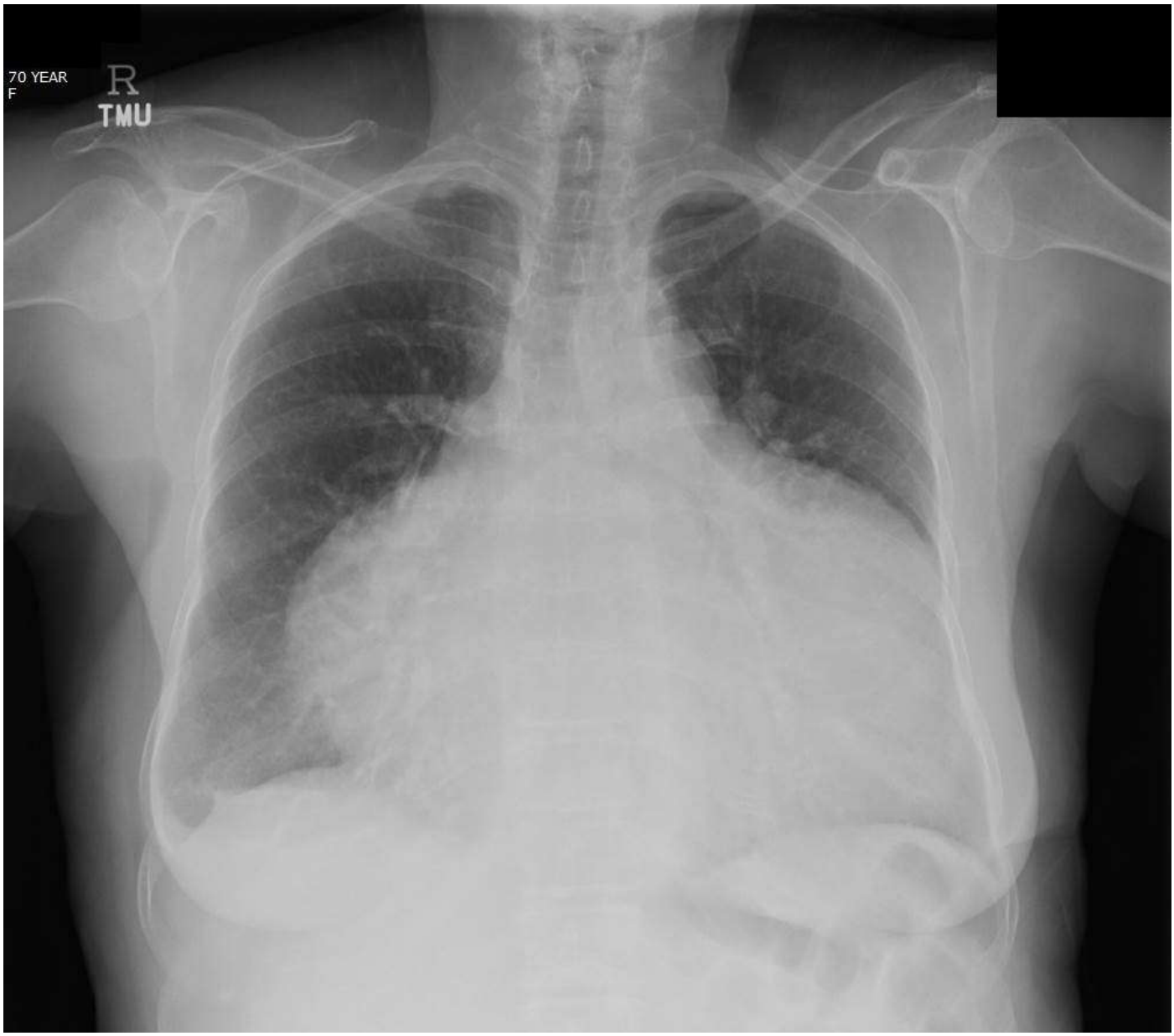
- Dialysis will be required in cases of uremic renal failure.

7. Note that CPR is relatively **ineffective** in cases of arrest due to cardiac tamponade.

Disposition

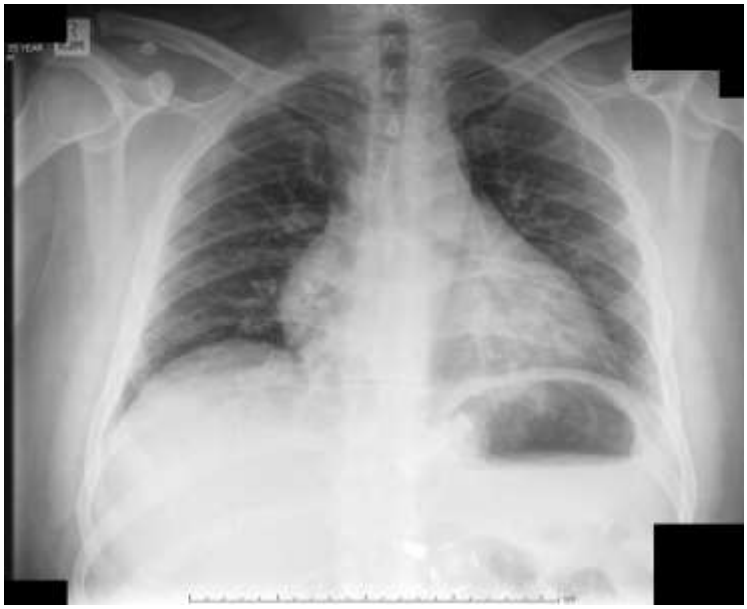
All cases will need HDU/ICU admission.

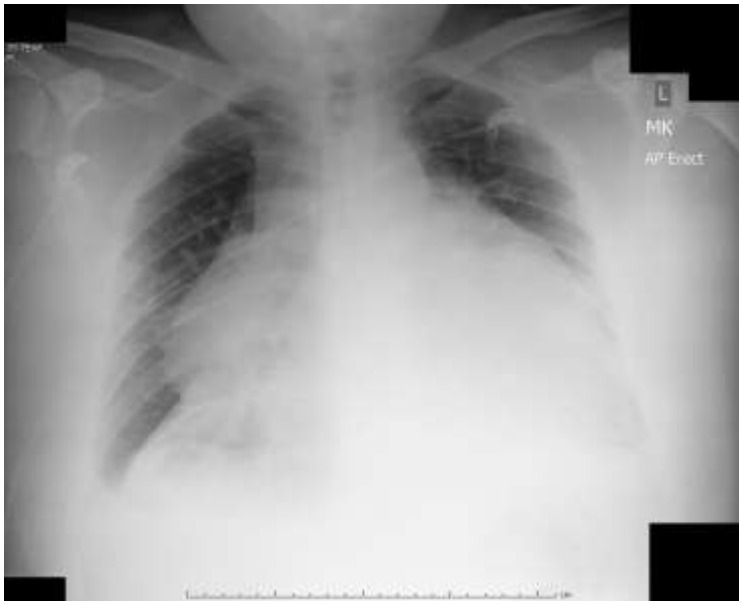
Appendix 1



Classic "water bottle" appearance of the cardiac silhouette with a very large (chronic) pericardial effusion in a 70 year old female. Effusion was confirmed on bedside ultrasound examination. This also demonstrated the early signs of echocardiographic cardiac tamponade. The patient was taken to theater and underwent an urgent pericardial window drainage.(Radiograph, courtesy Dr Nikki Velasco).

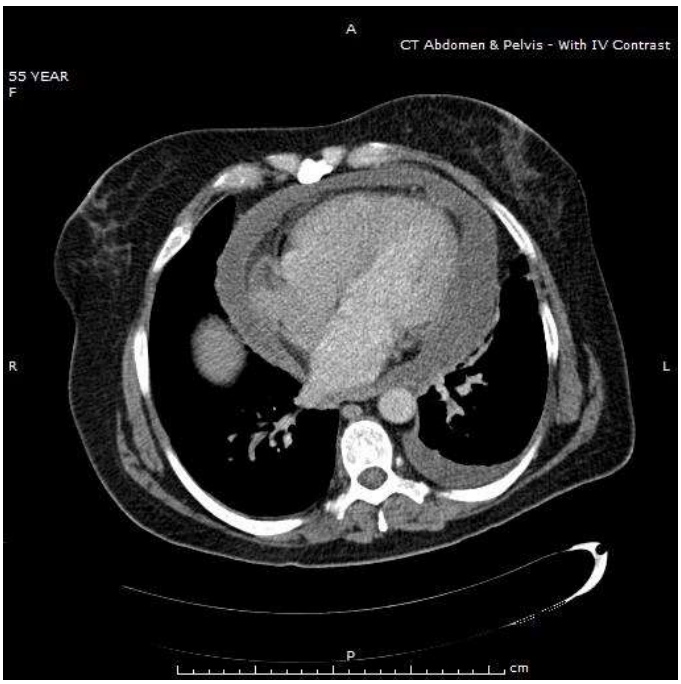
[Appendix 2: *Case Report*]



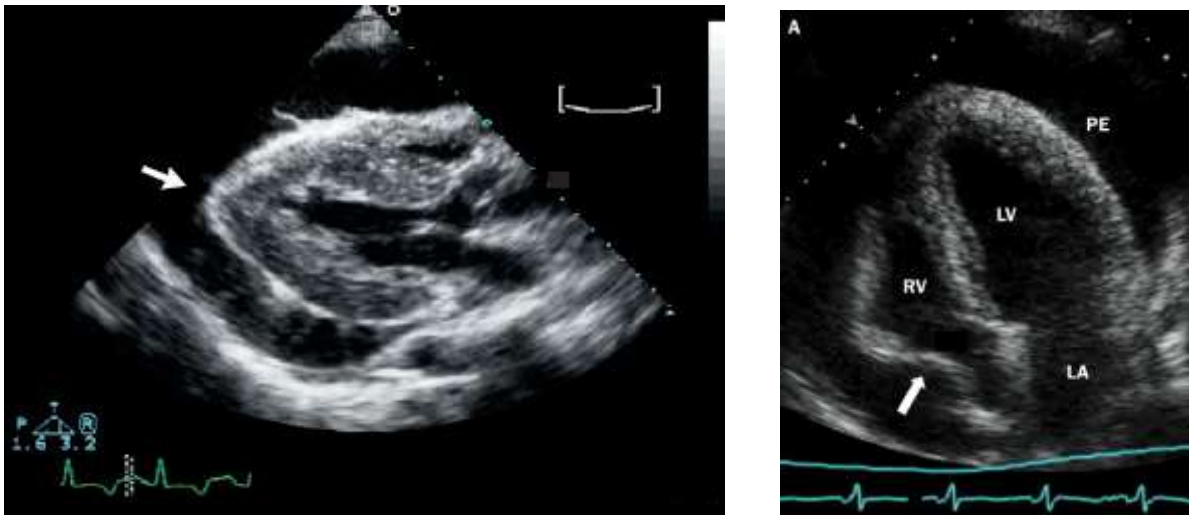


CXR 1: (11/6/2011), CXR 2: (23/6/2011)

Above: A 35 year old male with a history of shortness of breath presented on two occasions. On the first occasion his CXR perhaps shows some mild cardiomegaly, (in retrospect). He re-presented 12 days later with increasing symptoms. There has been a subacute and dramatic increase in the cardiac silhouette. There is also a (characteristic) associated suggestion of pleural effusion on the left. The lung fields on both occasion appear relatively clear. This scenario is strongly suggestive (but not diagnostic) of subacute pericardial effusion. Echocardiography confirmed the presence of a large pericardial effusion, as well as echocardiographic evidence of cardiac tamponade. 2.6 liters of blood stained fluid were subsequently aspirated from the pericardial space.



Left: CT scan of the chest of a 55 year old woman with lymphoma, who had developed a pericardial effusion. The effusion can be seen surrounding the heart within the pericardial space. There is also a small left pleural effusion.



Left panel shows typical echocardiographic appearance of a large pericardial effusion, (arrowed).

Right panel: PE=pericardial effusion, LV=left ventricle, RV=right ventricle, LA=left atrium, IVS=interventricular septum, IVC=inferior vena cava. An apical four-chamber view of LV, LA, and RV that shows large pericardial effusion together with right-atrial diastolic collapse, an early sign of cardiac tamponade, (arrowed).

References:

1. Roy CL et al. Does This Patient With a Pericardial Effusion Have Cardiac Tamponade? JAMA, April 25, 2007, Vol 297, No. 16, p. 1812- 1818

2. Spodick DH: Acute Cardiac Tamponade. NEJM 349:7, August 14 2003 p.684- 690.

3. Harney J, Kelly A.M, Hayes J. Pericarditis, Myocarditis and Cardiac Tamponade in Textbook of Adult Emergency Medicine: Cameron et al 4th ed 2015.

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Reviewed 23 May 2018.