Acute Cor Pulmonale

Sunday, 02 January 2005

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Definition

Acute cor pulmonale (ACP) can be defined as a clinical situation in which the right ventricle (RV) is suddenly subjected to an excessive afterload. ACP is essentially seen during massive pulmonary embolism (PE), or in the setting of acute respiratory distress syndrome (ARDS). In these two situations, the right ventricular outflow impedance is suddenly increased, which reduces the ejection volume, producing right ventricular dilatation by augmentation of the end-systolic volume. Thus, ACP combines systolic and diastolic overload of the RV. This is taken into account in the echocardiographic definition, which combines paradoxical septal motion (linked to systolic overload) and right ventricular dilatation (linked to diastolic overload) (1). These two signs are always accompanied by abnormal left ventricular relaxation, revealed by the Doppler pattern of mitral flow (1).

Reminder : ventricular independance

A brief reminder of this physiological phenomenon is necessary for a full understanding of the echocardiographic anomalies observed in ACP.

Normally, the right and left ventricles contract at the same time, during systole. When right ventricular ejection is hindered, as in ACP, right ventricular contraction is prolonged, whereas contraction of the left ventricle (LV) has already started its diastolic phase. The persistent pressure of the RV then reverses the transseptal pressure gradient, the pressure acting on the right ventricular face exceeding that on the left ventricular face. This pushes the septum to the left, as seen in ACP at the start of diastole (fig. 1).

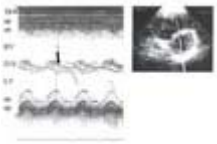


Figure 1: Paradoxical septal motion of acute cor pulmonale. Guided by the two-dimensional image, a short-axis parasternal approach is used for M-mode recording of the different structures crossed by the ultrasound: ThW: anterior thoracic wall, ep: epicardium, en: endocardium, RV: right ventricular chamber, IVS: interventricular septum, LV: left ventricular chamber. The thin vertical arrows indicate the end of ventricular contraction and the delayed end of right ventricular contraction on the left, inducing protodiastolic septal displacement, indicated by the thick black arrow. The septum remains displaced throughout diastole, and is again pushed towards the right ventricular chamber on the next left ventricular contraction (thick white arrow). Theoretical normal septal movement is shown in dotted lines to highlight the paradoxical motion.

This septal flattening persists throughout diastole, since the right ventricular filling pressure is greater, because of diastolic overload (fig. 1). But at the beginning of systole, the transseptal pressure gradient is again reversed, and the septum is pushed towards the right ventricular chamber (fig. 1). This results in the "paradoxical" movement of the interventricular septum.

Another important physiological feature to take into account is the rigidity of the pericardium surrounding the two ventricles. Any right ventricular dilatation occurs at the expense of the left ventricle, which is compressed (fig. 2).

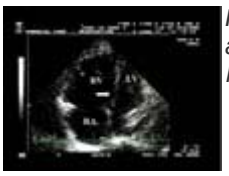


Figure 2: Major right ventricular dilatation during massive pulmonary embolism. The right atrium (RA) and the right ventricle (RV) are very dilated on this apical four-chamber view. The left ventricle (LV) appears compressed by septal displacement (arrow).

A final physiological parameter to recall is that right ventricular size varies with the quality of its filling: hypovolemia can markedly reduce the dimensions of the right ventricular chamber, and this disorder must be corrected before the echocardiographic examination if the results are to be interpreted correctly. Insufficient venous return affecting right ventricular size can be detected by ultrasound examination of the vena cavae (2, 3, 4). In particular, in a ventilated patient, partial or complete collapse of the superior vena cava on mechanical insufflation indicates hypovolemia (FILM 1, FILM 2).

Principal echocardiographic views used to study right ventricular function and detect ACP

Echocardiographic examination of the RV requires a long-axis view to measure chamber size, and a small-axis

view to evaluate the shape and movements of the interventricular septum. To these should be added a view of the right ventricular outflow tract used to assess the Doppler velocity and morphology of pulmonary artery flow, and a Doppler recording of tricuspid flow to detect tricuspid regurgitation and so measure the transtricuspid gradient and deduce from it the pulmonary artery systolicpressure (fig. 3).

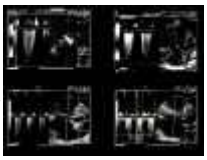


Figure 3 : Four recordings of tricuspid regurgitant flow. Doppler measurement of the peak velocity (V, arrow) is used to calculate the transtricuspid pressure gradient, which is equal to 4V 2 . If we know the right atrial pressure, or the central venous pressure (CVP), it can be added to this gradient to give the right ventricular systolic pressure. If the exact CVP value is not known, it can be estimated from the measurement of the diameter of the inferior vena cava (diam) at the end of expiration (CVP= 0.64diam + 0.77).

Lastly, it is necessary to examine mitral flow velocity to identify any abnormal left ventricular relaxation (fig. 4).

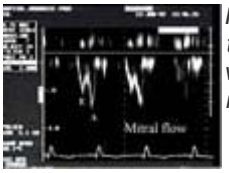


Figure 4 : Abnormal left ventricular relaxation. Abnormal septal motion and compression of the left ventricular chamber caused by right ventricular dilatation are seen in impaired left ventricular filling, which is characteristic of abnormal relaxation: the E-wave, of rapid filling, is reduced, whereas the A-wave linked to atrial systole, is preeminent.

All these effects and the respective views used to examine them are detailed in the section on the principal echocardiographic views.

When a patient is breathing spontaneously, as is usually the case in pulmonary embolism, the echocardiographic examination is done by the transthoracic route (transthoracic echocardiography, or TTE). In a patient on assisted ventilation, transesophageal echocardiography (TEE) is easy and should therefore be used as it gives better images. This is how ARDS patients are examined.

Echocardiography is above all a qualitative procedure. However, quantitative measurements can also be made during or after acquisition of sequences. Table 1 gives the principal normal values of our laboratory.

Systolic overload detected by echocardiography

As explained above, systolic overload prolongs right ventricular systole, while the ejection phase of this ventricle is often reduced (table 2). This result in paradoxical septal motion linked to the specific chronology of the pathological variations in transseptal pressure gradient. This gradient (left ventricular pressure minus right ventricular pressure) is always positive in physiological situations. In ACP, it becomes negative at the end of systole/beginning of diastole, remains negative during diastole because of right ventricular diastolic overload, and again becomes positive at the start of systole.

These septal anomalies are clear in a small-axis view (Fig. 5, FILM 3, FILM 4) and can be analyzed in more detail in motion mode (M-mode) (fig. 1).

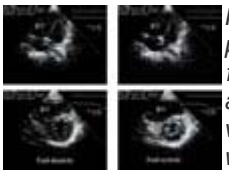


Figure 5 : Right ventricular deformation in acute cor pulmonale. In a young female patient presenting acute cor pulmonale complicating acute respiratory distress syndrome, the apical four-chamber view (above) shows right ventricular dilatation accompanied by a rounded appearance of the apex, at the end of both diastole and systole. The short-axis parasternal view (below) shows the loss of this crescent appearance of the right ventricular chamber, which becomes oval-shaped. The outcome is that the right ventricle resembles the left ventricle: a 180° rotation of the probe could lead to confusion in the long axis, as the right ventricle then appears on the right of the image, in the place of the left ventricle. In some countries (Russia, for example), the recording is presented backwards and so this confusion is possible. To avoid confusion remember that the tricuspid valve plane is always situated above the mitral valve plane.

A systolic overload that persists for more than a few hours also results in morphological changes in the right ventricular chamber:

First, the shape of the right ventricle changes. On the long axis, the apical region, which is normally triangular, becomes rounded. On the short axis, the right ventricle changes from a crescent to an oval shape. Together with dilatation, this deformation has the effect that the shape of the right ventricular chamber, which is normally very different from that of the left ventricular chamber, comes to resemble it somewhat (fig. 5).

Second, incipient hypertrophy of the free wall of the RV occurs, with accentuation of muscular trabeculae (fig. 6 and 7) and wall thickening (figs. 7 and 8). Values around 0.6 cm are common for the right ventricular free wall whose thickness normally does not exceed 0.3 cm. But parietal hypertrophy is never as marked as that seen in chronic cor pulmonale where values of about 1 cm are common.

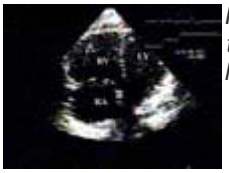
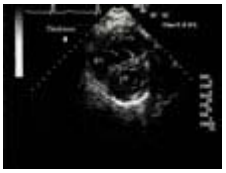


Figure 6 : Rapid right ventricular hypertrophy in acute cor pulmonale, first example. In this patient presenting acute cor pulmonale complicating acute respiratory distress syndrome, hypertrophic trabeculae are seen in the dilated right ventricular chamber (arrow).

Figure 7: Rapid right ventricular hypertrophy in acute cor pulmonale, second example. In this female patient presenting acute cor pulmonale complicating massive pulmonary embolism, hypertrophic trabeculae are seen in the dilated right ventricular chamber (arrows). Note also the thickness of the wall: 0.7 cm.



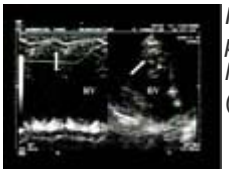


Figure 8: Rapid right ventricular hypertrophy in acute cor pulmonale, third example. In this patient presenting acute cor pulmonale complicating acute respiratory distress syndrome, hypertrophy of the right ventricular wall is clearly visible on a transgastric approach in M-mode (on left, arrow).

Severe systolic overload leads to a reduced ejection volume, which can be evaluated by the Doppler time-velocity integral of the pulmonary flow (table 2). A biphasic appearance indicates a large increase in resistance to pulmonary blood flow (fig. 9, FILM 5, FILM 6). The reduction in ejection volume is compensated for a while by tachycardia, but in the end leads to a drop in cardiac flow. The onset of ACP can therefore precipitate acute circulatory insufficiency.

Table 2

	Volunteers (n = 24)	ACP in ARDS (n =19)	ACP in PE (n = 18)
PA_{VTI} (cm)	18±3	11±4*	9±3*
Peak velocity (m/sec)	0.80±0.20	0.82±0.21	0.64±0.17*
ACT (msec)	125±23	76±27*	68±36*
ET (msec)	304±23	244±32*	252±32*
ACT/ET (%)	41±7	32±13*	25±8*

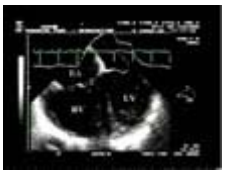
Figure 9: Biphasic appearance of pulmonary artery flow. The sudden rise in resistance to pulmonary blood flow alters the Doppler profile of pulmonary artery flow, which becomes biphasic. This anomaly is also seen in chronic pulmonary arterial hypertension.

Long-axis measurement of the right ventricular diastolic and systolic areas can be used to calculate the fractional reduction in right ventricular area. But this measurement, which is very useful when studying the quality of left ventricular systolic function, is, in our experience, of no value when studying the RV. This is because there is no fixed normal physiological value, and because pathological variations in this parameter can occur for a while in the same direction as variations in afterload.

Diastolic overload detected by echocardiography

Diastolic overload of the right ventricle dilates this chamber at the end of diastole. This dilatation is easy to observe but difficult to measure accurately. The particular shape of the right ventricle means that any volume calculation by echocardiography is impossible in practice. However, long-axis measurement of the right ventricular area is straightforward using an apical four-chamber view or the transesophageal route. It is therefore possible to establish the ratio of the end-diastolic areas of the two ventricles, which is normally less than or equal to 0.6. A ratio above 0.6 can therefore be considered as indicative of right ventricular dilatation. But this is not necessarily pathological and must be interpreted in the light of other echocardiographic signs, notably the presence or absence of a septal anomaly suggesting systolic overload. Likewise, a normal Doppler echocardiogram of mitral flow, when the RV seems slightly dilated, rules out a pathological cause. On the other hand, a E/A ratio equal to or lower than one indicates marked right ventricular dilatation and is always pathological (fig. 10, FILM 7, FILM 8).

Figure 10: Severe acute cor pulmonale in acute respiratory distress syndrome. This long-axis view by the transesophageal approach shows that the right ventricular (RV) area exceeds the left ventricular (LV) area. The right atrium (RA) is also very dilated.



The right ventricular dilatation observed during acute cor pulmonale is associated with right atrial dilatation (fig. 2, fig. 10), and enlargement of the inferior vena cava (fig. 11).

Figure 11: Dilatation of the inferior vena cava. A: in this female patient presenting acute right ventricular insufficiency, the inferior vena cava (IVC) and a hepatic vein (HV) are dilated. B: after injection of contrast agent in a vein of the superior caval network, the agent flows back into the IVC and IHV, indicating tricuspid regurgitation.

There is also tricuspid regurgitation which can be seen by contrast ultrasound (fig. 11), and which is utilized to measure pulmonary artery systolic pressure, using Doppler echocardiography (fig. 3). When right atrial pressure exceeds left atrial pressure, the foramen ovale, which had remained permeable, may reopen. This anomaly, which induces a right-left shunt, can be detected by contrast ultrasound (fig. 12) or color Doppler (fig. 13, FILM 9) (5, 6). It causes paradoxical arterial embolism in thromboembolic disease (7).

Figure 12: Contrast echocardiographic detection of patent foramen ovale (PFO). In this patient massive pulmonary embolism is complicated by acute cor pulmonale which is indicated by dilatation of the right atrium (RA) and right ventricle (RV), with reduced left ventricular (LV) size on an apical four-chamber view (A). Injection of contrast agent in a peripheral vein (B) opacifies the right chambers, but the contrast agent quickly passes into the left chambers, indicating patency of the foramen ovale.

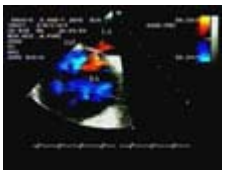


Figure 13:Color Doppler detection of patent foramen ovale (PFO). In this patient presenting acute cor pulmonale complicating massive pulmonary embolism, color Doppler examination of the interatrial septum (IAS) shows turbulent flow towards the probe (red), across the IAS and into the left atrium (LA). Note also the marked dilatation of the right atrium (RA).

Effects of acute cor pulmonale on the left ventricle

Sudden right ventricular dilatation within in an inextensible pericardium results in left ventricular compression, which is easily seen on echocardiographic examination (fig. 2, 5, 6, 7 and 10). Acute cor pulmonale therefore reduces left ventricular diastolic dimensions (tables 2 and 3) (8, 9, 10). In massive pulmonary embolism, this sudden drop in preload causes acute circulatory insufficiency (FILM 7). In ARDS, the decrease in left ventricular preload is usually more progressive, but may also contribute to circulatory insufficiency (FILM 10, FILM 11, FILM 12).

Left ventricular compression by right ventricular dilatation contributes more to the reduction in LV diastolic filling if it occurs when the pulmonary circulation is partly obstructed: proximal obstruction by a thrombus in massive PE, distal obstruction by the action of high alveolar pressure on pulmonary capillaries in ARDS managed by assisted ventilation (11).

In addition to the reduction in left ventricular diastolic dimensions, Doppler echocardiography reveals abnormal

relaxation which is seen in predominance of the A-wave over mitral flow (table 2 and 3) (fig. 4, FILM 13, FILM 14).

Table 3

	No ACP (n= 62)	Moderate ACP (n=13)	Severe ACP (n= 6)
RVEDA/LVEDA	0.54±0.12	0.81±0.10*	1.2±0.11*
HR Bat/mn	100±18	114±9	100±17
SI (Doppler) cm^3/m^2	32±9	25±8*	21±4*
CI (Doppler) $l/min/m^2$	3.1±0.9	2.8±0.8	2±0.8*
RVEDA cm^2/m^2	8.6±2.1	11.6±2.8*	15.2±3.5*
RVESA cm^2/m^2	5.6±1.9	7.6±2.2*	11.7±2.2*
LVEDV cm^3/m^2	61±16	45±8*	43±10*
LVESV cm^3/m^2	29±10	22±10*	20±8*
LVEF %	52±11	52±17	53±14
SPAP (Doppler) mmHg	28±11	51±3*	44±6*
E/A mitral	1.3±0.4	0.8±0.2	0.8±0.1
IVC diam mm	17±5	19±7	24±3

	No ACF (n= 32)	ACF without MA (n= 32)	ACF with MA (n =34)
RVEDA/LVEDA	1±0.2	1.2±0.3	1.4±0.5*
HR bat/mn	88±12	104±14*	109±18*
SI (Doppler) cm^3/m^2	31±8	22±8*	18±8*
CI (Doppler) $l/min/m^2$	2.7±0.7	2.2±0.7	1.9 ±0.9*
RVEDA cm^2/m^2	15.8±4.5	15.7±3.3	15.9±4
RVESA cm^2/m^2	11.7±3.8	11.7±3.1	12±3.3
LVEDV cm^3/m^2	54.3±11.3	41.3±15.2*	37.7±19.7*
LVESV cm^3/m^2	26.4±13.1	17.4±7.8	19.8±13.4
LVEF %	54±15	58±11	49±12
SPAP (Doppler) mmHg	55±18	48±15	48±16
E/A mitral	0.85±0.26	0.80±0.21	0.78±0.20
IVC diam mm	16±6	19±6	19±5

Table 4

Acute Cor Pulmonale complicating massive pulmonary embolism

The most frequent cause of acute cor pulmonale is obstruction of at least two lobar arteries by massive PE. Kasper was the first to underscore the value of echocardiography when assessing a patient suspected to have PE (12). He quantified right ventricular dilatation using the ratio of the ventricular diameters in M-mode. In view of the deformation of the right ventricle when it dilates, the area ratio we have proposed (1) is more reliable: a simple ratio of diameters ignores apical deformation.

Using our echocardiographic definition of ACP, which combines right ventricular dilatation and paradoxical septal motion, we noted ACP in 61% of 161 successive patients presenting massive PE. Diagnosis is usually made by TTE, since assisted ventilation is rarely used in a patient presenting massive PE. However, in certain emergency patients on assisted ventilation because of cardiorespiratory arrest, TEE can give an immediate diagnosis by detecting ACP, and even by visualizing the thrombus (13) (fig. 14, FILM 15, FILM 16, FILM 17, FILM 18). A thrombus in the right chamber is rarely visualized by TTE (FILM 19).

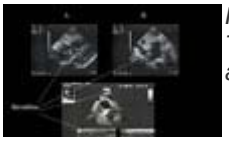


Figure 14: Visualization of a thrombus in the right chamber and pulmonary artery. Transthoracic echocardiography reveals a thrombus floating in the atrium right (A, B). In another patient (C), a floating thrombus is visualized in the right pulmonary artery.

Acute Cor Pulmonale during PE indicates a major obstruction but is not always accompanied by circulatory insufficiency, as defined by the need to use vasoactive drugs to maintain systolic blood pressure above 90 mmHg (10). Circulatory insufficiency is, however, frequent: we observed it in two thirds of patients with ACP complicating PE (10). When the patient does not develop metabolic acidosis, the prognosis of this circulatory insufficiency is excellent, with vasoactive support for a few hours using dobutamine in first-line treatment (14), and then adrenaline or norepinephrine if dobutamine does not rapidly maintain the blood pressure. Metabolic acidosis marked by a base deficit above 5 mmol/l is a serious sign, and the only sign in our opinion that justifies use of thrombolytic agents (10).

Acute Cor Pulmonale complicating acute respiratory distress syndrome

In this setting, two associated factors combine to raise right ventricular outflow impedance: 1/ underlying pulmonary disease, which usually causes permanent diffuse arteriolar obstructions (15); 2/ assisted ventilation (16), which results in microvascular, intermittent or permanent obstructions, by elevation of transpulmonary pressure (17, 18).

Bedside echocardiography provided the first description of this complication of ARDS, at a time when high tidal volumes (13 ml/kg) were used (FILM 20, FILM 21) (19). The frequency of this complication was 61% then, a value close to mortality of the syndrome. It is now known that these tidal volumes, and the high plateau pressure they induce, are excessive. Reduction in plateau pressure to below 30 cm H 2O significantly reduces the frequency of ACP to about 25% (9) (FILM 22, FILM 23).

The onset of ACP during ARDS is generally more gradual than during PE and is observed after a certain time on assisted ventilation (9). In certain patients ACP may occur on introduction of assisted ventilation (FILM 24, FILM 25) or can be triggered by untimely adjustment of respirator settings (FILM 26, FILM 27, FILM 28). In some patients, the later onset of ACP indicates a fibroproliferative phase, which can be arrested by corticosteroid therapy (FILM 29, FILM 30).

If ACP occurs during ARDS, the following measures should be implemented immediately:

- reduce plateau pressure to below 25 cm H 2O
- lower PEEP to below 8 cm H 2O
- reduce PaCO 2 to below 60-65 mmHg by use of a heater/humidifier in place of the filter (20), possibly by increasing respiratory frequency in certain patients. However, this maneuver is rarely effective and by generating an intrinsic PEEP often raises the plateau pressure, at the expense of right ventricular ejection (21). Remember that hypercapnia, which leads to systemic vasodilatation, has the reverse effect on the pulmonary circulation, resulting in arteriolar vasoconstriction (22).
- prone positioning if the ratio PaO 2/FIO 2 remains below 100 mmHg
- use TEE to check the absence of proximal PE

If ACP is accompanied by insufficiency circulatory, the most suitable vasoactive drug is norepinephrine, which restores systemic blood pressure and so improves right coronary flow and right ventricular systolic function (FILM 25, FILM 31).

When ACP appears after more than one week of assisted ventilation in a patient whose lung compliance is deteriorating, and in whom hypercapnia is increasing, this combination is strongly suggestive of a fibroproliferative phase. We then always use corticosteroid therapy.

Lastly, inhaled NO can also afford rapid relief and reduce or eliminate signs of ACP (FILM 32, FILM 33).

In our experience, immediate implementation of these measures, which presupposes rapid echocardiographic diagnosis, has meant that ACP no longer results in excess mortality in ARDS. ACP can greatly reduce the likelihood of cure if specific and timely measures are not taken (19).

Acute Cor Pulmonale in other clinical settings

Sudden obstruction of the pulmonary circulation by a gas or fat embolism causes acute pulmonary artery hypertension, which is often rapidly reversible. We have reported a case of ACP triggered by intravenous injection of drug powder (FILM 34) (23).

Acidosis, whether respiratory or metabolic, induces pulmonary artery hypertension, which has long been known to complicate primary lactic acidosis (24). We have observed several cases of ACP complicating primary lactic acidosis (1). Lactic ~~acidosis~~ caused by septic shock may also be involved in the onset of ACP (FILM 35, FILM 36).

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