03. SYSTOLIC OVERLOAD

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 (Figure 5), and can be analyzed in more detail in motion mode (M-mode) (Figure 1).
Film 3 : Transthoracic echocardiography (TTE) in a female patient on controlled mechanical ventilation because of ARDS due to pneumococcal pulmonary infection. The circulatory insufficiency in this patient was partly due to ACP. The parasternal short-axis

view of the LV shows a greatly dilated RV deformed at its outflow tract. There is paradoxical septal motion as shown by the interventricular septum pushing into the LV at the end of systole and start of diastole.

Film 4 : In the same female patient as in *film 3,* TEE completed the hemodynamic study. The transgastric view revealed paradoxical septal motion. Note also the presence of significant tachycardia.
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 (Figure 5).

» Second, incipient hypertrophy of the free wall of the RV occurs, with accentuation of muscular trabeculae (Figures 6 et 7), and wall thickening (Figures 7 et 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.

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. 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.

Film 5 : Transthoracic parasternal short-axis view of the vessels at the base of the heart in a spontaneously breathing female patient hospitalized following massive PE. Pulsed Doppler just upstream of the sigmoid valves of the pulmonary artery visualizes biphasic pulmonary artery flow characteristic of obstructed right ventricular ejection.

Film 6 : Biphasic pulmonary artery flow is also seen in ARDS in the case of ACP. This is apparent in a transgastric view in this female patient on controlled mechanical ventilation because of ARDS due to pneumococcal pulmonary infection.
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.

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.

Media

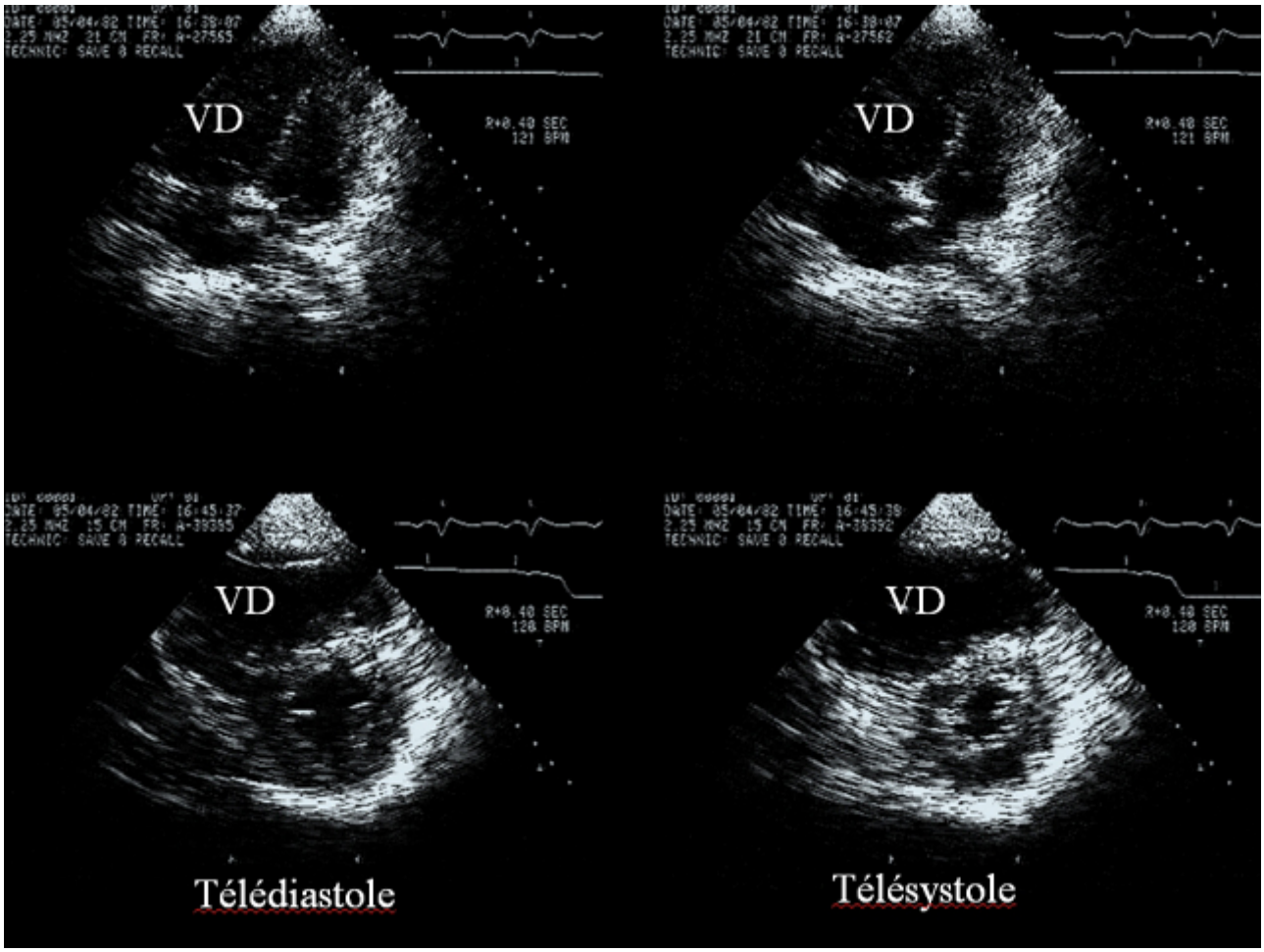

Figure 5 : Right ventricular deformation in acute cor pulmonale. In a young female patient presenting acute cor pulmonale complicating acute respiratory distress syndrome, the apical four-chamber view (above) shows right ventricular dilatation accompanied by a rounded appearance of the apex, at the end 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.

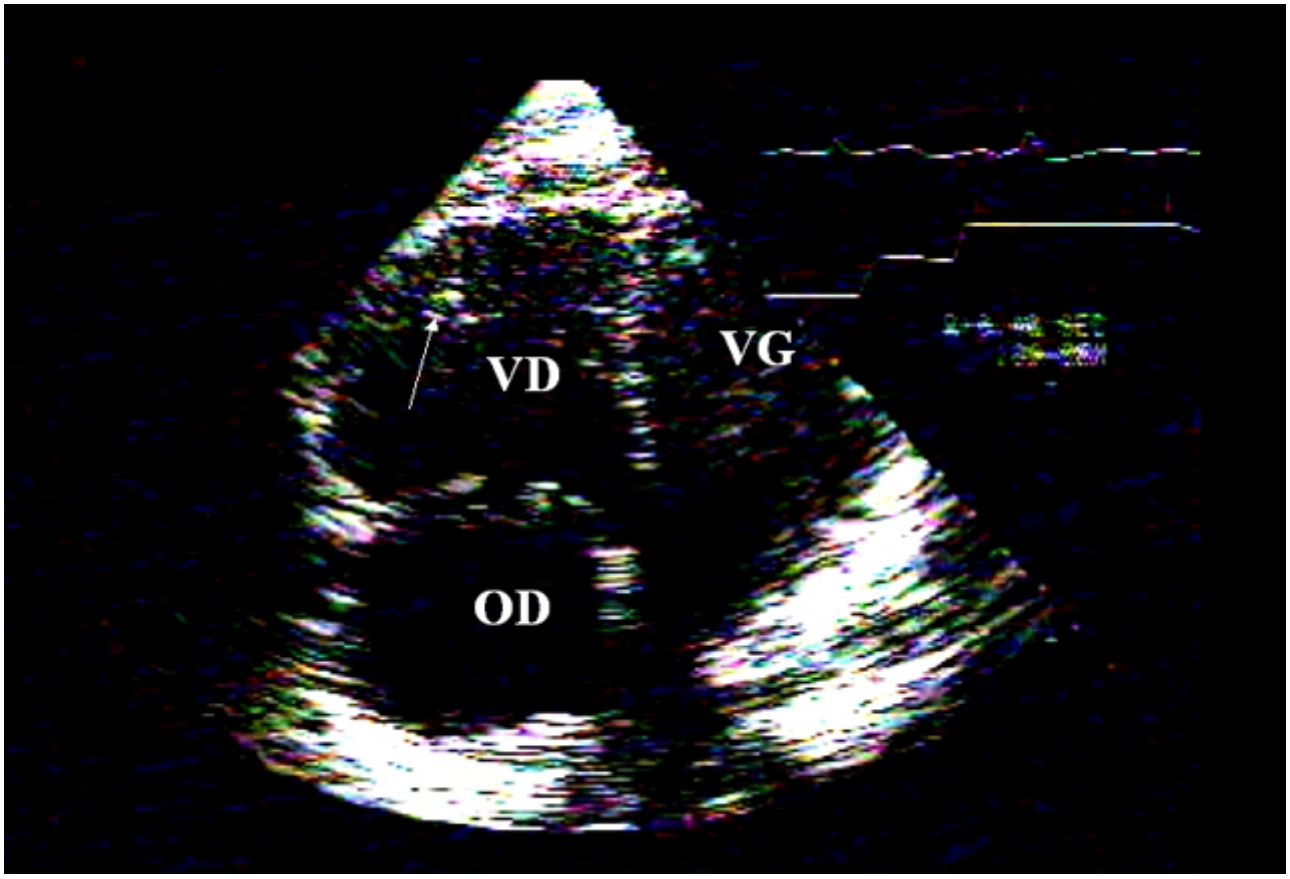

Figure 6 : Rapid right ventricular hypertrophy in acute cor pulmonale, first example. In this patient presenting acute cor pulmonale complicating acute respiratory distress syndrome, hypertrophic trabeculae are seen in the dilated right ventricular chamber (arrow).

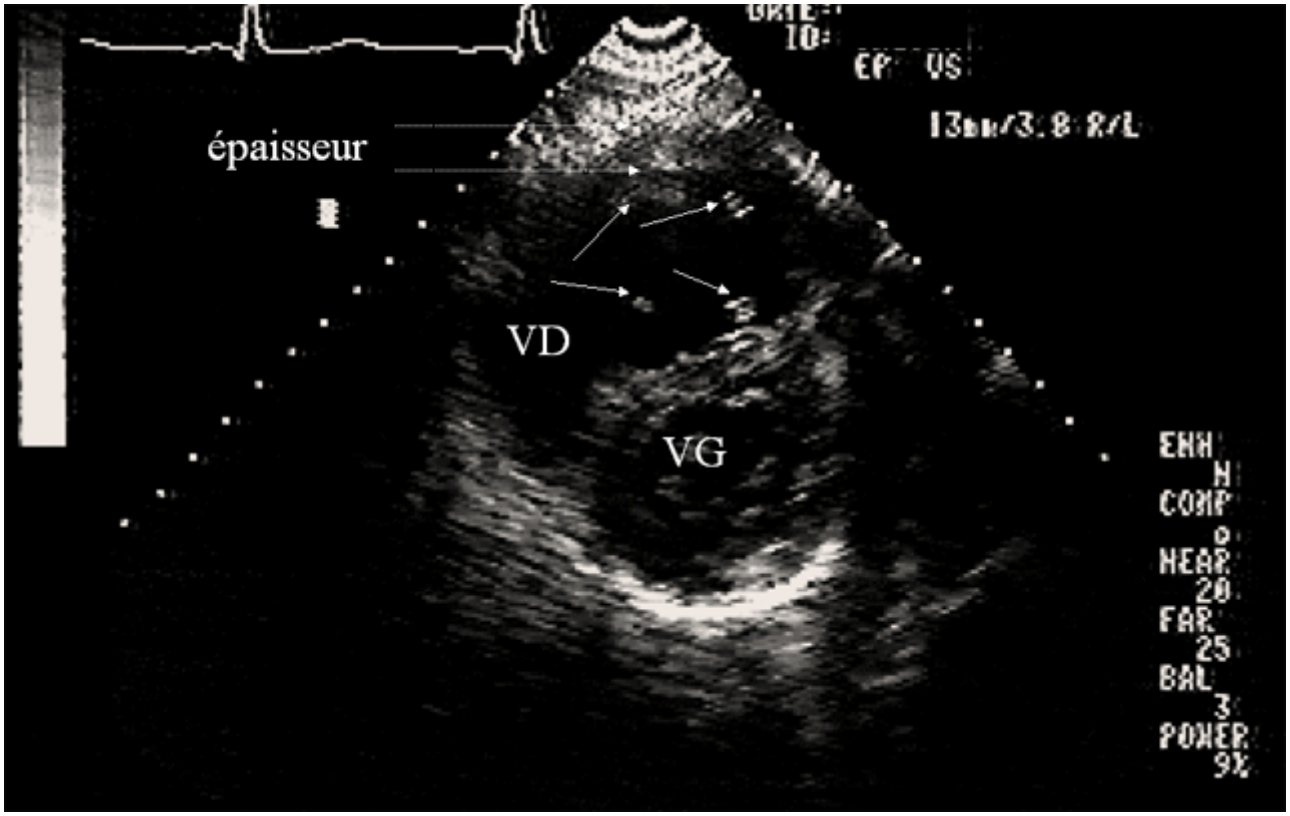

Figure 7 : Rapid right ventricular hypertrophy in acute cor pulmonale, second example. In this female patient presenting acute cor pulmonale complicating massive pulmonary embolism, hypertrophic trabeculae are seen in the dilated right ventricular chamber (arrows). Note also the thickness of the wall: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

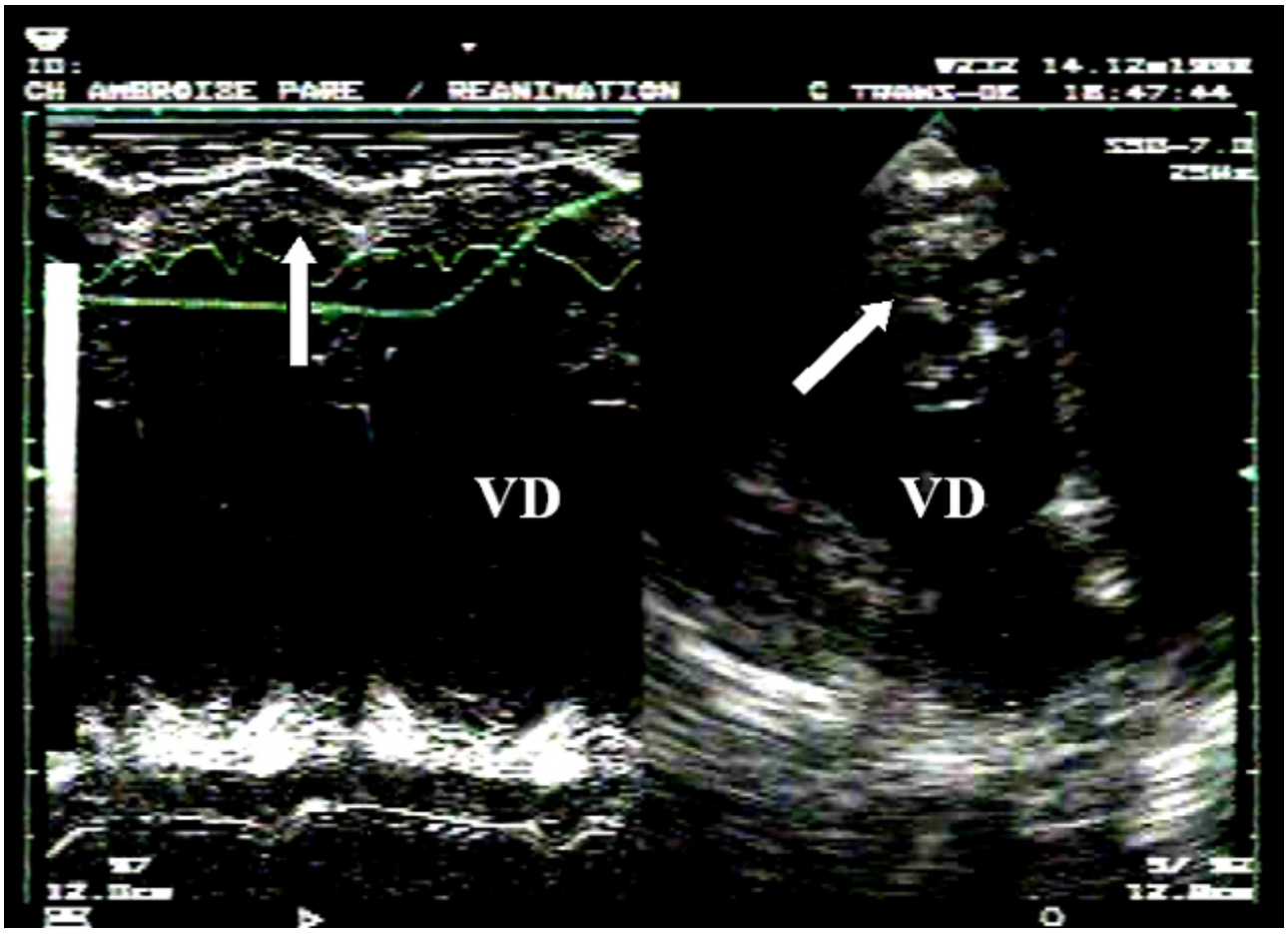

Figure 8 : apid right ventricular hypertrophy in acute cor pulmonale, third example. In this patient presenting acute cor pulmonale complicating acute respiratory distress syndrome, hypertrophy of the right ventricular wall is clearly visible on a transgastric approach in M-mode (on left, arrow).

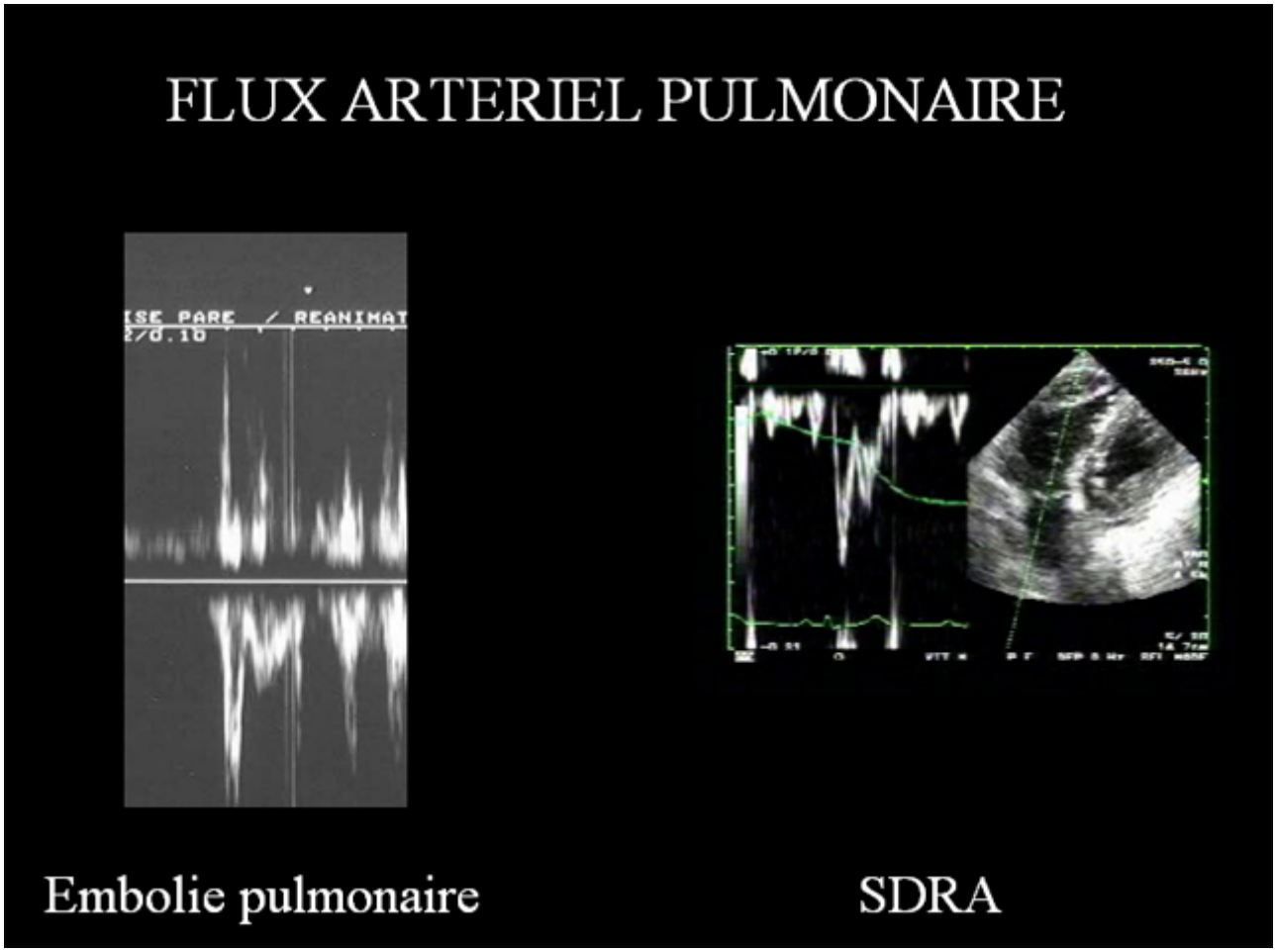

Figure 9 : Aspect biphasique du flux artériel pulmonaire.La survenue d’une augmentation brutale de la résistance à l’écoulement dans la circulation pulmonaire modifie le profil du flux Doppler dans l’artère pulmonaire, qui devient biphasique. Cette anomalie s’observe aussi au cours des hypertensions artérielles pulmonaires chroniques.