Silent Pericardial Effusion in Late Pregnancy

To the Editor:

In order to investigate the hemodynamic changes which occur during pregnancy, 40 healthy pregnant women (mean age 28 years) were studied at various stages of their gestation by Tm and two-dimensional echocardiography.

All pregnancies were uncomplicated and there was one twin gestation. Of 40, 20 women were in late pregnancy (32nd to 38th weeks of pregnancy) and complained with occasional dyspnea and/or palpitations. In all cases, cardiac examination was normal.

In 2 of 20, blood pressure was elevated respectively at 150/90 and 160/80 mm Hg; in all cases the electrocardiogram was normal or showed nonspecific ST-T changes.

The data concerning myocardial function are discussed elsewhere. We want to stress here the fact that surprisingly, in 8 of the 20 women who were in late pregnancy, echocardiograms showed definite signs of pericardial effusion as strictly defined: in these cases a separation between posterior left ventricular epicardium and pericardium continued through the cardiac cycle. According to Horowitz's criteria, the effusion was large in one, (Fig 1), moderate in two, and small in four cases.

Pericardial effusion was clinically silent as neither precordial pain nor pericardial friction rub was present; it appeared in late pregnancy and, in all cases, did not occur before the 32nd week; it was transient and could no longer be detected two months after delivery.

None of the pregnant women presented with eclampsia, recent viral illness or evidence of congestive heart failure. In both women with high blood pressure, pericardial effusion was respectively large and moderate.

To our knowledge, pericardial effusion has not been reported so far in the late pregnancy; it is likely to result from water and salt inflation; in the 8 pregnant women who showed a pericardial effusion, the mean weight gain was higher than in the 12 others who were free of any pericardial fluid at the same stage of gestation; however, the difference was not statistically significant, due to the small number of cases of each group.

Silent pericardial effusions may at times be present in the late pregnancy; as they cannot be detected by clinical examination and electrocardiogram, echocardiography could afford a safe and reliable approach of their diagnosis.

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REFERENCES

1 Halphen Ch, Halat R. Modifications électrocardiographiques induites par la grossesse. Coeur (in press)

Bedside Calibration Check of Pulmonary Artery Catheters

To the Editor:

The use of flow directed balloon-tipped catherers for hemodynamic monitoring of pulmonary artery pressures has gained both widespread acceptance and usage. A barrage of elaborate instrumentation is now available to give appropriate pressure readings. The electronics have become so sophisticated that many physicians must rely on special "monitoring teams" to set up and calibrate equipment. This often puts the physician in the unenviable position of making therapeutic decisions based upon numbers he cannot fully validate himself.

To review briefly, two basic calibrations of the monitoring instrument are required. The first is electronic, which assures that a given voltage input yields a given value on the

![Figure 1A](image-url)
Pulmonary Granulomatosis from Intravenous Use of Oral Medication

To the Editor:

Pulmonary granulomatosis due to intravenous injection of oral medications is a fascinating but at present incompletely understood entity. The recent CPC on drug-induced pulmonary granulomatosis in the July issue of Chest summarized a great amount of information, but there are several additional points that should be made.

While it was reported in the CPC that reduced expiratory flow rates may be seen in patients with drug-induced pulmonary granulomatosis, this physiologic abnormality has been an inconsistent finding. For example, in a review of the entity, Paré et al noted reduction in forced expiratory volume in one second (FEV1) and midexpiratory flow rate (MMF) in 11 of 17 patients. Douglas et al earlier reported reduced midexpiratory flow rates in three of six patients, all of whom, however, had a co-existing marked reduction in vital capacity. Furthermore, normal expiratory flow rates can occur in a patient with severe drug-induced pulmonary granulomatosis. In a recent case of anatomically-confirmed severe pulmonary granulomatosis, normal values for FEV1 and MMF were present.

Hypoxemia has also been noted as a variable finding, but significant hypoxemia, it should be emphasized, can occur in the course of the disease. Moderately severe hypoxemia (PaO2 72 mm Hg with P[A-a]O2 gradient of 28 mm Hg) was present, for example, in the 25-year-old patient noted in the case above. As this patient symptomatically improved, there was also improvement in her degree of oxygenation. An earlier group of patients with drug-induced pulmonary granulomatosis showed a similar reduction in PaO2 and increased P(A-a)O2 gradient.

Thus, although hypoxemia may not be a necessary accompaniment of drug-induced pulmonary granulomatosis, it can certainly occur in this entity, and it appears that the degree of hypoxemia may be used in serially following the course of the patient.

Lastly, although it was reported that pulmonary granulomatosis results after 2-20 years of intravenous use of oral medication, severe pulmonary impairment may occur much sooner. In the 25-year-old addict noted above, anatomically-proven severe granulomatosis occurred after nine months of intravenous use of ritalin. The variable duration of drug usage until the onset of symptoms and the difference in the degree of parenchymal versus vascular involvement between patients certainly suggests a differing response to this type of pulmonary insult.

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REFERENCES