However, the etiology of the transitory pulmonary artery-bronchial fistula detected in our patient during the bedside pulmonary angiography remains unclear. That patient did not show any clinical or radiographic evidence of pulmonary hemorrhage before the angiographic procedure. The only manipulation made was a slight withdrawal of the Swan-Ganz catheter to correct an intracardiac knotting a few times later on its introduction. We suggested several mechanisms to explain the complication observed, but we did not mention the increase of PWP was responsible for it. We believe that in our case the catheter was too far advanced into the pulmonary artery and the balloon could be only partially inflated every time a PWP measurement was made. In that case the catheter tip protrudes beyond the partially inflated balloon and can be deviated into the arterial wall producing a thinning of the wall of the pulmonary artery, as it has been shown experimentally in the dog. It is quite possible that a combination of a contrast injection through a deviated wedged catheter on a previously damaged wall produced the perforation of the pulmonary artery. The spontaneous recovery of our patient could be due to the absence of anticoagulant therapy.

It seems difficult to accept the idea suggested by Allen and Olsen that bedside pulmonary angiography merely documented a clinically silent fistula. We believe that pulmonary angiography can contribute to production of such an abnormality when the catheter can be wedged with such little balloon inflation and therefore we recommend withdrawing it.

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REFERENCE

An Unusual Mechanism for a Fistulous Communication between the Aorta and the Right Side of the Heart

To the Editor:

After I read the well documented article by Skorton et al in Chest (1980; 77:796), an unanswered question remained in my head: Did this young patient really have to die?

The facts are that a 18-year-old girl was admitted, after successful cardiopulmonary resuscitation, to a well known and optimally equipped university hospital. She presented the classic findings of acute severe aortic insufficiency and additionally left-to-right shunt. The result was cardiogenic shock recognized and treated with high dosage of dopamine. Multiple invasive and noninvasive diagnostic tests were undertaken, the gynecologist emptied the infected uterus, plans were made for hemodialysis, but no word is said about the possibility of active improvement of the desperate hemodynamic status by surgical intervention.

Even though the patient’s history was typical for sepsis two and septic shock could be considered, the clinical findings indicated clearly intractable cardiogenic shock. Early surgical intervention with closure of the defect and replacement of the aortic valve may have altered this patient’s deleterious course. Therefore, again the question: Did this “child” really have to die?

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To the Editor:

We read with interest the letter by Dr. Messmer, who suggests that the patient discussed in our case report may have died for want of a cardiac surgical procedure.

Although we certainly share his regret concerning the outcome, we are compelled to point out the complexity of the case. This patient’s critical condition was due to more than her cardiac defect. For example, her hemodynamic state was not due solely to cardiogenic factors, as suggested by Dr. Messmer, but clearly reflected a contribution of sepsis. She also had evidence of renal failure with oliguria, disseminated intravascular coagulation, and metabolic acidosis, presumably due in part to lactate accumulation. The physicians and surgeons involved in the case were convinced that cardiac surgery should be postponed until some correction of the patient’s sepsis and metabolic derangements could be accomplished. Unfortunately, this patient expired some 22 hours after admission to hospital, precluding hemodialysis, further antibiotic administration, and cardiac surgery, the intended courses of action.

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Internal Jugular Thrombosis and Pulmonary Embolism

To the Editor:

Venous thrombosis is considered an uncommon complication of indwelling central venous catheters. Pulmonary embolism and right internal jugular thrombosis at the site of an indwelling Cordis sheath introducer was noticed at a recent autopsy. Venous thrombosis was not found elsewhere, although careful examination of the femoral, pelvic, great venous structures and the heart was performed. This case prompted a review of all autopsies performed from December, 1979, through July, 1980, at our institution.

Fifty autopsies were performed during this time. Twenty-six had indwelling internal jugular Cordis sheath introducers at some time during the hospital course. Four of the 26 cases had ipsilateral gross internal jugular thrombi at the site of the Cordis sheath, and two of these four cases had significant pulmonary emboli (venous thrombosis elsewhere was not identified).

Three of the four cases with internal jugular thrombosis (Cordis site) also had Swan-Ganz pulmonary artery catheters placed through the internal jugular Cordis sheath introducer at one time during their hospital course. One of the three cases had both internal jugular thrombosis and pulmonary emboli at autopsy.

At our hospital, Cordis sheath introducers routinely have been placed within an internal jugular vein to establish a route for subsequent Swan-Ganz catheter or temporary cardiac pacemaker catheter placement. This procedure is routinely performed by a member of the medical resident staff, trained
in the technique, and is done sceptically at the bedside. Swan-Ganz catheters and cardiac pacemakers are usually removed within three days. Indwelling Cordis sheath introducers are often maintained for longer periods with the administration of crystalloid through the infusion arm of the Cordis sheath introducer. In this series, the duration of all Cordis sheath introducers ranged from 4 hours to 10 days, with the mean duration of 4 days. Local cellulitis of the sheath site was not evident from hospital records or autopsy. Daily maintenance by the nursing staff with aseptic techniques is adhered to strictly at our institution.

This report emphasizes the fact that venous thrombosis, as well as pulmonary thromboembolism, may represent significant hazards of internal jugular venous catheterization. This has not been reported or emphasized previously. Conversely, Turnbull et al reported no thromboembolic complications of internal jugular catheterization with the Cordis sheath introducer in 175 patients. Potential mechanisms for the development of internal jugular venous thrombosis in the autopsy series reported include: 1) the known thrombogenic features of the Swan-Ganz catheter; 2) potential thrombogenic influence of the Cordis catheter; 3) possible potentiation of 1 and 2.

In conclusion, we wish to emphasize the fact that internal jugular thrombosis can occur in critically ill patients, is often unrecognized, and can result in life-threatening pulmonary emboli.

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Combined Exercise Echocardiography

To the Editor:

We have been quite interested in the concept of exercise nuclear angiography and its application to the identification of the ischemic left ventricle.1-2 It was observed that in a large majority of cases, positivity or negativity of the examination could be predicted on the basis of changes in systolic diameter across the LV minor axis.

From this observation, we designed an exercise test using a combination of M-mode echocardiography for left ventricular chamber measurement and two dimensional images for spatial orientation. Two dimensional techniques were also used to exclude large functional aneurysms of the left ventricle.3,4 One hundred sixty-six consecutive patients being evaluated for coronary artery disease had combined exercise echocardiography. Excluded were patients with significant valvular disease or suspected cardiomyopathy. Two patients had technically inadequate exams and were excluded. Fifty of these patients subsequently had coronary angiography and were chosen for our study.

The combined echo stress protocol requires standard ATL two dimensional echo equipment and a motorized treadmill. Before exercise, all patients were examined in the supine position and the best echocardiographic “window” selected. An M-mode strip was then recorded across the minor axis using the two dimensional transducer for spatial orientation. Upright exercise was then carried out using a standard Bruce protocol. The patient was exercised to exhaustion or progressive chest pain. Following this, the patient was again placed supine and the heart was imaged using the same “window” in the same axis at the same phase of respiration. M-mode strips were again recorded across the minor axis under two dimensional guidance. All examinations were discontinued five minutes after exercise.

The test was considered abnormal if: end-systolic diameter increased, remained the same or decreased by less than 3 mm; or stress-induced wall motion abnormalities could be demonstrated.

Patients were subdivided according to the results of standard exercise testing and exercise echo examination. Of those patients with negative exercise ECGs and abnormal stress echoes, all had significant coronary artery disease. The echo stress test was equally helpful in predicting the presence or absence of coronary disease in patients with equivocal treadmill tests. Exercise stress testing appeared to be complementary when both exams were positive or negative.

Of the 50 patients studied, there were two false negatives (both had single vessel disease) and one false positive (a patient with Barlow’s syndrome).

Though these results are preliminary, our initial experience is encouraging.

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