

Systematic non-invasive measurement of respiratory mechanics was done using the pressure transducers and the pneumotachograph built into the 900C Servo Ventilator. An on-line infrared CO<sub>2</sub> analyzer (Hewlett Packard 78556A) was used for determining breath by breath end-tidal PCO<sub>2</sub>. Auto positive end-expiratory pressure (autoPEEP) was measured by occluding the expiratory port of the ventilator at end-expiration. Respiratory system compliance was determined by dividing the tidal volume by the difference between airway plateau pressure measured after an inspiratory occlusion of 2 s and the autoPEEP. Total inspiratory airway resistance was determined by dividing the difference between airway peak pressure and airway plateau pressure by inspiratory airflow. The time course of respiratory mechanics and gas exchange data are shown in Table 1.

**Table 1.** Lung mechanics and gasometric data immediately after the respiratory arrest up to recovery

	After arrest	1 h	4 h	8 h
Ppeak (cmH <sub>2</sub> O)	41.1	33.7	25	13
Pplat (cmH <sub>2</sub> O)	21.4	17.3	14.5	9.5
Raw (cm/l/s)	34.6	25.2	22.3	10.7
autoPEEP (cmH <sub>2</sub> O)	17	12	4	1
Crs (ml/cmH <sub>2</sub> O)	86	87	69	73
PaO <sub>2</sub> /FiO <sub>2</sub> (mmHg)	348	392	297	390
a-et PCO <sub>2</sub> (mmHg)	10.9	7.3	5.5	3

Ppeak, airway peak pressure; Pplat, airway plateau pressure; Raw, total inspiratory airway resistance; Crs, respiratory system compliance; PaO<sub>2</sub>/FiO<sub>2</sub>, arterial PO<sub>2</sub>/inspiratory oxygen fraction; a-et PCO<sub>2</sub>, arterial minus end-tidal PCO<sub>2</sub>

Bronchospasm progressively ameliorated and the patient was successfully extubated 3 h after discontinuation of sedation and relaxation and, 11 h after admission. At this time arterial blood gases showed pH 7.43 and PaCO<sub>2</sub> 34 mmHg. On discharge, 6 days after admission, spirometry revealed a slight obstructive defect with a FVC of 3.79 l, a FVC/FVC predicted rate of 104%, a FEV<sub>1</sub> of 2.68 l (88% of predicted) and a FEV<sub>1</sub>/FVC of 71%.

This case illustrates the evolution of lung mechanics and gas exchange during mechanical ventilation in severe asthma. Serial evaluation of data showed that, at similar values of respiratory system compliance and oxygenation, both autoPEEP and total inspiratory airway resistance have progressively decreased. This indicates that during the period of recovery, typically after 12–24 h of ongoing bronchodilator and corticosteroid therapy, autoPEEP and total airway resistance are the indexes that should be continuously monitored at the bedside. Additionally, amelioration of bronchoconstriction was paralleled by a decrease in the PaCO<sub>2</sub>-PetCO<sub>2</sub> gradient. This suggested that an improvement of ventilation-perfusion inequalities within the lung that led to an increase in CO<sub>2</sub> concentration during expiration was originated [3].

Early and serial monitoring of lung mechanics and gas exchange may also add some information concerning the development of respiratory arrest in fatal or near-fatal asthma [4]. In status asthmaticus, the increased ventilatory efforts needed to overcome the widespread airway narrowing preserves alveolar ventilation at the expense of increased work of breathing. Then, because the rate of lung emptying is slowed, end-expiratory lung volume exceeds predicted functional residual capacity and dynamic pulmonary hyperinflation occurs and progressively worsens. This dynamic hyperinflation, also named intrinsic or autoPEEP, has a number of adverse effects: the respiratory muscles operate at an unfavourable part of their length-tension curve, elastic recoil of the chest is directed inwards and, breathing takes place at the upper and less compliant portion of the pressure-volume curve of the lung [2, 5]. Therefore, assuming in our patient a similar degree of dynamic hyperinflation at the time of respiratory arrest to that obtained at the first set of lung mechanics measurement (Table 1), hyperinflation due to very high airway resistance should have placed the lungs at end-expira-

tion at near total lung capacity; spontaneous breathing could no longer be sustained and respiratory arrest did develop.

Yours faithfully,

L. Blanch, R. Fernandez, A. Ferrer, F. Baigorri and A. Artigas

## References

1. Broseghini C, Brandolese R, Poggi R et al (1988) Respiratory mechanics during the first day of mechanical ventilation in patients with pulmonary edema and chronic airway obstruction. *Am Rev Respir Dis* 138:355–361
2. Tuxen DV, Lane S (1987) The effects of ventilatory pattern on hyperinflation, airway pressures, and circulation in mechanical ventilation of patients with severe air-flow obstruction. *Am Rev Respir Dis* 136:872–879
3. Blanch L, Fernandez R, Artigas A (1991) The impact of autoPEEP on the capnogram in patients with acute respiratory failure during total ventilatory support. *Am Rev Respir Dis* 143:A485
4. Molfino NA, Nannini LJ, Martelli AN, Slutsky AS (1991) Respiratory arrest in near-fatal asthma. *N Engl J Med* 324:285–288
5. Tobin MJ, Lodato RF (1989) PEEP, autoPEEP, and waterfalls. *Chest* 96:449–451

Dr. L. Blanch, Servei de Medicina Intensiva, Hospital de Sabadell, Apartat de Correus 196, E-08208 Sabadell, Spain

## Pulmonary artery catheter placement and temporary cardiac pacing in a patient with a persistent left superior vena cava

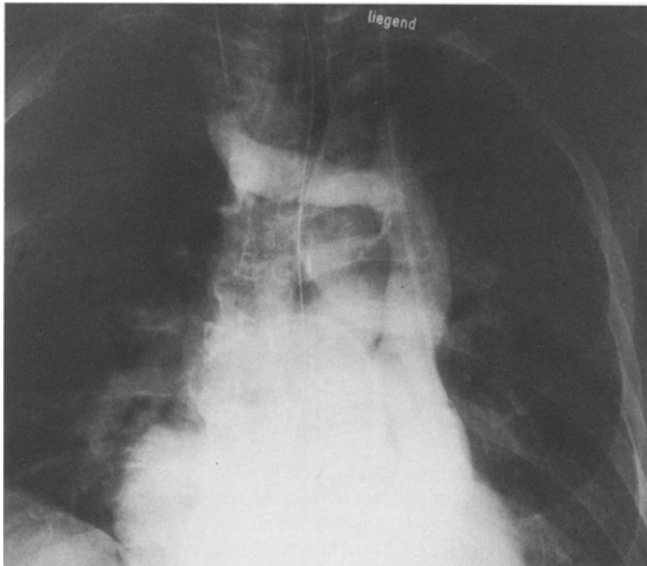
Dear Sirs,

We recently encountered a 76-year-old male patient who required admission to our ICU because of progressive mental deterioration and hemodynamic instability due to sepsis. A pulmonary artery catheter was introduced via the left internal jugular vein. Definite placement of the catheter in the pulmonary outflow tract proved to be difficult and the catheter had to be withdrawn and readvanced several times. After pulmonary artery and wedge tracings confirmed proper catheter placement, an AP chest film was taken. The radiograph showed an unusual position of the catheter, running parallel to the left midclavicular line with its tip in the right pulmonary artery. Contrast media was injected via the central venous catheter in the right internal jugular vein and the chest radiograph was repeated. This demonstrated an absent right superior vena cava, a right brachiocephalic vein and a persistent left superior vena cava (Fig. 1).

Transesophageal echocardiography was performed. The pulmonary artery catheter entered the right atrium via the persistent left superior vena cava and the coronary sinus. No other cardiac abnormalities could be demonstrated. The patient's haemodynamic status improved and the pulmonary artery catheter could be removed.

Four days later, he developed bradycardic episodes and cardiac arrest during airway suctioning and positioning. This required resuscitation and temporary cardiac pacing. A balloon tipped pacing electrode was introduced using the left internal jugular vein. Advancement into the right ventricle proved again to be difficult, several attempts were necessary until ventricular capture occurred at the pacer's maximal output setting. Because proper sensing of the device could not be demonstrated, the pacemaker was used in the asynchronous mode. The chest radiograph showed the tip of the pacing electrode floating closely to the wall of the right ventricle without demonstrating proper contact.

A persistent left vena cava is not a rare abnormality. Its incidence varies between 0.3% in healthy subjects with otherwise normal hearts to 4.3% in patients with congenital heart disease [1] and probably repre-



**Fig. 1.** The left superior vena cava is opacified with contrast media injected via the central venous catheter in the right internal jugular vein. The anomalous vessel follows the left paramediastinum and the left cardiac border. The right vena cava is absent and a right brachiocephalic vein present

sents the most common abnormality of venous return to the heart. It may occur as an isolated lesion or can be associated with an absent right superior vena cava as in our patient.

Pronounced technical problems can result from this entity, however, when right heart catheterization is attempted, and the right superior vena cava is absent as in our patient. Maneuvering a catheter through the narrow opening of the coronary sinus into the right atrium can be difficult [2] and proper placement of a pacing wire can be made impossible, as this case demonstrates. The clinician should be aware of the existence of this abnormality and an effort should be made to identify its presence when right heart catheterization is planned.

The clinical sign of abnormal left jugular pulsation in persistent left superior vena cava has been described [3]. On a plain chest film taken under standardized conditions, this entity may be indicated by a widening of the aortic shadow, a paramediastinal bulge below the aortic arch or a strip of lessened density along the left upper cardiac border [4]. Retrospective analyses of the preoperative chest radiograph in our patient, however, did not demonstrate any of these findings. When this condition is present, associated cardiac anatomic abnormalities should actively be excluded by echocardiography.

When emergency cardiac pacing is required in such patients [5], the initial use of a transcutaneous pacing system [6] maybe of advantage. Right heart catheterization could probably be easier performed by a femoral venous approach. The absence of the right superior vena cava could simplify catheter placement under these circumstances.

Yours faithfully,

G. Schelling, J. Briegel, K. Eichinger, W. Raum and H. Forst

## References

1. Campell M, Deuchar DC (1954) Left-sided superior vena cava. *Br Heart J* 16:423–439
2. Falltrick RT (1979) Pulmonary artery catheterization through a persistent left superior vena cava. *Anesthesiology* 50:155–156

3. Colman AL (1967) Diagnosis of left superior vena cava by clinical inspection; a new physical sign. *Am Heart J* 73:115–120
4. Cha EM, Khoury GH (1972) Persistent left superior vena cava. Radiologic and clinical significance. *Radiology* 103:375–381
5. Momma K, Linde LH (1969) Abnormal rhythms associated with persistent left superior vena cava. *Pediatr Res* 3:210–216
6. Dalsey WC, Syverud SA, Trott A (1984) Transcutaneous cardiac pacing. *J Emerg Med* 16:11

Dr. G. Schelling  
 Institut für Anaesthesiologie  
 Klinikum Großhadern  
 Marchioninstraße 12  
 W-8000 München  
 FRG

## Cisapride in intensive care

Dear Sir,

We used cisapride in patients admitted to the intensive care unit and having gastric dilatation and functional gastric paralysis. Cisapride has been shown to be an effective treatment of post-operative ileus [1, 2] and gastric stasis of different origins [3–5]. Eight patients (3 males and 5 females, aged between 18 and 83 years) were studied. Three had cranial and/or other multiple system trauma, 2 had acute respiratory insufficiency, one had needed cardiopulmonary resuscitation for septic shock, one had prolonged coma after ventriculo-peritoneal shunt procedure, and one had undergone major abdominal surgery. Enteral food remained undigested (although in 3 patients bowel sounds were heard at auscultation) and tube feeding was impossible; up to 2 l of gastric dilatation fluid per day was produced.

Cisapride treatment was started 24 h after the diagnosis of functional gastric paralysis. The drug was administered as an oral suspension (1 mg/ml) through the nasogastric tube, and was dosed at 10 mg q. i. d. With a view to preventing dilution of the medication in situ, the gastric contents were aspirated and discarded immediately before administration of the first dose of cisapride. Thereafter, gastric aspiration prior to drug administration was no longer deemed necessary.

The treatment lasted 2 days in 4 patients, and up to 10 days in the other patients. The onset of the effect of cisapride, as indicated by the restarting of gastric emptying, was observed after 2 h in one patient and after 24 h in 4 patients, and was considered as an excellent result. In the other patients, the effect of the cisapride treatment was good, gastric emptying having resumed with 2 or 3 days, except in the patient who had produced 2 l of dilatation fluid per day (onset after 9 days).

There were no complications related to the cisapride treatment; 2 patients had loose stools during the treatment.

In conclusion, these findings suggest that cisapride is useful in advancing the start of enteral feeding in intensive care patients with gastric stasis.

Yours faithfully

L.F. Lauwers

## References

1. Verlinden M, Michiels G, Boghaert A, De Coster M, Dehertog P (1987) Postoperative gastrointestinal atony: cisapride promotes recovery. *Br J Surg* 74:614–617
2. Bogaert A, Haesaert G, Mourisse P, Verlinden M (1987) Placebocontrolled trial of cisapride in postoperative ileus. *Acta Anaesthesiol Belg* 38:195–199