ly increased from 100% to 85, 70, and 50% of the initial pressure support, the frequency do not change at all after PS85. The comparison between Figs. 3 and 4 clearly shows that the average changes in P0.1 nicely paralleled changes in WOB, whereas this was not the case for any variable of breathing pattern, as clearly illustrated by Figs. 1 and 2. Indeed, the regression analysis through the individual data points (Figs. 5–6) provided additional strength to the message. The different statistical results obtained by Iotti and colleagues on our data may be explained by two facts: i) the approximation of data collection from the scattergrams, as they also stated; ii) the Bonferroni's correction implemented in our analysis upon the reviewers' request. However, the major point of the debate is the interpretation of changes in the breathing pattern more or less associated with levels of PSV. The amount of PSV can be set primarily either to sustain a pre-selected value of VE (for example to improve the arterial blood gases) or to reduce the ventilatory load upon the patient's respiratory muscles. We agree with Brochard and colleagues [3] that the latter was the main goal for which PSV was introduced. Hence we assumed that the patient's WOB and not VE was the leading physiologic variable to tune PSV. Then, we found that P0.1 appeared to be a simple, non-invasive and reliable variable to set PSV at a level where both insufficient and excessive support could be avoided [4]. Neither changes in VT nor in breathing frequency could provide the same piece of information. And not only for statistical reasons. Indeed, variations of VT were limited by the fact that the increase in patient's WOB with decreasing PSV prevented any proportional reduction in VT and VE [5]. Moreover the respiratory frequency, although better related than VT to WOB variations at decreasing levels of PSV, does not reflect the overall timing of ventilation. Ti/Ttot is the other important variable. In fact, if the patient's inspiratory muscles relax just after having triggered the pressure boost, the passive lung inflation is included in the inspiratory time (Ti) from the ventilatory point of view, but in the expiratory time (Te) for the relaxed inspiratory muscles and more importantly for the neural respiratory centers that "ceased firing". If the time required to deflate the lungs is considered, the expiratory time for the centers becomes even longer. Under those circumstances, a clear discrepancy between the "central" and the "ventilatory" Ti/Ttot occurs, which might be incompatible with the frequency set by the "central controller" [6]. If that discrepancy becomes excessive, the respiratory centers try to gather the control on the timing of ventilation by inducing either expiratory efforts during the lung inflation or inspiratory efforts during the lung deflation [7]. Therefore, the patient's breathing pattern is influenced not only by the level of support, but also by the activity of the central controllers adjusting the central drive to the variations of the respiratory load. P0.1 provides, with well-known limitations, a measurement of neuromuscular drive independent of timing. In other words, the modifications of the breathing pattern during PSV represent the final result of the patient-ventilator interaction, whereas changes in P0.1 may more closely reflect the respiratory muscle activation, which is what one wants to know, according to the initial purpose.

References

1. Alberti A, Gallo F, Fongaro A, Valenti S, Rossi A (1995) P0.1 is a useful parameter to set the level of pressure support ventilation. Intensive Care Med 21:547–553
2. Iotti G, Braschi A, Brunner JX et al (1995) Respiratory mechanics by least squares fitting in mechanically ventilated patients: applications during paralysis and during pressure support ventilation. Intensive Care Med 21:406–413
3. Brochard L (1994) Pressure support ventilation. In: Tobin MJ (ed) Principles and practice of mechanical ventilation. McGraw-Hill, New York, pp 239–258
4. Rossi A, Appendini L (1995) Wasted efforts and dyssynchrony: is the patient-ventilator battle back? Intensive Care Med 21:867–870
5. Stroetz RW, Hubmayr RD (1995) Tidal volume maintenance during weaning with pressure support. Am J Respir Crit Care Med 152:1034–1040
6. Younes M (1994) Proportional assist ventilation (PAV). In: Tobin MJ (ed) Principles and practice of mechanical ventilation. McGraw-Hill, New York, pp 349–370
7. Jubran A, Van de Graaf WB, Tobin MJ (1995) Variability of patient-ventilator interaction with pressure support ventilation in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 152:129–136

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Transient post-renal obstruction and renal protection against nephrotoxic damage

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Sir: We read with great interest the report of Navis et al. [1] describing ipsilateral protection of an obstructed kidney against nephrotoxic damage. Our experience with a similar observation we published previously [2] raises some additional comments.

We agree with the authors that unilateral renal obstruction may protect the ipsilateral kidney against nephrotoxic factors. Although relatively scarce, experimental and clinical observations suggest that protection may be directed against various subtypes of acute renal failure including cortical necrosis [3], reversible acute tubular necrosis [2] and glomerulonephritis [4]. We also agree with them that disseminated intravascular coagulation could have been a major factor of non-reversible unilateral acute renal failure. Although renal biopsy was not performed, the absence of recovery of the left kidney is consistent with cortical necrosis. More than 25 years ago, a very close animal model of unilateral cortical necrosis was reported by Watchi et al. [5] using the Shwartzman-Sanarelli reaction.

However, Navis et al. did not consider the potential aggravating factor of post-renal obstruction on the contralateral kidney. Indeed, experimental models have shown that unilateral ureteral obstruction can induce a contralateral renal arterial vasoconstriction [6] that could precipitate severe necrosis. In their observation, the unusual severity of the non-obstructed kidney injury might support this hypothesis.

Finally, this privileged observation could be of value in view of further experimental studies, since it emphasizes the theoretical interest of unilateral ureteral obstruction models to investigate the involvement of renal mediators in acute renal failure.

References

1. Navis GJ, Rommes JH, Baur C, De Jong PE (1995) Transient post-renal obstruction may protect the kidney against nephrotoxic damage. A case report. Intensive Care Med 21:610–611

2. Lelarge P, Bollaert PE, Mallié JP, Nace L, Bauer P, Larcan A (1992) Acute unilateral renal failure and contralateral ureteral obstruction. Am J Kidney Dis 20:286–288
3. Watchi JM, Magder E, Patte D et al (1969) Nécrose corticale unilatérale avec intégrité des artères rénales et sténose de l'uretère du rein contralatéral. Presse Méd 77:1609–1611
4. Editorial (1975) Unilateral glomerulonephritis. Lancet 1:206–207
5. Watchi JM, Altman J (1972) Role of ureteric pressure in protecting the kidney against cortical necrosis during the Sanarelli-Shwartzman reaction: an argument in favour of the primary role of vascular spasm in the mechanism of cortical necrosis. Rev Eur Etudes Clin Biol 17:982–987
6. Francisco LL, DiBona GF (1978) Role of the renal nerves in renal response to ureteral occlusion. Clin Res 26:690

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Reply

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Sir: We thank Dr. Bollaert and colleagues for their comments. Their observation of a patient in whom unilateral ureteral obstruction apparently protected the obstructed kidney against acute tubular necrosis shows a remarkable similarity to ours [1]. The similarity of these two cases, as well as an additional case they refer to, a patient with documented unilateral cortical necrosis with contralateral ureteral obstruction and a virtually normal kidney [2], supports the assumption that the renoprotection in the obstructed versus the non-obstructed kidney indeed reflects a modification of the disease process rather than a coincidental finding.

Dr. Bollaert offers the interesting hypothesis that the obstructed kidney may have aggravated the course in the contralateral kidney by eliciting reflex renal vasoconstriction in that kidney. Whereas experimental evidence supports the possibility of such a mechanism, the lack of information on separate renal blood flows in our patient does not allow us to confirm or refute the involvement of such a mechanism. Nevertheless our case, by the findings on urography at presentation, is the first one to provide a clue on the mechanisms involved. The nephrography in the non-obstructed kidney versus the absence of nephrography in the obstructed kidney shows that, at that time, filtration was absent in the obstructed kidney, whereas the contralateral kidney still filtrated the contrast medium. We suggested, therefore, that differences in filtration, and consequently in renal delivery of nephrotoxic substances may have been involved in the different outcome of the two kidneys. We are well aware, however, that this by no means excludes a role for differences in renal vascular tone as a contributing factor in the different outcome of the two kidneys. The renal vasomotor response to acute unilateral ureteral obstruction, however, is complex and evolves over time [3]. We agree with Dr. Bollaert, therefore, that experimental studies would be needed to unravel the mechanism of renal protection by ureteral obstruction and that the approach of studying models of unilateral ureteral obstruction may be a fruitful one to obtain new insights into the pathophysiology of acute renal failure.

References

1. Lelarge P, Bollaert PE, Mallie JP, Nace L, Bauer P, Larcan A (1992) Acute unilateral renal failure and contralateral ureteral obstruction. Am J Kidney Dis 20:286–288
2. Watchi J-M, Magder E, Patte D, Jungers P, De Montera H (1969) Nécrose corticale unilatérale avec intégrité des artères rénales et sténose de l'urètere due rein contralatéral. Presse Med 77:1609–1611
3. Harris KPG (1993) Models of obstructive nephropathy. In: Gretz N, Strauch M (eds) Experimental and genetic rat models of chronic renal failure. Karger, Basel, pp 156–168

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Fatal concomitant nosocomial legionnaires' disease and cytomegalovirus pneumonitis after cardiac transplantation

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Sir: We report a case of fatal septic shock due to infection with *Legionella pneumophila* serogroup 1 and cytomegalovirus (CMV) after cardiac transplantation. We draw attention to the need for an aggressive approach in patients with nosocomial pneumonia after cardiac transplantation and the diagnostic difficulties in detecting legionnaires' disease and CMV pneumonitis in the presence of other pathogens.

A 56-year-old man underwent orthotopic cardiac transplantation for dilatative cardiomyopathy and end-stage left ventricular failure. Immunosuppressive therapy was started with hydrocortisone (100 mg/day), cyclosporin (180 mg/day), and azathioprine (180 mg/day), with progressive reduction in dosage over the next few days. The immediate postoperative period was uneventful; the patient was extubated on day 3. On day 10 following transplantation, while receiving cyclosporin (60 mg/day) and prednisone (30 mg/day) – azathioprine was stopped on day 8 – the patient developed fever and clinical signs of pneumonia. Chest X-ray showed a new lobular infiltrate in the right upper lobe. Bronchoalveolar lavage (BAL) grew 10E3 cfu/ml of *Staphylococcus aureus*. Flucloxacillin treatment (4×2 g i.v./day) was started the same day. Direct immunofluorescence (DFA) for *Legionella* and early-antigen for CMV in BAL were negative. *Legionella* antigen detection in the urine is not routinely performed in our hospital. Despite antimicrobial treatment, the patient developed septic shock with multiorgan failure. A second BAL showed a positive DFA for *Legionella*. The same day, *L. pneumophila* serogroup 1 was isolated in cultures from the first BAL. Intravenous erythromycin (4×1 g/day) and rifampicin (1×600 mg/day) were added to the antimicrobial regimen. Thoracic computed tomography showed bilateral pleural effusions and retrosternal fluid collection. Drainage of these effusions was scheduled, but the patient died on the way to the operating room.