Correspondence

Fall in peak expiratory flow during haemodialysis in patients with chronic renal failure

It is becoming increasingly common, in various clinical settings, to infer the condition of the airways from determinations of peak flow measured alone, out of the context of a complete flow-volume profile. This practice is potentially problematic for a number of reasons, the principle one of which is evidenced in the report by Drs A Davenport and A J Williams (September 1988;**43**:693–6). The problem is that, much more than other measures of expiratory flow, peak flow is determined by volition and muscle strength in addition to airway calibre. As a result, while changes in other measures of flow may generally be taken to reflect changes in airway calibre or airway stability, changes in peak flow must always be suspect if the subject has lability of muscle strength or motivation.

The subjects in the study by Drs Davenport and Williams certainly fall into that category. Given the constitutional effects of a haemodialysis, the reported changes in peak flow could very well be the result either of the subject's having become (or become more) fatigued, weak, or debilitated or of changes in muscle potential. Accordingly, rather than being a contributory *cause* of the hypoxaemia observed, the fall in peak flow may just as well be the *result* of the hypoxaemia and other deleterious systemic changes temporarily induced by dialysis.

The fall in peak flow reported in this study may in fact represent precipitate airway narrowing, as the authors suggest. One must at least consider other culprits, however, particularly the possibility of a transdialysis diminution in the subject's ability to generate a concerted, maximal contraction of the respiratory muscles.

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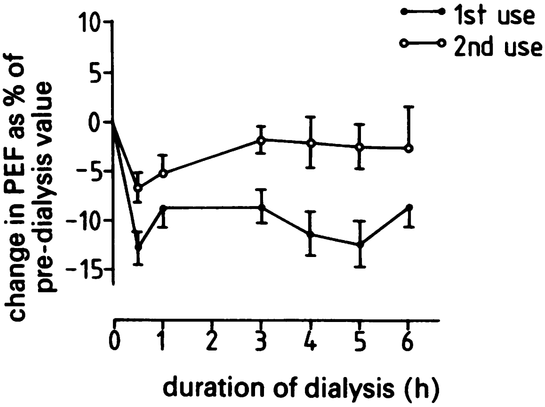
AUTHORS' REPLY We accept Mr Beauchamp's point that peak expiratory flow rates recorded close to total lung capacity during a forced expiratory manoeuvre are dependent not only on airway calibre but also on respiratory muscle strength and the patient's effort. The time course of the changes observed in peak flow during dialysis are not, however, consistent with a theory of altered muscle metabolic function, as both muscle glycogen reserves are normal (Davenport and King, unpublished data) and mitochondrial ATP production is enhanced.[1] Similarly, the changes in sodium, potassium, and calcium fluxes that occur during acetate dialysis would not be expected to cause a reduction in skeletal muscle function within the first hour of dialysis followed by an improvement.

In general, despite the theoretical reservations about peak flow rates, most subjects in practice produce reproducible values after minimal instruction.[2] In our study all patients were well motivated and had received prior instruction in peak flow technique. By taking the three maximum peak flow rates, provided that the difference was 20 l/min or less, we believe that changes in the patients' effort were minimised.

Evidence to support our contention that the reduction in airway calibre that occurs with acetate dialysis is related to using a cuprophan dialyser comes from our experience that the fall in peak flow observed during the first hour of dialysis did not occur in two patients having regular steroid treatment. In addition, when 15 of the study group reused the same dialyser after it had been cleaned with sodium hypochlorite and formalin, so altering its biocompatibility, a substantial amelioration in the fall in peak flow was observed (figure).



Peak flow rates (PEF) during dialysis (means with SEM).

Thus the changes we observed in peak flow rates correlate temporally with the activation of complement, neutrophils, and platelets and the development of both pulmonary hypertension and increased pulmonary vascular permeability that have been reported by other workers in the field of dialyser membrane bioincompatibility.[3]

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1 Barany P, Hultman E, Wilson R, Bergstrom J. Increased muscle mitochondrial ATP formation in haemodialysis patients with anaemia. *Nephrol Dial Transplant* 1988;**3–4**:536.
2 Gibson GJ. *Clinical tests of respiratory function*. London: Macmillan, 1984.
3 Walker JF, Lindsay RM, Sibbald JW, Linton AL. Acute pulmonary hypertension, leucopenia and hypoxia in early haemodialysis. *Proc EDTA* 1984;**21**:135–42.

Book notice

Surgery of the Oesophagus. G G Jamieson, ed. (Pp 934; £135.) Edinburgh: Churchill Livingstone, 1988. ISBN 0-443-034095.

Previous textbooks on this topic have been relatively slim