

Dalton reported that the premenstrual syndrome started after pregnancy in two fifths of 769 women,² so will this monozygous twin sister remain free from it after she has borne children?

Kantero and Widholm found a highly significant correlation for premenstrual symptoms (as opposed to the premenstrual syndrome) in Finnish adolescent daughters and their natural mothers.⁴ This further supports the suggestion that there is a genetic element in the aetiology of the premenstrual syndrome.

Our findings contribute to the theory that the study of nature, not nurture, holds the key to the aetiology of the premenstrual syndrome. Thus probably inquiries into antenatal factors and childhood environment are unfruitful topics for research into its aetiology and resources would be better employed investigating endocrinological factors.

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3 Erlenmeyer-Kinling L. Genetic approaches to the study of schizophrenia: the genetic evidence as a tool in research. *Acta Genet Med Gemellol (Rome)* 1983;32:53-88.

4 Kantero RL, Widholm O. Correlations of menstrual traits between adolescent girls and their mothers. *Acta Obstet Gynecol Scand* 1971;14 (suppl 14):30-6.

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London W1

KATHARINA DALTON, MRCS, FRCGP, obstetrician and gynaecologist

St James's University Hospital, Leeds LS9 7TF

MAUREEN E DALTON, MB, MRCOG, lecturer

KATHERINE GUTHRIE, MB, BSC

Correspondence to: Dr Katharina Dalton, 100 Harley Street, London W1.

Continuous arteriovenous haemofiltration in patients with hepatic encephalopathy and renal failure

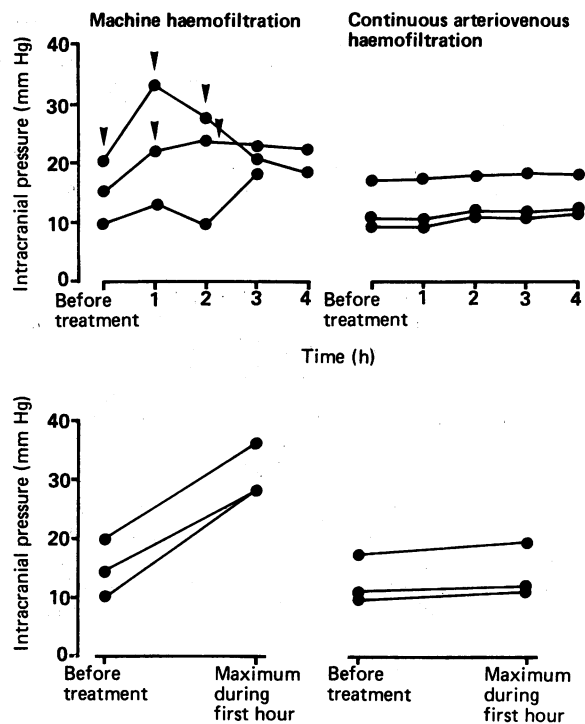
The mortality of patients admitted with fulminant hepatic failure remains high.¹ Charcoal haemoperfusion and dialysis with a polyacrylonitrile membrane have both been reported to increase survival.² Though death is often due to multiple complications, cerebral oedema is a common and serious problem.³ Treatments designed to control raised intracranial pressure are not always successful; hyperventilation does not produce a sustained reduction in pressure, and mannitol, which can control raised intracranial pressure, is not as effective in patients who have renal failure or appreciably increased pressures.⁴

We treated a patient who had hepatorenal failure by two methods of haemofiltration, with different effects on intracranial pressure.

Case report

A previously healthy 25 year old woman took 100 paracetamol tablets (0.5 g each) pending a divorce settlement. Three days later she started to feel unwell, became confused, and was admitted to the regional liver unit with grade IV hepatic encephalopathy. On arrival she responded only to deep pain and showed icterus with anuria. Blood biochemical measurements showed: paracetamol 36 mg/l, urea 18.4 mmol/l, creatinine 664 µmol/l, maximum prothrombin ratio 4.9, maximum alanine aminotransferase >3000 IU/l, and glucose 5.1 mmol/l. She was ventilated, and an intradural pressure transducer was inserted to monitor intracranial pressure. She was initially treated by machine driven haemofiltration with a Gambro FH77 haemofilter and a 17 litre exchange of fluid. Her intracranial pressure increased from 20 mmHg to 36 mmHg during the first hour of treatment despite bolus mannitol and then decreased to 19 mmHg after further mannitol.

The next day she was treated by continuous arteriovenous haemofiltration; there was no change in intracranial pressure during the procedure. On each of the following two days she received machine driven haemofiltration, and on both occasions her intracranial pressure increased during the first hour. Despite mannitol the intracranial pressure increased during treatment. She was then treated by continuous arteriovenous haemofiltration, and once again no change was noted in intracranial pressure (figure). After five days her urine output improved, and after a further week continuous arteriovenous haemofiltration was stopped. She made a rapid recovery and was discharged home with no residual clinical effects.



Changes in mean intracranial pressure during machine haemofiltration and continuous arteriovenous haemofiltration and maximum sustained intracranial pressure during first hour of treatment. ▼ = Points at which mannitol was given.

Comment

Evidence from the use of extracorporeal membranes in other studies is relevant to these findings. Patients who have chronic renal failure and are undergoing regular haemodialysis have been shown to develop cerebral oedema, and patients who have acute renal failure complicated by an increased intracranial pressure may deteriorate rapidly during haemodialysis.⁵ An increase in intracranial pressure has been reported in patients who have grade IV hepatic coma treated by polyacrylonitrile membrane haemodialysis.

It has been suggested that haemofiltration, by minimising systemic osmotic gradients, would be a better approach than haemodialysis and would diminish both the incidence and severity of cerebral oedema. In this case continuous arteriovenous haemofiltration, with an ultrafiltration rate of 1000 ml/hour, caused less change in intracranial pressure than machine driven haemofiltration, with a 17 litre exchange of fluid in three to four hours. Continuous arteriovenous haemofiltration may therefore have an advantage in the treatment of patients who have hepatic encephalopathy complicated by acute renal failure.

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Departments of Renal Medicine, Medicine, and Anaesthetics, St James's University Hospital, Leeds LS9 7TF

A DAVENPORT, MA, MRCP, research registrar in renal medicine

E J WILL, BM, MRCP, consultant nephrologist

M S LOSOWSKY, MD, FRCP, professor of medicine

S SWINDELLS, BSC, FFARCS, consultant anaesthetist

Correspondence to: Dr Davenport.