Charcoal Regeneration of Dialysate during Haemodialysis of Butobarbitone Intoxication in Dogs

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Yatzidis (1964) introduced charcoal in haemoperfusion to treat endogenous and exogenous intoxications. Twiss (1966) combined charcoal adsorption with haemodialysis. In the treatment of uremic patients he added charcoal to the recirculating dialysate in order to keep the concentration of various dialyzed substances low, thus imitating the single-pass dialysis system. This method, however, is not very satisfactory as urea and phosphate are poorly adsorbed to activated charcoal, while glucose and calcium are readily adsorbed. Regeneration of dialysate, however, would be very desirable because it would permit us to use a small dialysate reservoir and a small volume of dialysate. Activated charcoal adsorbs many substances, among them barbiturates. In the case of barbiturate intoxication the only substance to be removed is the barbiturate itself.

We constructed a calibrated cylindrical dialysate reservoir. The total volume was about 3 litres (Figure 1); 280 grams of charcoal was added to the 2 litres of dialysate. In in vitro experiments we found that this amount of charcoal kept the concentration of butobarbitone below 1 mg/100 ml during the dialysis of 20 litres of ox blood with an initial butobarbitone concentration of 20 mg/100 ml, during 8 hours.

The dialysate had the usual chemical composition, but an additional correction for glucose and calcium concentrations had to be made to cope with the adsorption to charcoal. The initial concentration of glucose was 400 mg/100 ml, in order to reach a final concentration of 110 mg/100 ml. The initial calcium concentration was 20 mg/100 ml, which owing to the adsorption fell to 6.2 mg/100 ml.

To investigate the usefulness of haemodialysis in butobarbitone intoxication and the effect of charcoal regeneration of dialysate we performed two series of six experiments on dogs intoxicated with butobarbitone.

In the first series the treatment consisted of rapid infusion only (method
of forced diuresis), while in the other series a 6-hour period of haemodialysis was included, superimposed on the forced-diuresis regimen.

Four dogs were used in both experiments, but the interval between the experiments was at least three weeks. It is known that repeated exposure to barbiturates results in accelerated breakdown owing to enzyme induction. For this reason the dialysis experiment was always done first in those dogs used in both experiments; in this way any influence of this accelerated breakdown would favour the results in the experiments with the forced diuresis only.

In both groups the dogs had a mean weight of 22 kg and were intoxicated orally with butobarbitone in a dosage of 100 mg/kg. Four hours after the intoxication infusion was started with a slightly alkaline solution of sodium chloride, sodium bicarbonate, potassium chloride and glucose. The rate of
infusion was 7 ml/kg/hr. In the middle of every 6-hour period of observation an intravenous injection of furosemide was given in a dosage of 0.3 mg/kg. This resulted in a forced diuresis.

The first group was treated with a forced diuresis regimen only. In the second group dialysis was started after 6 hours of forced diuresis. We dialyzed with only one layer of a conventional Kil dialyzer to make the results approximately comparable with those that might be obtained in human beings. The period of dialysis was 6 hours. In the meantime the treatment of forced diuresis was continued.

Ultrafiltration could easily be estimated in the calibrated dialysate container because of the small volume of the container and the loss of plasma water was corrected by administration of an extra amount of the infused fluid. Forced diuresis was continued after the dialysis period for at least 6 hours.

Figure 2 shows the duration of coma which was defined as the period between intoxication and the first active movements. The duration of coma in the dialyzed group was significantly shorter in comparison to the other group.

In Figure 3 the butobarbitone levels are shown, the thick line representing the mean of the six experiments. It is evident that the dialysis contributed much to a rapid fall in the level of butobarbitone, but after dialysis a certain re-equilibration between the blood and tissues occurred, sometimes resulting in a post-dialysis rise in the butobarbitone level.

Many dogs regained consciousness during dialysis and the experiment had to be completed under light inhalation anaesthesia.

In the dialysate butobarbitone levels never reached the 1 mg/100 ml level, thus simulating a single-pass dialysis for this substance.

CONCLUSION
1. Haemodialysis for butobarbitone poisoning in dogs significantly shortened duration of the coma in comparison with an identical group of dogs, treated with
Figure 3. Thick line represents the mean of six experiments

the method of forced diuresis only.

2. Regeneration of dialysate with activated charcoal permits the use of a small recirculating volume of dialysate as adsorption of the butobarbitone in the dialysate is practically complete, thus imitating the effect of single pass dialysis for this substance.

3. During dialysis forced diuresis could be maintained owing to the quantitative replacement of the ultra-filtrated plasmawater that could accurately be estimated in the calibrated cylindrical dialysate reservoir.

REFERENCES
