period of anuria. 48 hours later only 230 ml urine had been passed, and two intramuscular injections of frusemide 20 mg were given 18 hours apart, resulting in a diuresis of 1200 ml.

Four hours after the second dose of frusemide, while he was asleep leaning forwards over his left thigh, with his left leg bent up beneath him, a circulatory change was observed in the left leg. There was purplish discoloration extending to midcalf, the skin was cold, and no pulses were palpable below the femoral. He was treated with continuous intravenous heparin for 48 hours, followed by phenindione for 4 weeks. No further deterioration in the leg circulation occurred, and during convalescence, though the left leg was colder than the right, the collateral circulation opened up and all pulses were palpable, but of poor volume. As he became more mobile intermittent claudication developed. Pain recurred in the left calf after running 25 metres and he had to sit down until it had worn off. This symptom has improved but not disappeared and occasional pain has occurred in the calf at night. The nephrotic syndrome relapsed after an initial course of prednisolone, but eventually went into remission with tetracosactrin (Synacthen) and cyclophosphamide. Temporary hypertension occurred during the use of tetracosactrin. Renal biopsy which was deferred until he was normotensive, showed glomerulonephritis with predominantly mesangial proliferation and few capsular adhesions.

Both hypercoagulability and hypovolaemia occur in the nephrotic syndrome, and the blood volume may fall still further during diuretic therapy, and also on standing (Garnett and Webber, 1967). Frusemide gives a maximum diuresis of 14-30 ml/min, which is equivalent to 10 to 20% of the glomerular filtration rate. The duration of action of a single dose is 2 to 6 hours (Robson et al., 1964). In this case frusemide had been given 4 hours before the first signs of occlusion and had already produced a diuresis of 500 ml. High doses have been recommended in the nephrotic syndrome and considered to be safe (Snashall, 1971). This may not be so, if steroids, which also increase coagulability (Ozsoyolu, Strauss, and Diamond, 1962) are already being given. Use of the thromboplastin generation test before the use of diuretics might be useful to predict those susceptible to acute arterial occlusion, particularly if steroids are used (Mukherjee et al., 1970).

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Artificial Grunting in Respiratory Distress Syndrome

Sir,

Infants suffering from idiopathic respiratory distress syndrome (RDS) show the grunting type of respiration illustrated in the Fig. It has been shown that grunting is an effective means of raising arterial oxygen tension ($P_{aO_2}$) in RDS (Harrison, Heese, and Klein, 1968), and the ability to simulate this pattern of respiration during mechanical ventilation might therefore be useful in the treatment of RDS.

We have modified a simple ventilator to produce this pattern, which Gregory et al. (1971) would describe as continuous positive pressure breathing (CPPB), in the treatment of infants with RDS.

The East–Radcliffe ventilator* has been adapted for use in the neonate to produce intermittent positive pressure ventilation (IPPV) (Tunstall et al., 1968). It was further modified to produce CPPB by the addition of a gate-clamp to the expiratory tubing distal to the 'dummy lung'. With this arrangement the pressure changes may be monitored continuously on the manometer of the ventilator itself. The infant can then be supplied via a nasotracheal tube with oxygen-enriched heated, humidified air at any preselected pressure.

In the Fig., pressure-volume tracings obtained during artificial grunting are compared with tracings obtained during IPPV. During IPPV, airway pressure falls to zero midway through the ventilator cycle because of negligible resistance during the expiratory phase, possibly allowing further alveolar collapse to take place. With CPPB on the other hand there is continuing positive pressure during the expiratory phase, producing volume changes similar to grunting in RDS but with positive airway pressure remaining at the end of each cycle. This presumably increases the infant's functional residual capacity and helps to prevent alveolar collapse.

When CPPB is applied as described, the ventilator minute volume is considerably reduced and may even

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become less than the minute volume of a normal infant, with consequent risk of carbon dioxide retention. However, the flow rates during CPPB are not sufficiently low to result in reduced gas temperature to the infant even with the same humidifier temperatures as those used in IPPV.

It is therefore easy to perform CPPB with the East-Radcliffe ventilator, producing a grunting pattern similar to that observed in the natural course of RDS. It has been our impression that this type of ventilatory assistance leads to a rapid improvement in hypoxaemia with a less dramatic effect on respiratory acidosis, but this impression is based on the study of only four cases and further investigations are being undertaken to define the role of CPPB in the management of RDS.

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