premature baby from a small maternity hospital to a nearby larger one, ostensibly to receive a high level of vigilant care, when in practice one baby being ventilated there is diverting attention and limited resources away from the other babies.

The emergence of different patterns of neonatal referral will influence neonatal survival rates and the incidence of long-term handicap reported from individual referral units. The success of intensive care methods in improving neonatal survival is not disputed and is illustrated by the pronounced fall in the early neonatal mortality of babies born in St Mary's since 1976 when mechanical ventilation was introduced on a wide scale. The optimistic long-term prognosis in heterogeneous groups of low-birth-weight babies reported since the introduction of improved neonatal care,⁷⁻⁹ however, is not applicable to critically ill babies with respiratory failure transferred to a regional ICU.^{10 11}

Caution is necessary before advocating the emergency transfer to another maternity hospital of women who develop complications of pregnancy. Our experience of babies referred with severe hyaline membrane disease, however, makes us speculate that when caesarean section is contemplated before term and fetal pulmonary maturation is uncertain or suggestive that hyaline membrane disease will occur women ought to be transferred for delivery to a maternity hospital with the appropriate facilities for definitive mechanical ventilation of neonates.

We conclude that the desirable number and siting of referral ICUs in each region depend on the nature of the referral pattern that is envisaged. Our experience in the North-west suggests that a pattern of referral orientated towards impending or actual respiratory failure is feasible but that it requires close collaboration with referring SCBUs and parallel development of their facilities and skills. A complementary but distinct pattern of referral is necessary to cater for high-risk but well babies born

in hospitals that lack the appropriate staff and facilities to provide the vigilant care that is so necessary.

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In My Own Time

Treatment of acute renal failure with artificial kidneys

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Today's patient with acute renal failure has a choice of two effective treatments. Peritoneal dialysis with one- or two-litre bags and a Trocath is relatively safe and can be performed in many district general hospitals. It is even safer when carried out by trained nurses using automated equipment. Even so, it is the more uncomfortable treatment of the two and the one I would avoid for myself if I had a choice.

The alternative is haemodialysis in a renal unit. Soon after admission the patient has a Teflon-Silastic shunt inserted in his forearm, or a Seldinger catheter placed in his subclavian vein with reasonable certainty that it will stay patent as long as it is

needed. The consultant prescribes daily or alternate-day dialysis according to the catabolic rate. An efficient well-monitored machine, prepared by a skilled technician, is wheeled to the bedside; it has a double-pump attachment if the subclavian vein catheter is used, rendering a return line unnecessary. A trained nurse connects the patient and monitors the procedure. The sterile, pyrogen-free, disposable dialyser will remove as much fluid by ultrafiltration as she requires, allowing the patient a reasonable fluid intake of up to two litres a day and making total intravenous feeding possible. Between his four-hour dialyses, if his primary disease permits, the patient sits out of bed, eats at a table, and watches television in the day room.

Bad dreams of the past

It was not always so. In the 1940s and early '50s the conservative treatment of acute renal failure was a grim affair, and included strict fluid restriction that led to intolerable thirst. Patients would climb out of bed, trailing their tubes behind

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them, to sneak an illicit drink from the wash-hand basin; we sometimes had to screw down the taps to prevent unintended suicide by water intoxication. Normal food was withheld, and energy supplied as sickly carbohydrate solutions. For a brief period "Bull's milk" (peanut oil, dextrose, and emulsifier) was mixed with strained vomitus and fed down a nasogastric tube. Patients with more benign forms of acute renal failure—such as crystalluria or postpartum tubular necrosis—often recovered, but looked back on their fortnight in the unit as a bad dream. That dream was made more frightening in the early 1960s, when staphylococcal cross-infection was at its zenith, and we had to cocoon patients in barrier nursing cubicles. Several emerged believing that they had been nursed in a submarine or space craft.

The artificial kidney did not change the scene overnight. It had many false starts. Haas gave it a brief clinical trial in 1926, but was thwarted by lack of a suitable anticoagulant. Kolff deserves full credit for persisting against formidable odds in war-torn Holland and for selling his idea to the world, but it took time. The artificial kidney he gave to Hammersmith after the war was used to treat only 12 patients before being consigned to the basement; those he distributed to Holland, Poland, and Canada fared no better. Although there were other pioneers, including Darmady at Portsmouth, it was not until 1953, when Swan and Merrill published their classic paper on the use of an improved Kolff kidney, that it began to gain acceptance.

By 1958, Parsons at Leeds and Shackman at Hammersmith had confirmed Merrill's results and there was an obvious need for artificial kidneys in Britain's regional centres. Newcastle got in early, and paid the price of innovation. (Sir) Ralph Jackson at RAF Halton was already using Kolff's new tank with disposable coils, but it seemed wildly extravagant—those who feel hard up in 1979 have forgotten the Health Service of 20 years ago (or were not there). So, John Anderson was dispatched to Sweden to learn from Nils Alwall. Working independently, Alwall had started haemodialysis soon after Kolff, and his static coil seemed to offer some advantages over the complicated rotating coil. There was no independent witness to warn us that things are not always what they seem.

John returned in a month with a silver-one-star in haemodialysis, and a set of packing cases. As they were unpacked, it transpired that there was a patient in the hospital with acute-on-chronic renal failure who might benefit from dialysis. The machine was rapidly assembled, if you can call such a process "rapid." The dialyser consisted of two concentric drums, carved from solid stainless steel. The inner drum was fixed to the wall, and Cellophane tubing was wound on (fig 1). Now

FIG 2—Outer drum being put into position.

came the crunch: the outer drum was slid into position (fig 2). If the axis was a millimetre out the tubing was snagged as surely as nylon tights pulled over badly trimmed toenails. The snag was often unnoticed until the double drum had been winched into its bath of water and pressure tested with air. Then, if it passed the test, it was sterilised by inserting a metal pipe from the hospital steam supply. The condensing steam made a noise like the dying agonies of a rhinoceros, and great clouds of vapour billowed up from the cauldron. Nurses in rubber aprons and "wellies" flitted in and out like the witches in *Macbeth*. Paint peeled in strips from the walls of that basement cubicle ("the Black Hole") that was to be our temporary quarters for the next 10 years.

On that first occasion, the drums were wound again and again, and each time there was a telltale stream of bubbles. As the first day drew to a close, the workers (John Anderson, Walter Elliott, and Molly Hill (now Hall)) decided there were irregularities in the metal that were puncturing the tubing. The dental department was opened up, and its whole stock of dental burs was used to smooth off real or imaginary spicules of metal. Late the next day the machine was finally assembled intact, and the patient prepared. Cannulas were inserted in the forearm. We made them by heating PVC tubing in liquid paraffin and drawing them out like Pasteur pipettes. The smell of charring paraffin, and the sad sight of a bin full of discarded cannulas—like the trunks of thalidomide elephants—live with me to this day. By now, a crowd had gathered. Senior consultants in evening dress offered helpful advice. The Sigma pump was switched on. "It's running well," said (Sir) George Smart, feeling the pulsating tubing. "Then why are the red cells separating from the plasma?" asked Walter Elliott. Then it dawned on the bleary-eyed workers that they had forgotten to inject the heparin; it took only 10 minutes for the anticoagulant in donor blood to dialyse out. The patient was wheeled back to the ward, where she promptly had a diuresis. She was full of praise for her doctors and their new wonder machine.

Over the next year we used the Alwall kidney with increasing success but were beset by a problem that had plagued the artificial kidney from the first human dialysis in Germany—pyrogen reactions. You could set your watch by them. An hour after the start the patient would complain of *angor animi* (if he

FIG 1—Winding Cellophane over drum.

knew the words), chest pain, feeling cold; his blood pressure would plummet and his temperature soar as he was wracked with rigors. By the end of the dialysis (10 hours in those days), the fever had passed, he felt battered and washed out but was otherwise none the worse for his experience. The next day, only a spreading facial herpes reminded us of what he had been through.

Like so many others since then, we made the mistake of looking first at the bath water. Pyrogens are large molecules which should be (and probably are) excluded by cellulose membranes, and the reactions occur at a time when the bacterial count is low. Of course the open recirculation tank of 1959 bred a rich flora and fauna of bacteria, and one cannot commend dialysis against pyocyanus soup—the smell alone is enough to discourage it—but eliminating the bacteria did not stop the reactions. Eventually, they were traced (with Frank Parsons's help) to the nylon connectors we were then obliged to reuse, and which became pyrogenic after autoclaving. Ever since, I have preached to my colleagues (usually in vain): "If you encounter pyrogen reactions, look first at the blood pathway; that is where we have always found the culprit."

Much of the progress in technology that has transformed the treatment of acute renal failure, and which is at long last reducing its mortality to a more respectable figure, has come from industry. It dates from the explosive growth in regular dialysis for chronic renal failure from the late 1960s onwards, when the market became large enough to justify research and development. Before that, we chafed at the slow pace. It took Travenol 10 years to introduce the first improvement in the twin-coil kidney, and several more years passed before any firm introduced a rival that was not a straight copy. As I look back over that period I recall its frustrations and disappointments. Inefficient artificial kidneys did not control uraemia adequately, and infection ran riot. Our first Christmas was spent treating a young man who had celebrated his 21st birthday too well and walked in front of a bus. His haemopneumothorax became an empyema, and the staphylococci were impervious to the antibiotics to which they succumbed *in vitro*. We used daily dialysis for the first time, but for 10 days we could not keep his predialysis plasma urea below 67 mmol/l. He died of septicaemia on New Year's Day—just as his diuresis began—reducing the staff to tears.

Ingenuity and team spirit

Nevertheless, I remember just as vividly some benefits gained. It was a period of great camaraderie. We look back to it in the same wistful way that veterans of North Africa recall the second world war. Two staff nurses volunteered for the job. Sheila was slim and brunette, Pauline slim and blonde—running an artificial kidney unit kept you slim. They were indestructible. When a dialysis ended at 2 am (about par for the course), we would don aprons, hose the floors, rebuild the dialyser, and tidy up; there was no one to tell them it was not a nursing duty. The nurse who should have been off at 8 30 pm always stayed to the end and if you offered her a lift home said, "no thanks, the party in Framlington should still be on." This total contempt for working hours, days off, and weekends was a little unusual—even in 1959.

It was a period of great ingenuity. What you could not buy, you made. Canoe paddles, converted saucepans, Hoover motors, industrial pressure gauges, and pressure hosing were our stock in trade. The first great step in dialyser design in a decade was made when Hoeltzenbein mended the upholstery in his Volkswagen. He found some plastic mesh, which he recognised as a potential membrane support. It is still in use. We believed that candidates for a consultant post in a renal unit should be interviewed in dungarees, spanner in hand. Our impatience with the defects of early artificial kidneys gave birth to bio-engineering in this university and several others.

There is no need for today's nephrologist to acquire such

skills, but I hope they could emerge again in some developing specialty. I am not optimistic, however, because all activity is now subordinated to the worship of safety. The structure of our hospitals and universities has been defaced beyond belief to ward off fire risks that were often too low to calculate. If the high priest (alias fire officer) speaks, any expense or act of vandalism is accepted without demur. This destruction of anatomy will soon be dwarfed by the disruption of function caused by the Health and Safety at Work Act. The improvisations that saw us through the first 10 years of haemodialysis would be inconceivable in this era of defensive medicine. Most units can afford to wait for ideal equipment to arrive and for the AHA to buy it, but, if the disease is uncommon and the market small, few new techniques can hope to progress as fast as haemodialysis. It is already a familiar problem with new drugs.

We acquired other skills. A small renal laboratory was set up to serve the new unit and, although its *raison d'être* was soon gone, it has survived with the acquiescence—if not the enthusiastic support—of our colleagues in clinical biochemistry. It was a phenomenon reproduced in other centres and reflected in the higher medical training programme for nephrology. Generations of young nephrologists have learnt the rudiments of biochemistry there, and have acquired an understanding of laboratory error and an appreciation of the cost of investigations that they could never experience with a centralised service. We discovered a level of personal service that is the envy of the hospital. The SHO can nip around 20 patients and drop their labelled specimens on "the girls round the corner" knowing that within two hours he will receive their completed flow sheets in time to decide on the day's dialyses. There is no form filling for the doctor—which has saved us the cost of one SHO (seldom included in the planner's sums).

Small laboratories are now anathema to the planners. It will be difficult to keep them going and ours will soon be merged with the hospital service, but not for the usually quoted reasons. Staff satisfaction is high, and the cost of automated equipment and economies of scale are offset by other savings, but we have run into the problem highlighted by Zuckerman—poor career prospects for MLSOs in a cul-de-sac. I hope someone can overcome this while keeping alive the close contact between ward and laboratory which has contributed so much to the development of nephrology.

What might be the cause of a 35-year-old woman growing in size? Her feet have lengthened—but not widened—from a 5½ to a 6½ shoe fitting.

One investigation is certainly merited—that is, the plasma growth hormone response to hyperglycaemia. Although the patient does not yet show the classic features of acromegaly, the history suggests overgrowth of the soft tissues of the feet, which is probably the result of enhanced growth hormone production. A random growth hormone concentration is unlikely to be conclusive as growth hormone levels show considerable fluctuations during the day and growth hormone production is increased by stress whether this is physical, psychological, or metabolic. The growth hormone response to a glucose load should establish the diagnosis.

Correction

High altitude pulmonary oedema

The first sentence of this Any Question (14 July, p 115) should have read "High altitude pulmonary oedema is caused by a too rapid ascent."