characteristics of the high pressure bladder, removing even a small (<100 ml) volume of urine cannot prevent a rapid drop in intravesical pressure with consequent brisk onset of the diuretic phase, which may precipitate some bleeding from either lower or upper urinary tract. We find it impossible to regulate the decompression process with enough precision in the ward and prefer instead to concentrate on the problem of the often mild but occasionally severe salt losing state that sets in after the obstruction has been removed.

Operation is generally by transurethral resection. Bimanual examination under anaesthesia may be very misleading in these patients, as the prostatic capsule may be extremely muscular (greater than 1 cm thick), deceiving the operator and making enucleation difficult if an open procedure were to be chosen.

Postoperatively careful monitoring of weight, blood pressure (erect and supine), and electrolyte values is required. It must be emphasised that diuresis induced dehydration may ensue with great rapidity in these patients, and a rising urea or creatinine concentration after operation is as likely to be due to this cause as to the readily blamed "residual obstruction." Depending on the nephron damage, such losses may continue for many days postoperatively, though not necessarily to such a severe degree. Before discharge an estimation of creatinine clearance will allow nephron recovery to be followed with precision. Follow up of our patients at one year showed that initial clinical progress was being well maintained.

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SHORT REPORTS

Measurement of respiratory rate in the newborn

Infants who appear normal at birth are transferred to postnatal wards where twice daily measurements of temperature are usually the only regular nursing observations. During the past 10 years more infants have been admitted to postnatal wards instead of special care baby units to avoid separation from their mothers. We found that there was no satisfactory method of observing these babies when four apparently normal infants were found dead in their cots in one postnatal ward nursery between October and December 1976. All these deaths occurred in the early hours of the morning and the nursing staff were terrified of starting the night shift for fear of a further death. No cause of death was found despite extensive investigation of the circumstances and postmortem examinations. At the time Lancefield group B streptococci were being recognised as a cause of rapidly progressive disease in our unit.[1] As it is the most common pathogen in the newborn and usually causes a raised respiratory rate[2] we decided to assess the value of regular measurements of respiratory rate to detect occult disease.

Patients, methods, and results

From January 1977 the respiratory rates of all infants in the postnatal wards have been recorded hourly for the first 12 hours and two hourly for the subsequent 24 hours. Initially the respiratory rate was counted by observation, which was not reliable, or by the nurse placing her hand on the baby's chest, which disturbed the infant. A new method of detecting respiration was therefore developed,[3] in which an air filled capsule was taped to the abdominal wall so that expansion of the abdomen compressed it and generated a pneumatic signal. This signal was carried along a flexible tube to a small (12×5×2 cm) hand held box in which it was converted to an electrical and then audible and visible signal. The sensor capsules were very simple and cheap, so that one could be attached to each baby shortly after birth, allowing the end of the tube to protrude from the cot. Each ward was supplied with a box, which could be carried round by a nurse and connected to each baby in turn for counting the respiratory rate. Analysis of the respiratory rates in 54 infants admitted consecutively to one postnatal ward showed that the highest rate (mean 48±SD 6/min) was at the age of 3 hours. Infants with a respiratory rate above 60/min persisting for more than one hour were transferred to the special care unit.

During February 1981 to February 1982, 2789 infants were admitted to the postnatal wards, and 29 of them were transferred to the special care unit with a raised respiratory rate (table). Four had evidence of colonisation or infection with group B streptococci and one had heart failure due to hypoplastic left heart syndrome. Nine had a raised respiratory rate for only a few hours and were given no treatment, but the remaining 19 had a raised respiratory rate for a considerable period and received antibiotics.

Comment

Use of the new respiratory rate detector has shown that about 1% of apparently normal infants in the postnatal wards have a raised respiratory rate and that in most of them it persists for a long period. Most of the 19 infants without reported evidence of group B streptococcal infection probably had delay in lung fluid absorption. No investigation is helpful in confirming this diagnosis and the respiratory rate falls to normal spontaneously. Despite the negative reports some of the 19 infants may have had group B streptococcal infection, as routine laboratory methods do not identify all cultures of this organism.

Group B streptococcal infection has a mortality rate between 20 and 50%[1] and early treatment is the most important factor in prognosis.[4] It has been suggested that the mortality from this infection can be reduced only by giving antibiotics prophylactically to all infants in a unit.[5] We consider that it is preferable to select a group of infants for

Details of infants with raised respiratory rates who were transferred from postnatal wards to special care unit

Case No	Birth weight (g)	Weeks of gestation	Age (hours) when raised respiratory rate noted	Duration (hours) of raised respiratory rate	Group B streptococci cultured from*: Mother	Group B streptococci cultured from*: Infant	Treatment
1	2680	39	3	6	0	0	Nil
2	3500	40	1½	5	0	0	Nil
3	2800	39	3	35	0	Neg	Nil
4	2820	38	2½	10	0	0	Nil
5	3760	40	2	72	0	Neg	Antibiotic
6	3100	40	8	6	Neg	0	Nil
7	2980	40	6	120	0	Neg	Antibiotic
8	3480	41	29	15	Pos	Pos	Antibiotic
9	3260	41	26	19	Neg	Neg	Nil
10	2980	41	32	3	0	Neg	Nil
11	3700	41	12½	136	0	Neg	Antibiotic
12	3820	40	4	23	0	Pos	Antibiotic
13	3240	37	3½	8	Neg	0	Nil
14	3150	38	9	55	0	Neg	Antibiotic
15	4110	39	28	35	0	Pos	Antibiotic
16	3280	40	5	27	0	Neg	Antibiotic
17	3140	40	8	72	0	Neg	Antibiotic
18	3880	41	5	48	0	Pos	Antibiotic
19	2700	39	23	29	0	Neg	Antibiotic
20	3420	40	14	30	Neg	Neg	Antibiotic
21	3660	41	5½	72	0	Neg	Antibiotic
22	3010	39	12	50	0	Neg	Antibiotic
23	3860	40	24	168	0	Neg	Antibiotic
24	3530	41	22½	96	Neg	Neg	Antibiotic
25	3410	40	24	†	Neg	Neg	Antibiotic
26	2480	36	2	17	Neg	Neg	Antibiotic
27	3480	39	8½	17	Neg	Neg	Antibiotic
28	2950	38	4	7	0	Neg	Nil
29	2800	34	2	66	0	Neg	Antibiotic

* 0 = No investigation.
† Hypoplastic left heart syndrome.

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close observation and to give them antibiotics only on clinical indications.

Since 1976 no infants have been found dead or moribund in the postnatal wards. Measurement of the respiratory rate in the way described is the only effective method of observing an infant next to his mother in a dark postnatal ward. It has been acceptable to the nurses and reassuring to the mothers.

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Use of local anaesthetic drugs in hospital practice

Major adverse reactions have been produced by intravenous regional anaesthesia,[1 2] and three deaths have been reported in the national press.[3] We report the results of an inquiry into the use of local anaesthetic drugs by a cross section of junior hospital doctors.

Method

A questionnaire comprising seven questions was answered by 57 junior doctors below the grade of senior registrar, drawn from many specialties and trained in various hospitals. The questions were presented to the doctors in the course of one afternoon and all those approached answered the questions.

The doctors were first asked when they had last used a local anaesthetic and for what purpose. Further questions sought to find which drug had been used, its concentration, and route of administration. Finally, the doctors were asked to indicate the recommended maximum safe dose of plain lignocaine in milligrams per kilogram body weight and to state the number of milligrams per millilitre of the drug in a 1% solution.

Results and comment

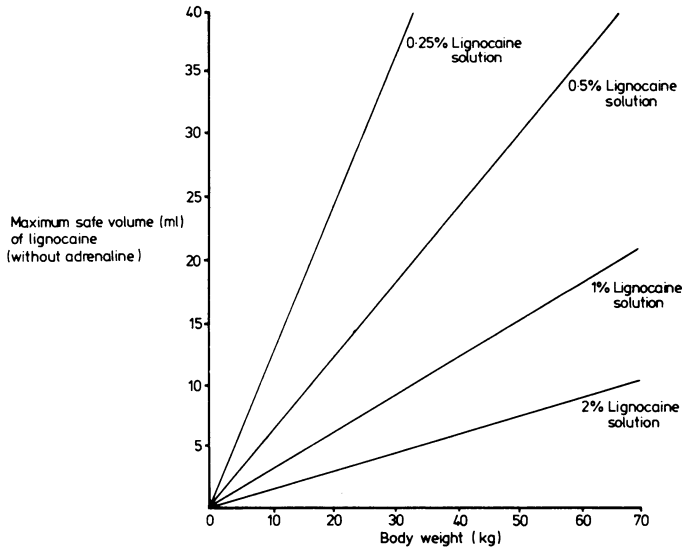
The results provide some insight into the way local anaesthetic drugs are used in this hospital. Of the doctors questioned, 59·6% had used a local anaesthetic drug during the previous week and 80·6% within the previous month. Only one doctor had not used a local anaesthetic within the past year. Lignocaine was the most frequently used drug (by 94·6% of respondents). The majority (64·3%) used lignocaine by local infiltration for minor surgical procedures. The second most common mode of administration was topical (30·3% of respondents)—mainly for use in bladder catheterisation, bronchoscopy, eye surgery (0·5% proxymetacaine), and ear, nose, and throat procedures (cocaine).

Comment

Although 0·5% lignocaine is an adequate concentration for local infiltration,[4] 31 out of 36 doctors used a higher concentration. Four out of 16 doctors used topical lignocaine without knowing its concentration and 68·4% did not know the maximum recommended dose of lignocaine for infiltration; 71·4% did not know how to calculate the dose they were giving.

Although many doctors claimed to know the safe dose of lignocaine (in terms of volume of a known percentage solution) for a man weighing 70 kg, there are many instances where more accurate calculations of the dose must be made—for example, when used in the elderly and children. The results are perhaps not surprising since information regarding the correct dose of local anaesthetic is often presented in a way that requires calculations to be made.[5]

We have designed a simple chart (figure) whereby the correct maximum volume of lignocaine solution can be easily calculated from the patient's body weight and the percentage solution to be used. It also shows graphically the undoubted advantage of using the most dilute solution that will effectively produce analgesia.



Maximum safe volume of plain lignocaine according to body weight. (Maximum safe dose of lignocaine—without adrenaline 3 mg/kg, with adrenaline 7 mg/kg; uses of different solutions—0·25-0·5% for infiltration and intravenous regional anaesthesia, 1% for nerve blocks, epidural anaesthesia, and intravenous regional anaesthesia, 2% for nerve blocks.)

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Overdose of trilostane

Trilostane is a new blocker of synthesis of glucocorticoids. We report the first recorded case of trilostane overdose.

Case report

A 29 year old woman was admitted after ingesting lorazepam 50 mg, 40 tablets of a combination diuretic (each containing hydrochlorothiazide 50 mg and amiloride 5 mg), and 60 trilostane capsules (each containing 60 mg). She was taking trilostane for premenstrual tension, which was thought to be a component of her long standing depression. There was no history of hypertension or renal impairment. On examination she was drowsy but

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