

13. Mitri F, Hofmeyr GJ, Van Gelderen CJ. Meconium during labour: self-medication and other associations. *S Afr Med J* 1983; **71**: 431-433.
14. Grant A. Monitoring the fetus during labour. In: Chalmers I, Enkin M, Keirse MJNC, eds. *Effective Care in Pregnancy and Childbirth*. Vol. 2. Oxford: Oxford University Press, 1989: 846-882.
15. Suzuki A, Hashino M, Chiba H, et al. Correlation between the levels of catecholamines (noradrenaline, adrenaline) and adrenal steroids (DHA-S, cortisol) in maternal and fetal blood during pregnancy and labour. *Nippon Naibumpi Gakkai Zasshi* 1989; **65**: 704-714.
16. Schneider H, Progler M, Ziegler WH, Huch R. Biochemical changes in the mother and the fetus during labour and its significance for the management of the second stage. *Int J Gynaecol Obstet* 1990; **31**: 117-126.
17. Lagercrantz H, Bistoletti P. Catecholamine release in the newborn infant at birth. *Pediatr Res* 1973; **11**: 889.
18. Stern L, Lees MH, Leduc J. Environmental temperature, oxygen consumption and catecholamine excretion in newborn infants. *Pediatrics* 1965; **36**: 369.
19. Walters DV, Walters RE. The role of catecholamines in lung liquid absorption at birth. *Pediatr Res* 1978; **12**: 239.
20. Mendelson CR, Boggaram V. Hormonal and developmental regulation of pulmonary surfactant synthesis in fetal lung. *Baillieres Clin Endocrinol Metab* 1990; **4**: 351-378.
21. Anand KJ, Hansen DD, Hickey PR. Hormonal-metabolic stress responses in neonates undergoing cardiac surgery. *Anesthesiology* 1990; **73**: 661-670.
22. Padbury JF, Roberman B, Oddie TH, Hobel CJ, Fisher DA. Fetal catecholamine release in response to labour and delivery. *Obstet Gynecol* 1982; **60**: 607-611.
23. Gillman AG, Goodman LS, Rall TW, Murad F, eds. *The Pharmacologic Basis of Therapeutics*. 7th ed. New York: Macmillan, 1985: 155.

Persistent pulmonary hypertension of the neonate in a developing country — does extracorporeal membrane oxygenation have a role to play?

J. SMITH, G. F. KIRSTEN

Abstract A retrospective study was undertaken of survival after conventional management of 35 infants suffering from persistent pulmonary hypertension of the neonate (PPHN). The outcome of infants weighing more than 2 000 g and who also qualified for extracorporeal membrane oxygenation (ECMO) therapy on the grounds of published criteria was assessed.

The admission incidence of patients with PPHN was 1,1%. Secondary PPHN was more common than primary. The overall survival rate of 69% in this study reflects the trend in recently reported improved survival rates of infants with PPHN, treated with conventional techniques. Sixteen of 28 infants weighing more than 2 000 g qualified for ECMO therapy; 4 of them died. Had ECMO been available as an alternative mode of therapy, only 2 of the 4 might have been saved. The other 2 were considered to have conditions incompatible with a normal quality of life. We therefore assessed the requirement for ECMO in our population to be approximately 0,6/1 000 live births. Although ECMO may be promising, the introduction of this technique in developing countries should rather be delayed until more substantial data refute this. Because PPHN could be related to a potential preventable cause in almost 80% of cases, we propose the support of more cost-effective strategies such as continuing obstetric and perinatal education programmes.

S Afr Med J 1993; **83**: 742-745.

Persistent pulmonary hypertension of the neonate (PPHN) is a syndrome in which severe hypoxaemia and right-to-left shunting of blood through

the foramen ovale and/or ductus arteriosus occur in neonates without recognisable cardiac abnormalities. It is often a complication of acute or chronic perinatal asphyxia, meconium aspiration syndrome, severe hyaline membrane disease, hyperviscosity, lung hypoplasia syndromes, and β -haemolytic streptococcal and other pneumonias.¹⁻³ It is primarily a disease of full-term and post-term infants and is associated with high morbidity and mortality rates. Despite aggressive management with mechanical hyperventilation, plasma volume expanders and vasopressor drugs, the mortality rate of PPHN reportedly varies between 34% and 60% (average 40%).^{1,4-6} Alternative modes of therapy, such as high-frequency oscillatory ventilation and extracorporeal membrane oxygenation (ECMO), have been suggested to improve the outcome of neonates whose respiratory failure is refractory to conventional mechanical ventilation management.⁷⁻⁹ ECMO entails the use of a modified cardiopulmonary bypass circuit to supply temporary support and lung rest for near-term infants with respiratory failure.

ECMO, however, is costly and labour-intensive, the benefits are controversial, and it has as yet not been used in developing countries. It is also clear from the reported experience of some centres that the survival rate without ECMO is equally good.¹⁰⁻¹²

In this report we describe the incidence, management and short-term outcome of infants with PPHN who were mechanically ventilated, and discuss the possible role of ECMO in a developing country.

Patients and methods

The records of all neonates with PPHN who were admitted to the neonatal intensive care unit (NICU) of Tygerberg Hospital between June 1986 and October 1990 (53 months) were analysed retrospectively.

PPHN was diagnosed if a clinical suspicion of the condition existed based on the presence of severe, labile hypoxaemia disproportionate to the severity of pulmonary disease (confirmed by a positive response to hyperoxia-hyperventilation) and/or echocardiography.³ Babies with a congenital diaphragmatic hernia or congenital heart lesions were excluded.

Department of Paediatrics and Child Health, Division of Neonatology, Tygerberg Hospital and University of Stellenbosch, Parowvallei, CP

J. SMITH, M. MED. (PAED.)

G. F. KIRSTEN, M. MED. (PAED.), F.C.P. (S.A.)

Each baby's chart was reviewed and the severity of respiratory failure assessed for the first 10 hours after initiation of conventional mechanical ventilation. The alveolar-arterial oxygen difference (AaDO₂) (kPa), and arterial alveolar oxygen ratio (a/APO₂) were calculated (reflecting the degree of compromise in oxygenation) as well as the oxygenation index (OI) and ventilatory index (VI 1) (reflecting the severity of respiratory compromise and the amount of ventilatory support required, respectively).^{13,14} These indices were also calculated to evaluate whether patients who met the criteria for ECMO^{3,7,8} would have benefited from that therapy, and to assist in the prediction of outcome, i.e. survival or death. Entry criteria for ECMO were similar to those used in former studies for infants weighing more than 2 000 g, with minor modifications.^{3,7,8,10} To qualify for ECMO, infants had to fulfil one or more of the following criteria: (i) average arterial alveolar oxygen ratio $\leq 0,1$; and/or (ii) mean peak inspiratory pressure (PIP) ≥ 30 cm H₂O (mean airway pressure (MAP) ≥ 15 cm H₂O) over the first 10 hours of mechanical ventilation; (iii) air leaks (pulmonary interstitial emphysema or pneumothorax); and (iv) an AaDO₂ > 80 kPa for > 4 hours.

Ventilatory and medical management

During the study period the objective of mechanical ventilation in infants with PPHN was to select the ventilator settings necessary to achieve a 'critical' arterial carbon dioxide tension (PaCO₂) which would reverse the right-to-left shunt and ensure an acceptable PaO₂ response. Infants were intubated and ventilated with a conventional time-cycled, pressure-limited infant ventilator (Sechrist, model IV 100B, Sechrist, Calif.). The infants were initially ventilated at high rates (85 - 110/min) to try to achieve the 'critical' PaCO₂ at the lowest possible inflating pressure. If this approach failed the ventilation rates were decreased and the inflating pressure increased. Throughout, we tried to maintain the positive end-expiratory pressure below 6 cm H₂O. The fractional concentration of inspired oxygen was adjusted according to the preductal O₂ saturation, and was kept between 86% and 94%. In all infants the inspiratory time was $\leq 0,45$ seconds. A continuous infusion of sodium bicarbonate (4,2%) at a rate of 1 mmol/kg/h was administered to infants where a pH $\geq 7,42$ could not be achieved by ventilation alone. Tolazoline infusion was administered via a peripheral vein in a bolus of 2 mg/kg followed by a continuous infusion of 0,3 - 1 mg/kg/h, when the inflating pressure equalled or exceeded 35 cm H₂O, and the alkaline infusion failed to reverse the pulmonary arterial pressure. The response to this drug was monitored continuously by means of a transcutaneous PaO₂ electrode (Novamatrix Medical Systems Inc.) and/or pulse-oximeter (OHMEDA BIOX 3700 pulse oximeter, BOC Health Care). Prophylactic oral antacid therapy was administered to infants receiving tolazoline infusion.

The infants were sedated with intermittent intravenous morphine sulphate (0,1 mg/kg) or bolus doses of fentanyl (2 μ g/kg). Only 1 of the patients was paralysed. The mean arterial blood pressure was continuously monitored and kept $\geq 40 - 45$ mmHg by means of continuous cardio-inotropic support (dopamine, 5 - 20 μ g/kg/min) and bolus infusions of either stabilised human serum or fresh-frozen plasma (10 - 20 ml/kg). An attitude of 'minimal' handling was adopted for all the infants. Infants were aggressively weaned from the ventilator as they improved clinically and PaCO₂ levels between 7 kPa and 9 kPa accompanied by a pH $\geq 7,26$ according.

Statistics

Means and standard deviations were computed for each of the variables and the differences in numerical data between groups compared using the two-tailed Student's *t*-test. Proportion was compared with the chi-square test; in cases of small numbers, Fisher's exact test was used. Statistical significance was accepted at $P < 0,05$. The values in the tables are expressed as the mean of the mean, unless stated otherwise.

Results

Thirty-five neonates with PPHN were evaluated retrospectively. Their mean gestational age was $36,9 \pm 3,5$ weeks at birth with a mean birth weight of $2,67 \pm 0,88$ kg. Diagnosis was confirmed by the presence of hyperoxia-hyperventilation in 43% (15/35), preductal-postductal PaO₂ difference of > 2 kPa in 11% (4/35), and by means of echocardiography in 40% (14/35) of the infants. The associated diagnoses of the study population are shown in Table I. The infants comprised 1,1% of the total admissions to the NICU and represented an incidence in our population of 1:1 600 live births during the study period. Their clinical characteristics are displayed in Tables II and III. Comparison of the clinical, ventilatory and laboratory findings of the survivors v. the non-survivors (Table IV) showed significant differences in the mean PaO₂ ($P = 0,02$), pH ($P = 0,009$) and a/A ratio ($P = 0,03$) over 10 hours, and highest pH achieved ($P = 0,01$), first pH ($P = 0,04$), and first PaCO₂ ($P = 0,02$). Surviving infants were mechanically ventilated for 5 ± 3 days and received supplemental oxygen for an additional 6 ± 4 days.

TABLE I.
Characteristics of associated conditions in the infants with PPHN

	No.	%
Meconium aspiration syndrome	12	34,3
Asphyxia (no lung disease)	4	11,4
Hyaline membrane disease/pneumonia	7	20
Septicaemia		
Group B streptococci	3	8,6
Unidentified organism	1	2,9
Pulmonary hypoplasia	1	2,9
Idiopathic pulmonary haemorrhage	1	2,9
Rhesus incompatibility	2	5,5
Transient tachypnoea	3	8,6
Twin-twin transfusion syndrome	1	2,9
Total	35	100

TABLE II.
Clinical characteristics of the 35 infants with PPHN

	Mean \pm SD	Range
Gender (M/F)	18:17	
Birth weight (kg)	$2,7 \pm 0,88$	0,89 - 4,64
Gestational age (wks)	$36,9 \pm 3,5$	28 - 42
Apgar (5 min)	$6,9 \pm 2,5$	2 - 10
Air leaks (pneumothorax)	9 (26%)	
No. on drug therapy (%)		
Tolazoline	19 (54%)	
Sodium bicarbonate (weight for volume 4,2%)	18 (51%)	
Combination therapy	13 (37%)	
Inborn/outborn	23:12	
Mortality	11 (31%)	
Caesarean section	15 (43%)	

TABLE III.
Ventilatory characteristics of the 35 infants with PPHN during the first 10 hours after initiation of ventilation (mean \pm SD)

		Range
F _{IO₂}	0,93 \pm 0,09	0,66 - 1,0
PIP (cm H ₂ O)	29 \pm 7,5	19 - 46
PEEP (cm H ₂ O)	4,7 \pm 1,3	2 - 9
MAP (cm H ₂ O)	15,8 \pm 4,3	7 - 27
Ventilation rate (/min)	78,6 \pm 11,6	40 - 100
IPPV (d)	4,8 \pm 3,2	1 - 14
PIP (highest value)	35,8 \pm 10,1	20 - 60
Ti (s)	0,34 \pm 0,03	0,29 - 0,4

F_{IO₂} = fractional concentration of inspired oxygen; PEEP = positive end expiratory pressure; IPPV = intermittent positive pressure ventilation; Ti = inspiratory time.

TABLE IV.
Profile of survivors v. non-survivors

	Survivors	Non-survivors	P-value
	(N = 24)	(N = 11)	
	Mean \pm SD		
Birth weight (g)	2 829 \pm 790	2 334 \pm 1 060	NS
GA (wks)	37,4 \pm 3,0	35,7 \pm 4,2	NS
Apgar (5 min)	7,4 \pm 2,4	5,8 \pm 2,7	NS
Pao ₂ (kPa)			
(mean for 10 h)	10 \pm 3,5	7 \pm 3	0,02
First Paco ₂ (kPa)	6,2 \pm 1,9	8,0 \pm 2,8	0,02
Paco ₂			
(mean for 10 h)	5,2 \pm 1,0	6 \pm 2	NS
Lowest Paco ₂ (kPa)	3,3 \pm 0,8	3,9 \pm 1,5	NS
pH (first)	7,20 \pm 0,19	7,05 \pm 0,18	0,04
pH (mean pH over 10 hours)	7,31 \pm 0,1	7,18 \pm 0,16	0,009
Highest pH	7,49 \pm 0,1	7,36 \pm 0,16	0,01
VI 1	2 396 \pm 737	2 460 \pm 992	NS
AaDo ₂ (kPa)	72 \pm 9,5	74 \pm 12	NS
a/APO ₂	0,13 \pm 0,05	0,09 \pm 0,05	0,03
OI	24 \pm 14	36 \pm 27	NS

GA = gestational age.

Twelve infants were extra-uterine transferrals from peripheral (level II) nurseries. Differences between the inborn and outborn infants were only found for gestational age ($P = 0,04$) and birth weight ($P = 0,02$) (Table V). Seventeen infants (48,5%) were of a gestational age above 37 weeks and 14 (82%) of them had either meconium aspiration syndrome ($N = 12$) or asphyxia neonatorum ($N = 2$).

TABLE V.
Characteristics of the inborn infants v. outborn infants (mean \pm SD)

	Infants		P-value
	Inborn	Outborn	
	(N = 23)	(N = 12)	
Birth weight (g)	2 431 \pm 918	3 139 \pm 675	0,02
GA (wks)	36 \pm 3,9	38,5 \pm 1,7	0,04
Apgar 5 min	6,5 \pm 2,5	7,5 \pm 2,7	NS
Survival	15 (65%)	9 (75%)	NS
MAS	6/23	6/12	NS
a/APO ₂	0,12 \pm 0,05	0,11 \pm 0,06	NS

MAS = meconium aspiration syndrome.

Tolazoline was administered to 19 infants (54%) and resulted in improved oxygenation (positive response) in 47% of cases. Air leaks (pneumothorax) developed in 26% of the infants.

The best markers for a very poor outcome were a single a/APO₂ value \leq 0,05 ($N = 3$) (100% mortality) or an OI value \geq 60 ($N = 3$) (100% mortality). Twenty-

eight infants had a birth weight $>$ 2,0 kg and 16 of them were eligible for ECMO on the grounds of fulfilling the entry criteria. Of the abovementioned 16 infants, 4 (25%) died. Their diagnoses included severe lung hypoplasia with renal dysplasia ($N = 1$), meconium aspiration syndrome ($N = 1$), β -haemolytic *Streptococcus* ($N = 1$) and severe refractory asphyxia neonatorum ($N = 1$). Excluding the last-mentioned infant (who also had multi-organ failure and was assessed as having a condition incompatible with a normal quality of life) only 2 infants of this group of infants who succumbed, might ultimately have been saved by means of ECMO (i.e. the 1 infant with meconium aspiration syndrome and the 1 with β -haemolytic *Streptococcus* infection), had it been available.

Discussion

PPHN secondary to meconium aspiration syndrome and asphyxia is still relatively common in developing countries such as South Africa. This is partly due to factors such as no or poor antenatal attendance at clinics, socio-economic factors, logistic problems such as transport, and incorrect assessment and resuscitation of distressed infants by attending physicians and/or nursing staff. Until these conditions can be prevented or rectified, it is important for paediatricians to be familiar with the diagnosis and treatment of PPHN. To improve survival rates, these infants must be managed where specific technology and experience exist. In South Africa these facilities are mostly limited to the tertiary institutions, as well as some large provincial and private hospitals.

At Tygerberg Hospital the treatment of infants with PPHN focuses on mechanical ventilation, as other alternatives such as ECMO and high-frequency oscillatory ventilation are either too expensive or unobtainable. With this approach the survival rate of 69% in the present study compares well with those from similar studies reported by Hageman *et al.*⁶ (71%) and Bifano and Pfannenstiel¹⁵ (72%). Potential problems related to aggressive mechanical ventilation include acute and chronic lung injury and the effects of hypocarbia on cerebral blood flow.¹⁵⁻¹⁷ The incidence of pneumothorax in this study (26%) is lower than that reported by Fox¹⁸ ($>$ 50%), Hageman *et al.*⁶ (35%), Wung *et al.*¹⁰ (40%) and Kohelet *et al.*⁷ (46%). The incidence of bronchopulmonary dysplasia among the survivors in the present study was 3% while 1 infant developed a wheezy chest after the neonatal period. The abovementioned low frequency of lung sequelae reflects our unit's conservative approach to positive pressure ventilation, where higher Paco₂ values (5,4 \pm 1,2 kPa) are regarded as acceptable during the acute stages of PPHN, and infants are aggressively weaned during the transitional phase of their disease. To achieve this, transcutaneous Pao₂ and Paco₂ monitors are continuously utilised.

This study also showed that non-survivors spent significantly more time poorly oxygenated ($P = 0,02$) and acidotic ($P = 0,009$) compared with the survivors. This is probably a reflection of more severe PPHN in the non-survivors with increased right-to-left shunting (a/APO₂ ratio difference significant, $P = 0,03$).

The development of PPHN in the present study could be related to a potentially preventable predisposing factor in 80% of the enrolled infants. Meconium aspiration syndrome was the primary diagnosis in 12 infants (34%) and remains one of the principal, preventable causative factors in PPHN. Of great concern is the nearly doubled incidence of meconium aspiration syndrome among outborn infants (50% v. the 26% incidence ($P = NS$) in the inborn infants). Since the introduction of ECMO by Bartlett *et al.*⁹ in 1976, more than

3 000 term or near-term infants have been treated with this form of therapy, resulting in a survival rate of almost 83%. ECMO, however, is not benign or inexpensive, as the morbidity and incidence of permanent neurological injury in patients who undergo ECMO are substantial. Approximately 10 - 15% of neonates may die and 10 - 30% of surviving infants have an adverse neuro-developmental outcome.^{17,19} The technique of ECMO is labour-intensive and requires the constant attention of an experienced physician assisted by trained technologists and nursing staff.

In conclusion, our data reveal a PPHN incidence of 1,1% of admissions to the NICU, with meconium aspiration syndrome (34%) the leading causative factor. The survival rate of 69% achieved in the present study with conventional techniques reflects the trend of improved survival reported recently.²⁰ Had ECMO been available as an alternative mode of therapy at Tygerberg Hospital, only 2 additional infants might have been saved (requirement for ECMO: \pm 0,6/1 000 live births). It seems that ECMO has little to offer to improve survival of infants with PPHN in our situation, and that in a developing country such as South Africa, it would be of more value to focus on the improvement of obstetric and perinatal education programmes.²¹

REFERENCES

1. Gersony WM, Duc GV, Sinclair JC. 'PFC' syndrome (persistence of the fetal circulation). *Circulation* 1969; **40**: 111.
2. Fox WW, Duara S. Persistent pulmonary hypertension in the neonate: diagnosis and management. *J Pediatr* 1983; **103**: 505-508.
3. Dworetz AR, Moya FR, Sabo B, Gladstone I, Gross I. Survival of infants with persistent pulmonary hypertension without extracorporeal membrane oxygenation. *Pediatrics* 1989; **84**: 1-6.
4. Davis JM, Spitzer AR, Cox C, Fox WW. Predicting survival in infants with persistent pulmonary hypertension of the newborn. *Pediatr Pulmonol* 1988; **5**: 6-9.
5. Abu-Osba YK. Treatment of persistent pulmonary hypertension of the newborn: update. *Arch Dis Child* 1991; **66**: 74-77.
6. Hageman JR, Adams MA, Gardner TH. Persistent pulmonary hypertension of the newborn. Trends in incidence, diagnosis and management. *Am J Dis Child* 1984; **138**: 592-595.
7. Kohelet D, Perlman M, Kirpalani H, Hanna G, Koren G. High-frequency oscillation in the rescue of infants with persistent pulmonary hypertension. *Crit Care Med* 1988; **16**: 510-516.
8. O'Rourke PP, Crone RK, Vacanti JP, et al. Extracorporeal membrane oxygenation and conventional medical therapy in neonates with persistent pulmonary hypertension of the newborn: a prospective randomized study. *Pediatrics* 1989; **84**: 957-963.
9. Barlett RH, Gazzaniga AD, Jefferies MR, et al. Extracorporeal membrane oxygenation (ECMO) in infancy. *Trans Am Soc Artif Intern Organs* 1976; **22**: 80-93.
10. Wung J-T, James LS, Kilchevsky E, James E. Management of infants with severe respiratory failure and persistence of the fetal circulation, without hyperventilation. *Pediatrics* 1985; **76**: 488-494.
11. Greenough A, Emery E. ECMO and outcome of mechanical ventilation in infants of birthweight over 2 kg. *Lancet* 1990; **336**: 760.
12. Elliot SJ. Neonatal extracorporeal membrane oxygenation: how not to assess novel technologies. *Lancet* 1991; **337**: 476-478.
13. Horbar JD. A calculator program for determining indices of neonatal respiratory distress syndrome severity. *Am J Perinatol* 1987; **4**: 20-23.
14. Ortega M, Ramos AD, Platzker ACG, Atkinson JB, Bowman CM. Early prediction of ultimate outcome in newborn infants with severe respiratory failure. *J Pediatr* 1988; **113**: 744-747.
15. Bifano EM, Pfannenstiel A. Duration of hyperventilation and outcome in infants with persistent pulmonary hypertension. *Pediatrics* 1988; **81**: 657-661.
16. Bancalari E, Gerhardt T. Bronchopulmonary dysplasia. *Pediatr Clin North Am* 1986; **33**: 1-23.
17. Brett C, Dekle M, Leonard CH, et al. Developmental follow-up of hyperventilated neonates: Preliminary observations. *Pediatrics* 1981; **68**: 588-591.
18. Fox WW. Arterial blood gas evaluation and mechanical ventilation in the management of persistent pulmonary hypertension of the neonate. In: Peckham G, Heymann M, eds. *Cardiovascular Sequelae of the Newborn*. Columbus, Ohio: Ross Laboratories, 1982: 102-110.
19. Schumacher RE, Palmer TW, Roloff DW, LaClaire PA, Bartlett RA. Follow-up of infants treated with extracorporeal membrane oxygenation for newborn respiratory failure. *Pediatrics* 1991; **87**: 451-457.
20. Weigel TJ, Hageman JR. National survey of diagnosis and management of persistent pulmonary hypertension of the newborn. *J Perinatol* 1990; **10**: 369-375.
21. Woods D. Assessing the perinatal education programme. *Proceedings of the 11th Conference on Priorities in Perinatal Care in South Africa*. Department of Paediatrics, University of Cape Town, 1992.

Selective posterior lumbosacral rhizotomy for the management of cerebral palsy spasticity

A 10-year experience

J. C. PETER, L. J. ARENS

Abstract One hundred and sixty-eight patients had selective lumbosacral posterior rhizotomies for the treatment of cerebral palsy spasticity at Red Cross War Memorial Children's Hospital and Groote Schuur Hospital during the 10-year period 1981 - 1991. There was no mortality and insignificant early postoperative morbidity. Long-term follow-up on 110 patients has revealed satisfactory tone reduction in 95% of cases. The majority showed improvement in standing, sitting and locomotion.

Thirteen patients had minor persistent sensory disturbances and 20% have developed asymptomatic spondylolysis or grade I spondylolisthesis. Most therapists, patients and parents remain enthusiastic about the results of this procedure.

S Afr Med J 1993; **83**: 745-747.

Department of Paediatric Neurosurgery and Institute of Child Health, Red Cross War Memorial Children's Hospital and University of Cape Town

J. C. PETER, M.B. CH.B., F.R.C.S.
L. J. ARENS, B.S.C., M.B. CH.B.

Selective posterior lumbosacral rhizotomy has been successfully used in the management of spasticity for almost 100 years. Since Otrid Foerster's¹ comprehensive description of its use in the treatment of cerebral palsy, many refinements have been introduced to make the procedure more specific for spasticity.^{2,4} The selection of nerve roots by electrical stimulation helps preserve sensory fibres and consequently minimises some of the sequelae that were a worry in the past.^{2,3,5}

Accepted 10 Sept 1992.
Reprint requests: Dr J. C. Peter, Dept of Paediatric Neurosurgery, Institute of Child Health, Red Cross Children's Hospital, Rondebosch, 7700 RSA.