provided with the appropriate bag of tricks. Equipped with a conceptual framework, on the other hand, they can apply it to almost any situation.

The teaching of research methodology is an essential component of any programme in the behavioural sciences, but those available as instructors often teach it at a level inappropriate for students who will be future practitioners. The typical medical student is unlikely to do original research; if he does, he will have ample opportunity to consult with professional methodologists in matters of research design. But every medical student will, for the rest of his life, read the professional literature. Hence, he needs to understand methodology from the point of view of the consumer of research and not the producer. He needs to know the right questions to ask of a piece of finished research, not how to design a complex study.

Similarly, the typical student is unlikely to become a professional epidemiologist. What he needs to know as a practitioner is the differential exposure to risk suffered by various segments of the population and the relationships between certain disease categories and the demographic characteristics of the victims. What he needs to know as a member of his community is a rank-ordering of diseases that can be reduced or eliminated through modification of the environment and the ways in which the criteria of cost-benefit or cost-effectiveness can be applied to various public health programmes.

SOME IMPLICATIONS FOR STAFFING

Although this paper has openly criticized some current programmes in Australian medical schools, the criticism has focused on their substance and their relevance, not on the intentions or the competence of those administering or executing them. Indeed, given the serious shortage of thoroughly trained personnel and adequate Australian data, one can express nothing but admiration for the ingenuity and perseverance with which some programmes are being carried out. But programmes that can be evaluated as "excellent, given the circumstances" may fall far short of the high standards of excellence characteristic of many of the clinical areas in Australian medical schools. And there is danger that present programmes may crystallize into the system simply because better programmes seem unfeasible.

The fundamental problem is, of course, staffing. The few well-trained, empirically oriented behavioural scientists that Australia currently is producing are not gravitating to medical schools or to institutions in which they can train the future generations of competent professionals. And they are producing neither the demographic data nor the text materials that are essential to an adequate programme. One solution to this problem may lie in a cooperative effort among the several medical schools to attract a cadre of behavioural science faculty members who, if distance precluded their teaching on several campuses, could at least assemble text materials, organize curricula, initiate the gathering of useful demographic and epidemiological data, and supplement the training of local faculty members at each school. Such a scheme need not produce a rigid uniformity among schools; it can, however, provide an economy of scale necessary in a situation of manpower shortage. Over the long term, such a cadre could set up postgraduate programmes to train young behavioural scientists in the teaching of medical and paramedical students and to train clinicians who become interested in research involving behavioural factors.

Over the short term, however, any solution must include the importation from abroad of well-trained, empirically oriented individuals who are committed to teaching in a medical and paramedical context. Such individuals are not available in great numbers anywhere in the world, but there are few industrialized countries in which they are in as short supply as in Australia. The fact that such a recommendation is very difficult to carry out should not influence the reader's assessment of its validity.

Preliminary Communications

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TREATMENT OF PARAPLEGIC SHEEP WITH HYPERBARIC OXYGEN

The results of the use of hyperbaric oxygen therapy to control the onset of paraplegia after recent spinal cord injury in sheep are described. This preliminary report suggests that hyperbaric oxygen therapy instituted within two hours of the injury will result in improved motor recovery.

THE REVERSIBILITY of graded spinal cord trauma in the experimental animal after three exposures to 100% oxygen at three atmospheres absolute pressure was first reported by Hartzog, Fisher and Snow.[1] The therapy was given three, 11 and 21 hours after the injury, with each treatment of 45 minutes' duration. Significant clinical improvement occurred and the neurological status remained stable for one month.

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Kelly *et alii*[2] reported significant recovery of function in injured dogs treated with hyperbaric oxygen. The degree of recovery was equal to that after treatment with glucocorticoids, intrathecal injection of methyl prednisolone and hypothermia.

These results appeared encouraging and further trials with hyperbaric oxygen were warranted with sheep as the experimental model. The recovery of motor power after a controlled experimental contusion injury to the spinal cord of the treated animal is reported. The experimental technique has been described in other papers.[3 4 5]

METHODS AND MATERIALS

Under general anaesthesia (sodium pentobarbitone, 20 mg/kg/hr, given intravenously), nine animals were subjected to a controlled injury from a 50 g weight dropped vertically within a Teflon tube, 20 cm in diameter, on to the exposed spinal cord at the T10 level. With the dura mater removed, the degree of contusion of the spinal cord was photographed.

Within 30 minutes, the wounds were closed and four animals transferred to the hyperbaric chamber provided by the Royal Australian Navy. Each animal was taken to a depth of 66 feet (pressure, three

atmospheres) over a five-minute period. Oxygen was administered to the animals through an intratracheal tube at a rate of 14 l./min. Within the chamber at three atmospheres pressure, the reduced oxygen flow was estimated at 8 l./min. Hyperbaric oxygen therapy was maintained for 90 minutes and the animal then allowed to ascend from this depth over a ten-minute period. One animal was maintained at three atmospheres for a period of only 60 minutes. After removal from the chamber, each animal was examined to assess the degree of hind limb paralysis and loss of pain sensation below the T10 spinal level.

The four treated animals and the five controls survived for eight weeks, and during this period were nursed in special slings to support the trunk and hind limbs and to prevent trophic skin ulceration. The neurogenic bladder emptied within 24 hours by reflex detrusor contraction, so that there was no necessity for catheterization. The animals were examined on the day following injury and then three times a week for a period of eight weeks. The motor recovery in both hind limbs was recorded and graded as follows: 0, absent; 1, movements against gravity; 2, movements against resistance; 3, normal. Movements above and below the femorotibial joint were recorded separately and the scores then added to give a total score for each hind limb. Thus normal motor function for the complete hind limb would be scored as 6 units (that is, 3+3). The recovery in the control (untreated) animals was compared with the recovery found in the treated animals.

After eight weeks, autopsies were performed on all animals and sections of the spinal cords were taken 1 cm above, 1 cm below and at the level of the injury (T10). Sections were stained by haematoxylin-eosin, and Luxol fast blue with van Gieson stains. The histopathology is to be reported in a separate communication.

RESULTS

Over eight weeks, improved clinical recovery was evident in paraplegic sheep treated with hyperbaric oxygen within two hours of injury (Figure 1). The mean motor function recovery in the treated animals was always greater than in the control group. A statistical analysis of the results was not possible because of the scoring system used for recording motor recovery.

The results showed a degree of improvement similar to that reported when paraplegic sheep were treated with alpha methyl paratyrosine, an inhibitor of tyrosine hydroxylase.[5]

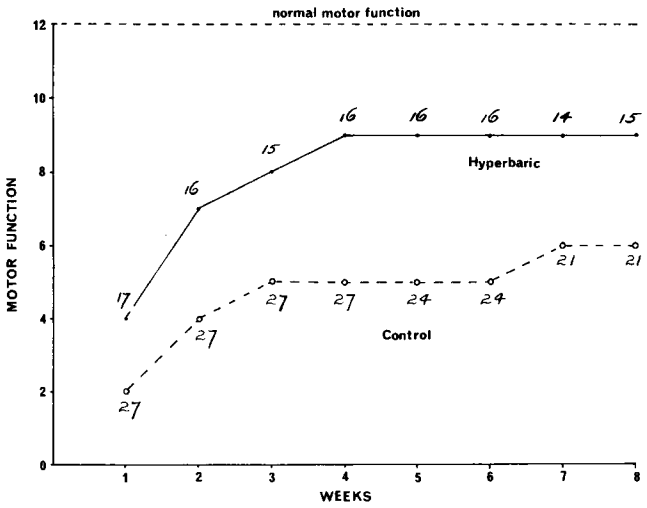

FIGURE 1: The mean recovery of motor function in control and hyperbaric treated sheep. The numbers indicate the number of clinical examinations made during each week for both groups of animals.

DISCUSSION

Madea[6] reported persistent tissue hypoxia occurring for 72 hours after neurological damage. Tissue hypoxia may be related more to tissue oedema than to impaired tissue diffusion of oxygen and ischaemia from secondary vasospasm. However, accumulation of spinal cord tissue lactic acid has been reported after circulatory arrest in primates.[7] This accumulation is probably the result of circulatory deprivation with inadequate tissue perfusion and oxygenation. Using monkeys, Locke *et alii*[8] found that lactic acid accumulation in the injured spinal cord tissue supported the concept that ischaemia plays a role early in the traumatic processes which follow spinal cord injury. Lactic acid levels remained elevated for 18 hours after the injury.

Hyperbaric oxygen therapy increases the available tissue oxygen[9] and decreases tissue oedema; whether cerebral vasoconstriction also occurs is debatable.[10] Ducker and Perot[11] showed that relative hypoxia occurred within two or three hours of injury, and was a local phenomenon occurring adjacent to the site of the cord injury. However, the tissue Po_2, both one centimetre above and one centimetre below the site of the impact was almost normal. The level of maximum Po_2 disturbance was therefore associated with the region of maximum cord swelling from congestion, extravasation and intracellular and extracellular oedema.[5]

Kelly *et alii*,[2] demonstrated that tissue Po_2 in the spinal cord of normal dogs can be modified by ventilating the animals with oxygen and carbogen. They reported that: (i) the tissue Po_2 responded only to hyperbaric oxygen; (ii) paraplegic animals treated with hyperbaric oxygen recovered to a greater degree than those in the untreated control group. Over a period of four months they claimed that most improvement occurred in: (i) those animals treated with hyperbaric oxygen four hours after injury; (ii) those animals given multiple treatments one hour, 24 hours and 48 hours after injury.

This study supports the results reported by Hartzog *et alii*[1] and Kelly *et alii*.[2] The significant degree of motor recovery is similar to that previously described when paraplegic sheep were treated with alpha methyl paratyrosine.[5] Inhibiting the accumulation of norepinephrine appears to enhance clinical recovery and reduce further destruction of neuronal tissue at the site of the cord injury. Osterholm and Mathews[12] suggest that the release of norepinephrine around neuronal fibres adjacent to the damaged cord tissue causes further tissue damage. This effect could result from vasoconstriction of microvasculature and ischaemia.

Hyperbaric oxygen provides oxygen to bruised, hypoxic cord tissue. The preliminary results of this experiment suggest that hyperbaric oxygen therapy will produce significant recovery of motor power in paraplegic sheep which have suffered recent contusion injuries. Further animal experiments with hyperbaric oxygen are nearing completion and a therapeutic trial will soon be commenced in patients who have suffered recent spinal cord injury.

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REFERENCES

[1] HARTZOG, J. T., FISHER, R. G., and SNOW, C., Spinal cord trauma effect of hyperbaric oxygen therapy, Proc Annu Clin Spinal Cord Inj Conf., 1969, 17: 70.

[2] KELLY, D. L., LASSITER, K. R. L., VONGSVIVUT, A., and SMITH, J. M., Effects of hyperbaric oxygenation and tissue oxygen studies in experimental paraplegia, *J. Neurosurg.*, 1972, 36: 425.

[3] YEO, J. D., PAYNE, W., COLLINS, L. T., Controlled experimental contusion of the spinal cord of sheep, *Aust. N.Z. J. Surg.*, 1971, 1: 89.

[4] YEO, J. D., PAYNE, W., HINWOOD, B., and KIDMAN, A. D., Experimental contusion injury of the spinal cord in sheep, Proceedings of the Australian Society for Medical Research, *Clin. exp. Pharmacol. Physiol.*, 1975, 2: 78.

[5] YEO, J. D., PAYNE, W., HINWOOD, B., and KIDMAN, A., The experimental contusion injury of the spinal cord in sheep, *Paraplegia*, 1975, 12: 275.

[6] MADEA, N., Experimental studies on the effect of decompression procedures and hyperbaric oxygenation for the treatment of spinal cord injury, *J, Nara med. Ass.*, 1965, 16: 429.

[7] FELDMAN, R. A., YASHON, D., LOCKE, G. E. *et alii*, Lactate accumulation in primate spinal cord during circulatory arrest, *J. Neurosurg.*, 1971, 34: 618.

[8] LOCKE, G. E., YASHON, D., FELDMAN, R. A. *et alii*, Ischaemia in primate spinal cord injury, *J. Neurosurg.*, 1971, 34: 614.

[9] LAMPHIER, E. H., and BROWN, I. W., In *Fundamentals of Hyperbaric Medicine*, National Academy of Science National Research Council, No. 1298, Washington, D.C., 1966: 33.

[10] LEDINGHAM, I., MCDOWELL, D. G., and HARPER, A. M., in *Hyperbaric Medicine*, edited by I. W. Brown, Jr, and B. G. Cox, National Academy of Science National Research Council, No. 1404, Washington, D.C., 1966: 33.

[11] DUCKER, T. B., PEROT, F. L., JR, Spinal cord oxygen and blood flow in trauma, *Surg. Forum*, 1971, 22: 413.

[12] OSTERHOLM, J. L., and MATHEWS, G. J., Altered norepinephrine metabolism following experimental spinal cord injury, Parts I and II, *J. Neurosurg.*, 1972, 36: 386.

Short Papers

Med. J. Aust., 1976, 1: 540–542.

ANTIBODY RESPONSES TO INFLUENZA VIRUS SUBUNIT VACCINE IN THE AGED

Elderly patients were immunized with one dose of subunit influenza virus vaccine, and their antibody responses were compared with those in a group of younger adults. Antibody responses in the aged compared favourably with those in the younger group.

IT has been recorded that over 80% of the influenza-associated deaths occur in patients over 65 years of age.[1] Influenza vaccination has been recommended in this group by medical authorities in Europe, the United States, and in Australia, but few antibody or protection studies have been reported.[2] This study formed part of a larger protection study, but in the absence of epidemic influenza no protection data became available.

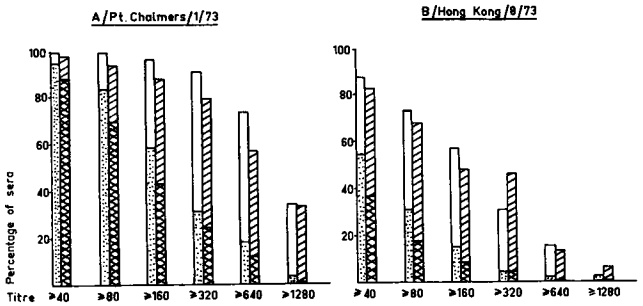

FIGURE 1: Geometric mean haemagglutination-inhibition titres in sera from aged and adult groups. The stippled columns represent titres in the aged group before vaccination; the white columns represent titres in this group after vaccination. The cross-hatched columns represent titres in the adult group before vaccination; the hatched columns represent titres in this group after vaccination.

MATERIALS AND METHODS

Sixty-one ambulant volunteers between 68 and 93 years, with a median age of 81 years, were compared with 52 healthy volunteers aged between 21 and 60 years, with a median age of 35 years. Both groups were given one dose of subunit vaccine containing 16,000 HA units of A/Port Chalmers/1/73 vaccine and 8,000 HA units of B/Hong Kong/8/73 vaccine.

Blood samples were collected before and after vaccination from the participants, and were subjected to haemagglutination-inhibition (HI) titrations by standard laboratory techniques.[3]

Address for reprints: Dr B. J. Feery, Commonwealth Serum Laboratories, 45 Poplar Road, Parkville, Vic. 3052.

TABLE 1

*Results of Haemagglutination Inhibition Tests**

Group Vaccinated	Geometric Mean Antibody Titre*			
	A/Port Chalmers		B/Hong Kong	
	Before Vaccination	After Vaccination	Before Vaccination	After Vaccination
Aged (68 to 93 years) ..	186	708	32	138
Adult (21 to 60 years) ..	105	500	20	110

* Reciprocal of geometric mean antibody titre.

RESULTS

The results of the HI studies are shown in Tables 1 and 2 and in Figure 1. Substantial antibody increases occurred in both groups. Tables 1 and 2 show that the antibody titres before and after vaccination were higher for the A/Port Chalmers strain in the aged group, but approximately equal for the B/Hong Kong strain. Figure 1 demonstrates the range of titres before and after vaccination.

TABLE 2

Proportionate Increase in Antibody Titres

Increase in Titre	A/Port Chalmers		B/Hong Kong	
	Aged	Adult	Aged	Adult
0–2 fold	6	3	8	2
2–4 fold	19	12	18	10
4 fold	36	37	35	40
Total number of patients	61	52	61	52