physical therapy is likely to have its greatest effect it may be possible to deploy more of our limited resources in this area.

We did not deal in detail with recovery after six months but referred to another of our publications where this has been done and it is from that that Drs Knox and Horrocks have derived their figures. They are quite right in reporting that 21% of 92 survivors assessed for mobility at the end of a year showed some improvement between six and 12 months. What they have not indicated, however, is that a closer analysis of these patients showed that most of them were minimally disabled in the first place and in most of them improvement in mobility between six and 12 months lies in the difference between being able to get about in the house and being able to get about outdoors. No doubt in some cases this improvement coincided with discharge from hospital and in others with increasing confidence. Furthermore, these would not have become long-stay patients. Also the 6% of survivors who showed an improvement in the "independence" score did not actually achieve full independence after six months but rather showed some improvement.

We would stress that every patient with stroke must be treated as an individual. We also know that stroke patients who have been untreated or improperly treated during the first six months may show improvement even if their treatment comes only a year or more following their stroke.¹ We would urge, however, that if the main effort of physical therapists were devoted to the first six months this would be the best investment (and incidentally should diminish the proportion of pressure sores). As we stressed, our study does not allow a comparison between comparable groups of patients treated with and without physiotherapy or between Bobath and other methods of treatment and we urge the need for prospective trials to settle these matters.

We are certainly aware of the need for the maintenance of independence once this has been achieved and the maintenance of morale in stroke patients at all levels of disability. We suggest, however, that personnel other than physical therapists might be more involved in those particular functions.

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¹ Lehmann, J F, et al, *Archives of Physical Medicine and Rehabilitation*, 1975, **56**, 375.

Histamine release in exercise-induced asthma

SIR,—In your leading article "Exercise-induced asthma" (27 August 1977, p 536) you discussed the possibility of mediator release. We have demonstrated, for the first time, an increase (from resting levels) of arterial plasma histamine during exercise-induced asthma (EIA). This increase was diminished by a beta-agonist drug at a time when circulating levels of adenosine 3',5'-cyclic phosphate (cyclic AMP) were elevated by the combined effect of the drug and exercise.

Plasma histamine and plasma cyclic AMP concentrations were measured in arterial blood samples taken from an indwelling cannula. Blood was collected at rest, and during and three times after

8-10 min of treadmill running exercise.¹ The peak expiratory flow rate (PEFR) was measured before each collection with a Wright peak flow meter. Six asthmatic patients known to have EIA performed two tests 2 h apart, the first after a placebo aerosol and the second after inhaling 2-3 mg of aerosolised terbutaline sulphate. Histamine was assayed fluorometrically² and cyclic AMP was estimated by a radiosaturation technique.³ All values are expressed as a percentage change from resting levels.

Following exercise on placebo all patients had a significant fall in PEFR (mean $48.7\% \pm 5.2$ SEM, range 34.1-68.7%) and a rise in arterial plasma histamine (mean $105\% \pm 57.0$ SEM, range 26.4-388%). Following the second exercise test (after terbutaline) the fall in PEFR was significantly reduced (mean $10.9\% \pm 2.8$ SEM, range 2.4-20.7%, $P < 0.001$). The rise in histamine was abolished in two patients and diminished in three (mean rise $21.8\% \pm 9.8$ SEM, range 0-59%). Repeated exercise alone did not prevent the release of histamine during EIA. After terbutaline the values for cyclic AMP were significantly greater at rest (mean increase $501\% \pm 217$ SEM, $P < 0.005$) and at the end of exercise (mean increase $47.3\% \pm 18$ SEM, $P < 0.01$) when compared with placebo.

Previous work from this laboratory⁴ suggested that aerosol administration of beta-agonists may inhibit mediator release from lung mast cells in addition to relaxing smooth muscle. Our present results support this duality of action and indicate that the protective effect of terbutaline against EIA may depend on a modification of mediator release associated with increased cyclic AMP. While mediators other than histamine may contribute to EIA, these results show that histamine, a broncho-constrictor agent, increases in arterial plasma after exercise and may be actively released from the lung.

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¹ Anderson, S D, Silverman, M, and Walker, S R, *Thorax*, 1972, **27**, 718.

² Lorenz, W, et al, *Hoppe-Seyler's Zeitschrift für physiologische Chemie*, 1972, **353**, 911.

³ Trembath, P W, and Shaw, J, *British Journal of Clinical Pharmacology*, 1976, **3**, 1001.

⁴ Anderson, S D, et al, *American Review of Respiratory Diseases*, 1976, **114**, 493.

Confusion about New Zealand abortion?

SIR,—Your correspondent from New Zealand, Dr Richard Smith (10 June, p 1533), states that "most doctors were against the [new] law." He offers no support for this opinion. Again he states that "the department of community health at Auckland University has conducted a survey among *all doctors* in New Zealand" (my italics). Did all doctors really receive a questionnaire and were all returned? If so, New Zealand has something for the *Guinness Book of Records*.

Dr Smith might have given us his impression of New Zealand qua abortion with another slant—for example, "New Zealand having viewed the disastrous effects of mass abortion over the last 10 years in the UK has rejected the ethic of disposal of the unborn declared unwanted, preferring to limit abortion to the protection of the health of a mother genuinely threatened by the continuation of pregnancy." This threat must obviously include the mother greatly distressed at the prospect of an imperfect child. For the mother who, though distressed at the risk

or certainty of an abnormal child, declines abortion the Prime Minister's promise to help the handicapped child and the family must be welcome. Surely this speaks of tolerance and compassion.

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Carcinoma of the male breast and oestrogen metabolism

SIR,—Carcinoma of the male breast makes up 1% of all carcinomas of this organ. The important predisposing factors appear to be Klinefelter's syndrome, exogenous oestrogens, and possibly gynecomastia.¹ There is equivocal evidence concerning oestrogen excretion in the male patient with carcinoma of the breast.^{2,3} Much of the work does support an association with increased oestrogen excretion, although some suggests that this increase is only a reflection or a marker of malignant disease.⁴

We have recently seen a patient with Klinefelter's syndrome who had a left radical mastectomy in 1971 for carcinoma of the breast. He continued in good health until 1977, when he presented with a pleural effusion. Further investigations showed he had disseminated disease with bony involvement. Urinary excretion of oestrogen breakdown products was found to be increased. It was therefore decided to start treatment with tamoxifen (an oestrogen antagonist). This produced resolution of the pleural effusion and a 50% decrease in the size of the lesions in the T9 and L3 vertebrae. There was also a decrease in the size of the lytic areas in the ribs.

It is not clear whether there is a genuine predisposition to carcinoma of the male breast with Klinefelter's syndrome. The association may be a reflection of the increased breast tissue mass. The person with Klinefelter's syndrome has about 20% of the breast tissue of a female and therefore it is perhaps not surprising that the incidence of carcinoma approximates to 20% of that of the female.

With the introduction of tamoxifen there is a drug which may be of considerable benefit in carcinoma of the male breast. It is therefore important to discover whether there is a definite relationship between male breast carcinoma and altered oestrogen metabolism.

We are grateful to Dr C L Joiner for allowing us to report this patient under his care.

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¹ Everson, R B, et al, *Lancet*, 1976, **1**, 9.

² Dao, T L, *New England Journal of Medicine*, 1973, **289**, 138.

³ Scheike, O, et al, *Journal of Steroid Biochemistry*, 1973, **4**, 489.

⁴ Sadoff, L, et al, *New England Journal of Medicine*, 1973, **289**, 863.

SIR,—The anti-oestrogen tamoxifen is a useful tool in the treatment of breast carcinoma, particularly in postmenopausal women presenting soft-tissue metastases. Its value in male breast carcinoma is not established.

A 55-year-old male patient was referred to us for multiple subcutaneous metastases of an adenocarcinoma of the right breast diagnosed two years previously by radical modified mastectomy. He was treated for several