periods. Intravenous brinase (100 mg.) increased T cells from 34% to 85%; after ten days, with a fall back to 34% ten days later. A lymphocyte "wash" on the separator increased T cells from 34% to 70% after eight days, but they were back again at 34%, by day 12. There was no clinical improvement. A fuller report will be published later.

MECHANISM OF LACTIC ACIDOSIS

Sir,—I appreciate the comments of Dr Cohen and his colleagues (Aug. 17, p. 405) concerning the relationship between lactate utilisation and the pathogenesis of lactic acidosis. Their suggestion that tissue utilisation of lactate occurs as the acid rather than the anion is reasonable, although I am not aware of evidence which supports that position. Not only may lactate utilisation be associated with hydrogen-ion consumption, but also the oxidation or conversion of lactate to glucose by the liver results in bicarbonate generation; thus, impaired utilisation of lactate could theoretically cause acidosis by both these mechanisms.

My letter (June 29, p. 1351) related specifically to the question of isolated hepatic impairment of lactate utilisation. I doubt whether selective hepatic inhibition of lactate utilisation would result in excess hydrogen ions or a deficit of bicarbonate and systemic lactic acidosis unless the perfusion and/or utilisation of lactate by all tissues was concomitantly impaired. While the kidneys and liver are important in lactate homeostasis the role of skeletal muscle should not be minimised. Lactate utilisation by skeletal muscle may be associated with hydrogen-ion consumption, but also the oxidation or conversion of lactate to glucose by the liver results in bicarbonate generation; thus, impaired utilisation of lactate could theoretically cause acidosis by both these mechanisms. This suggests that the underutilisation of lactate or overproduction of lactic acid by the liver would not produce lactic acidosis or hyperlactataemia due to a concomitant increase in lactate utilisation by other tissues (skeletal muscle). The fact that arterial lactate concentrations do not rise excessively in dogs when hepatic lactate extraction is inhibited by hypoxaemia and hyperperfusion supports such a concept. I do not know whether the above concept would apply in bacteremic shock, but if perfusion of skeletal muscle remained intact I see no reason why it should not. I suspect, however, that the perfusion of all tissues would be reduced in bacteremic shock as it is in other types of low-flow states, thereby impairing the uptake and/or oxidation of lactate with resulting hydrogen-ion accumulation and diminished bicarbonate generation. In patients with clinical lactic acidosis regional measurements indicate that the production of lactic acid is generalised.

Thus, the evidence that Dr Cohen and his colleagues requested supports the contention that isolated impairment of lactate utilisation by the liver does not produce lactic acidosis and that lactic acidosis is predominantly an overproduction acidosis. While underutilisation of lactate may contribute to lactic acidosis when perfusion of all tissues is impaired, I am unaware of any clinical or experimental evidence that supports such a proposal.

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neoplastic cells were accompanied by mixtures of lymphocytes, eosinophils, and occasional plasma cells. "Focal" involvement of lymph-nodes by Hodgkin's disease has been described 2,3; and Lukes 4 comments on the difficulty of classifying the histological type of Hodgkin's disease from such small focal lesions.

The sites of early involvement by lymphomas in lymph-nodes and spleen are of more than theoretical interest. They are of practical importance to the pathologist in the diagnosis of lymphomas, especially in detecting small early foci in tissue removed at staging coeliotomy.

According to the suggestion of Lennert that the first site of infiltration in a lymph-node (and, in this instance, also the first site of infiltration in the spleen) might give an indication of the origin of the lymphoma, a T-cell origin of Hodgkin's disease is favoured.

PHENOLPHTHALEIN IN "ASPIRIN"

Sir,—Not everything in medical textbooks is necessarily correct. The usual teaching about proprietary preparations containing more than one drug is that these are undesirable. One reason is that flexibility of dose is removed. In practice, however, some of these mixtures are quite useful. Many compound mixtures for asthma which include both ephedrine and theophylline may be singularly successful: perhaps the drugs potentiate each other and prevent tachyphylaxis. Also taking one tablet may save the patient the trouble of coping with two or more pills.

In many cases, compounded mixtures seem to be more trouble than they are worth. Recently, one of us observed a new example of this. A 65-year-old man was seen with a fixed drug eruption. He denied taking any phenolphthalein laxatives. The only tablet he was taking was "aspirin". This, however, was found to be a new preparation which contained phenolphthalein and codeine in addition to aspirin. We consider this to be an undesirable mixture of drugs to be taken over long periods. The continued use of irritant purgatives is hazardous and the increasing awareness of this has greatly diminished their use in clinical practice.

The incorporation of phenolphthalein into an analgesic preparation which could be taken over prolonged periods appears to be a retrograde step.

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TRAINING OF NURSES

Sir,—Your review (Aug. 10, p. 323) states baldly about Russian methods "that a system of nurse training which leaves nursing administration and supervision in medical hands" is not preferable to our own system. This would be understandable if our own system were not inefficient with an inherently high wastage. Our system produces some good and some very good nurses, but there are far too many nurses who, during their training or as soon as they qualify, drop out of nursing. The major reason is the hieratic authoritarian system they have met during practice. This, however, was found to be a new preparation which could be taken over prolonged periods, so that this disease is a distinct pathological entity and that the predominant cell is an atypical lymphocyte, most probably of T-cell type. This applies equally to Sezary's syndrome. 6