

beef insulin, but there is no correlation whatsoever to the *mode* of insulin delivery.

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### LECITHIN/SPHINGOMYELIN RATIO IN TRACHEAL ASPIRATE

SIR,—Dr Weller and his colleagues (Jan. 3, p. 12) report on lecithin/sphingomyelin (L/S) ratios in tracheal aspirate in 156 newborn babies. The reference to the L/S ratio in multiple pregnancies is interesting. There were two sets of triplets and one set of quadruplets, all of whose mothers had not been treated with betamethasone before delivery. In all these multiple births, the firstborn showed adequate lung maturity according to the L/S ratio in the tracheal aspirate; this maturity was confirmed clinically. The later babies had much lower L/S ratios. In one of the triple pregnancies and in the quadruple pregnancy all babies except the firstborn died soon after birth. These findings seem to confirm the contribution of stress towards the maturation of fetal lungs; and they may support the mechanism of increased glucocorticoid excretion as a response to the stress associated with vaginal delivery, and its effect towards acceleration of fetal lung maturation.

In our own series (unpublished) we have encountered in multiple births striking differences in the L/S ratio in tracheal aspirate between the firstborn and the rest. This difference was not always encountered in clinical manifestations because the L/S ratio of the later-born infants showed lung maturity, though the ratio was lower than in the firstborn. We have also noted a difference between the L/S ratio in amniotic fluid at the beginning of labour and that obtained in the baby's tracheal aspirate; the L/S ratio in the tracheal aspirate was higher. Similar differences have been observed in single births. This difference may be related to the enrichment in lecithins, originating in the lungs during labour. It may also be compared to the surcharge in lecithins of the aspirate after recovery from respiratory-distress syndrome.<sup>1</sup> The tracheal aspirate may prove to be for the obstetrician and neonatologist a fairly accurate estimate of fetal lung maturity at birth, more so than the L/S ratio in amniotic fluid.

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### ARCHIVE FOR RESEARCH DATA

SIR,—Dr J. K. Cruickshank and I have described some results of a detailed questionnaire survey of medical students at the University of Birmingham.<sup>2</sup> Within a few weeks of publication we received a letter from the Social Science Research Council Survey Archive, at the University of Essex, a body of which I had previously been unaware, asking if we would be willing to deposit a copy of our machine-readable data at the archive. The purpose of this deposition would be to allow secondary analysis of our data by any interested researchers at any time in the future. We readily agreed to this request.

Data from any form of research, especially large questionnaires and population surveys, are acquired at the expense of a large amount of time, effort, and money. Very often far more data are collected than can be analysed by the research team. Later on these data may well be of interest to others. In studies

of long and medium term changes it would be very useful to be able to see the original data of previous workers.

Most large-scale research nowadays involves data in a computer-readable form which can be readily stored on magnetic tape. These data should not be allowed to rest at the bottom of the researcher's drawer, but should be made available to others by a central agency for data collection. The obvious body to create such an archive for medical data would be the Medical Research Council, preferably in conjunction with the S.S.R.C.'s own unit.

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### GLOMERULAR CRESCENT CELLS

SIR,—Dr Atkins and his colleagues (April 17, p. 830), reporting on human glomeruli cultures, noted three types of cells in the outgrowths—type I, morphologically resembling epithelial cells; type II, resembling mesangial cells; and type III, phagocytic cells resembling macrophages. In outgrowths from glomeruli of controls and from patients with non-crescentic glomerulonephritis, cells of types I and II were predominant and type-III cells were rare. In contrast, glomeruli from patients with rapidly progressive (crescentic) glomerulonephritis showed large numbers of type-III cells while type I was rare. Atkins et al. interpreted these results as meaning that crescents are formed mainly by an accumulation of macrophages. This is in contrast to the more commonly held view that crescents arise by proliferation of epithelial cells (visceral, parietal, or both).

The presence of macrophage-like phagocytic cells has been observed by electron microscopy in glomerular crescents<sup>1-3</sup> together with other structural forms of crescent cells (epithelium-like, fibroblast-like, and intermediate forms). The origin of these cells, however, deserves comment. Atkins et al. hold that the crescent is formed by an accumulation of circulating macrophages which later may transform into other types of crescent cells. The participation of extraglomerular cells in crescent formation has previously been suggested<sup>1-3</sup> but the presence of actively phagocytic, mobile cells in the outgrowths from crescents are not in conflict with the traditional view that the crescents are formed mainly by proliferation of epithelial cells. Epithelial cells from normal as well as nephrotic kidneys have phagocytic abilities both *in vivo*<sup>4,5</sup> and *in vitro*.<sup>6,7</sup> These potentials might be stimulated by some pathogenetic factor active in crescent glomerulonephritis and cause the proliferating epithelial cells to assume a macrophage-like structure and function. Further changes might also be induced *in vitro*. It seems probable that such changes take place in view of the large proportion of macrophages in the tissue culture as compared to the more heterogeneous cellular composition of crescents seen by electron microscopy in renal biopsies.<sup>2,3</sup>

The results of Atkins et al. clearly demonstrate the presence of a special phagocytic cell type in crescentic glomeruli incubated *in vitro*, but its origin is not unequivocally established. In our opinion the classical theory about the epithelial origin of glomerular crescents has still neither been proven nor disproven.

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