the relevance of the analogy and argue that social workers, if not in managerial or teaching positions, are essentially technicians rather than professionals. I am quite certain that there are technician-type jobs to be done in social service departments but equally that many social work tasks demand high levels of professional skill and professional knowledge.

By far the greater part of the "classroom" education of social work students is carried out by people with considerable experience of social work practice. Academic sociologists, contrary to your assumption, play quite a small role. But they, together with representatives of other social science disciplines as well as of medicine, have an essential function in helping our student to understand the nature and causes of the problems they encounter in practice. Some of these problems are of a horrendous complexity, and common sense and good will, though essential, do not provide an adequate basis for wise assessment and informed decisions: we need systematic knowledge as well. Anti-intellectualism is rather in fashion at present, and it has some adherents among social workers. But it is sad to see a distinguished medical journal offering its support.

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Caesarean section and respiratory distress syndrome

SIR,—A number of points arise from your leading article on this subject (24 April, p 978). In quoting figures from the 1958 British Perinatal Mortality Survey you do not mention the significantly higher incidence of "hyaline membrane disease" in pre-labour than in during-labour sections and in the latter compared with vaginal deliveries.[1] It has long been my impression that there is a lower incidence or a milder form of respiratory distress syndrome (RDS) associated with caesarean sections performed after rupture of the membrane compared with sections in the presence of intact membranes. Experimental work on the rabbit shows that full aeration of the lungs occurs within 10 minutes of vaginal delivery but that after caesarean section it takes six hours.[2] Indeed in the lamb the lungs contain a volume of fluid that is equivalent to the functional residual capacity after breathing has been established and similarly this takes about five hours to be removed, via lymphatics and the interstitial space of the lungs.[3] The high surface tensions of a surfactant deficiency state would hinder the reabsorption of this inconvenient mass of fluid, thus contributing to the respiratory difficulties. We must not dismiss the chest-squeezing seen in normal delivery and in the procedure of Reis *et al*, which you quote. The significance of the inability to drain liquor may be relevant in the case of twins. The incidence of RDS and of resultant deaths is higher in the second twin.[4 5] In one series[5] the incidence of RDS (from which the infant recovered) was 3·2 times greater in the second twin than in the first; similarly there were 2·1 times more deaths in second twins as the result of RDS. The higher overall mortality of the second twin due to anoxia cannot completely explain this difference and I suggest that it is due to the fact that drainage of liquor from the amniotic sac lessens the incidence of RDS.

If only pulmonary maturation did occur "precisely at 35 weeks of gestation"! It is true to say that the lecithin : sphingomyelin ratios of a population tend to rise at 35 weeks, but individuals do not always conform. Indeed, I have recently demonstrated that the mean regression line for female infants reaches 2·0 at 35½ weeks but that for males it does not do so until almost 36½ weeks. Thus for twins of different sexes, perhaps regardless of birth order, the boy is more likely to develop RDS than the girl at any given gestational age in the risk period up to, say, 37 completed weeks. In this borderline area of susceptibility to RDS all possible measures must be taken to prevent it.

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[1] Fedrich, J, and Butler, N R, *Lancet*, 1972, **2**, 768.
[2] Adams, F H, *et al*, *Journal of Pediatrics*, 1971, **78**, 837.
[3] Strang, L B, in *Development of the Lung* (CIBA Symposium), p 348. London, Churchill, 1967.
[4] Cohen, M M, Weintraub, D H, and Lilienfeld, A M, *Pediatrics*, 1960, **26**, 42.
[5] Neligan, G, Robson, E, and Hey, E, *Pediatrics*, 1969, **43**, 143.
[6] Pearson, R, to be published.

SIR,—Your leading article on this subject (24 April, p 978), while providing a timely reminder that delivery by caesarean section may be associated with a serious neonatal hazard, contains a dangerously misleading error—namely, the statement that "it is precisely at 35 weeks of gestation that pulmonary maturity is attained." If this was so, then the management of almost all pregnancies in which placental dysfunction is suspected would indeed be a very simple matter—delivery would be arranged for exactly 35 weeks.

The fact of the matter is very different in that the developmental time-table for different tissues and systems, including the lungs, varies from fetus to fetus. In this journal my former colleagues and I described how there is sometimes adequate fetal lung surfactant as early as the 32nd week of gestation, but that in other pregnancies this may not occur until almost term.[1] Unless the now widely available amniotic fluid surfactant tests are used (in preference to an imaginary fixed gestational time-table) to confirm sufficient pulmonary maturation in the fetus readily avoidable neonatal deaths from respiratory distress will continue to occur and some high-risk fetuses with already "safe" lungs will be left to die in utero.

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[1] Whitfield, C R, *et al*, *British Medical Journal*, 1972, **2**, 85.

Oestrogens as a cause of endometrial carcinoma

SIR,—Your leading article (3 April, p 791) reviewed the papers of Smith *et al*[1] and Ziel and Finkle[2] and concluded with the unwarranted recommendation that synthetic oestrogens and oestrone sulphate in its various forms should be avoided in the treatment of postmenopausal women. Ziel and Finkle did not consider the effect of the initial selection of their 94 patients into the care of the Kaiser Permanente Medical Center in Los Angeles or reflect on the original indications for oestrogen therapy in this group of privately insured, middle-class patients.[3] They did not examine the effect of parity alone or the interactions between parity, obesity, and age of menopause, nor did they examine the incidence of diabetes, hypertension, or previous anovulation. The pathological diagnoses were not examined independently; it is notoriously difficult to make an unequivocal diagnosis of endometrial adenocarcinoma in an oestrogen-treated patient. The apparent lesion may be extremely localised to the superficial stratum of the endometrium and treatment with a progestogen or curettage some six weeks after stopping the medication may result in the disappearance of the lesion. In the subsequent correspondence Ziel and Finkle[4] indicated that only 15 of the 94 patients had deep myometrial invasion, suggesting that a large proportion of these patients may even have had their disease diagnosed earlier than expected because they were under continuing medical supervision. The incidence of undiagnosed endometrial malignancy can be only conjectural in the control patients who had not had the benefit of specific medical inquiries about any vaginal bleeding. Hysterectomy is unlikely to have influenced the data as the type of patient having hysterectomy is more likely to be the parous woman, not one of high risk for endometrial carcinoma; indeed, quite different hysterectomy rates in the United States and the United Kingdom do not appear to have influenced the respective incidences of the disease in the two countries.

The paper of Smith *et al*[1] shows that the relative risks of endometrial carcinoma vary by disease of the comparison group (cervical, vulvar, or ovarian carcinoma) and by hospital examined as well as year of diagnosis and age of diagnosis, suggesting that "the attempt to quantify patient heterogeneity is incomplete." They indicate that "a pattern [emerges] of endometrial carcinoma developing in large numbers of persons who do not possess the previously reported constitutional physiologic features associated with the disease," yet they themselves state that the second and third National Cancer Surveys showed essentially no change in the incidence of endometrial carcinoma between the years 1947 and 1970. These two papers cannot then be taken as authoritative assessments of the risk of oestrogen in postmenopausal patients.

The hypothesis of Siiteri and MacDonald is incompletely set forth. It states[5] that "the exclusive production of estrone *in the presence of the proper genetic background* and other initiating agents, whether they be virus or carcinogens, appears to play an important role in the development of neoplasia of estrogen target organs" (my italics) and again "the constitutional stigmata that give rise to increased extraglandular estrone production are precisely those that appear to favor an increased occurrence of endometrial neoplasia."[6] These qualifications suggest that controls matched for these constitutional characteristics are essential in any study to test the role of oestrogen in this disease and that the hypothesis relates exclusively to *endogenous* production of hormone.

The biochemical arguments marshalled against oestrone also need careful appraisal. It is true that oestradiol is much more tightly bound to preparations of premenopausal human endometrial nuclei than oestrone.[7 8] However, studies using *whole* cells[9] have shown that oestradiol and oestrone enter the endometrial cell with equal facility, that they are