They compared 15 "cot death" cases with 15 "controls." The ages of the cot death patients ranged from 11 days to 2 years, though most workers in this field agree that the sudden infant death syndrome probably does not occur before 4 weeks of age and is rare after 9 months.¹ Seven of the 15 control cases died within four days of birth, and since they may have been subject to postnatal respiratory problems they cannot be considered comparable with a series of cot deaths. It was stressed that no evidence of degeneration was found in the atrio-ventricular bundle. However, remoulding of the junction between the atrioventricular node and the bundle of His where it penetrates the central fibrous body, an area we term the isthmus, does occur.^{2,3} Furthermore, such remoulding is not present at birth but is apparent within 2-3 weeks.⁴ Perhaps this reshaping of the atrioventricular conducting tissue is not apparent by the method of sectioning the heart described by Dr. Anderson and his colleagues, but it is easily seen if the complete heart is processed intact and serially sectioned by the technique of Ferris and MacLennan.⁵ The atrio-ventricular node receives its blood supply from the right coronary artery or the circumflex branch of the left and the His bundle from the anterior descending branch of the left coronary artery. It is interesting to note that this remoulding occurs at the junction of these separate blood supplies.

Haemorrhages were reported in both the cot death and control series. However, no cause of death was given in the control series, and since we contend that petechial haemorrhages in the heart occur in death associated with terminal hypoxia and a rapid change in intrathoracic tension it is important that any terminal hypoxic episode should be recorded. There is much evidence⁶ in favour of an acute respiratory death associated with virus infection in some cot deaths. In a partly completed study of 38 cot deaths and 30 control cases we have found petechial haemorrhages in the hearts of 18 of the cot death cases and in only two of the controls (see table). One of the two control cases with cardiac haemorrhage died from massive inhalation of vomit and the other from acute myocarditis. In a series reported by Ferris⁷ haemorrhages were found within the atrial wall, and while the histological identification of internodal tracts is disputed there is considerable physiological evidence for the existence of such tracts.⁸ Haemorrhages close to these tracts may be an important factor in the failure to survive in cases of what might otherwise be acute recoverable hypoxia.

In one of the cases illustrated by Dr. Anderson and others (their fig. 2) there was a history of cardiac arrest followed by cardiac massage. The illustration of the atrioventricular node shows abnormal vascularity and an unusual pattern of nodal tissue similar to that seen in hypertrophic

cardiomyopathy.⁹ In this case the nodal haemorrhage may be due either to hypoxia associated with cardiac arrest or to direct trauma. We think that the presence of cardiac petechial haemorrhages in the sudden infant death syndrome supports the view that these infants die during an acute hypoxic episode and that haemorrhages in the conducting tissue structures probably prevent survival.—We are, etc.,

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- 1 Froggatt, P., Lynas, M. A., and Marshall, T. K., *American Journal of Cardiology*, 1968, 22, 457.
- 2 James, T. N., *American Journal of Cardiology*, 1968, 22, 479.
- 3 Ferris, J. A. J., *Proceedings 13th International Congress of Pediatrics*, vol. 4 pt. 19, p. 81. Vienna, Academy of Medicine, 1971.
- 4 Ferris, J. A. J., and Kendeel, S. R., in preparation.
- 5 Ferris, J. A. J., and MacLennan, J. R., *Medicine, Science and the Law*, 1973, 13, 285.
- 6 Ferris, J. A. J., et al., *British Medical Journal*, 1973, 2, 439.
- 7 Ferris, J. A. J., *British Medical Journal*, 1973, 2, 23.
- 8 Davies, M. J., *Pathology of Conducting Tissue of the Heart*, London, Butterworths, 1971.
- 9 Ferris, J. A. J., *Beiträge zur Pathologie*, 1973, 148, 296.

Skin Reactions to Practolol

SIR,—I have read with interest the letter from Drs. R. H. Felix and F. A. Ive (11 May, p. 333) since I am at present preparing for publication the details of a group of patients who have presented with skin reactions and eye signs following treatment with practolol.

The skin eruptions have tended to be of psoriasiform type and the eye symptoms and signs have either developed with the skin eruption or have followed it. The usual finding has been a shrinkage of the conjunctiva with some xerosis. The shrinkage has atypical features and is unlike the conjunctival scarring seen in association with either erythema multiforme or benign mucous membrane pemphigoid. It seems probable that patients on long-term medication with practolol who develop skin eruptions should also have a careful ophthalmic examination.—I am, etc.,

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Vitamin A, Pregnancy, and Oral Contraceptives

SIR,—Mrs. Jennifer Wild and her colleagues (12 January, p. 57) have joined Briggs *et al.*¹ in confirming our original findings that the serum vitamin A levels are raised in women taking oral contraceptives.^{2,3} Finding an "accurate" method for vitamin A estimation is a recurrent problem in vitamin A research,⁴ and it is therefore important that these studies, using different techniques

(fluorometric, ultraviolet irradiation, and colorimetric), revealed similar trends. Some of the methods and conclusions of Mrs. Wild and her colleagues could, however, be considered open to criticism.

In Great Britain the present pattern of serum vitamin A levels in pregnancy is different from that observed 30 years ago in the United States.⁵⁻⁷ During normal pregnancy the maternal serum vitamin A concentration decreases until about the end of the first 12 weeks, then increases gradually until term (pattern consistent between individuals).⁷ Because of this changing pattern Mrs. Wild and her colleagues' method of comparing their first trimester values with the second and third trimester values of studies conducted many years ago does not seem appropriate.

It may also be misleading to draw conclusions from studies of pregnant patients when they are grouped according to the time interval between stopping oral contraception and conception, unless the stage of pregnancy is stated. With these methods it would be difficult to establish whether or not the serum vitamin A is raised in early pregnancy following oral contraception, as our figures have shown that women in their fourth month of pregnancy have levels similar to those of women on oral contraceptives.^{2,7} In the non-pregnant state at least three months is required for the serum vitamin A level to revert to normal after oral contraception.³ By investigating patients four months after such therapy Mrs. Wild and her colleagues probably missed this stage, which may explain why their index and control groups showed no differences. According to my observations, if pregnancy occurs during this time the vitamin A concentration is higher than average in both maternal and fetal tissues.⁸

The authors' study gives no reassurance regarding the teratological significance of the raised vitamin A levels since their observations were made on women past the crucial early stages of pregnancy. Their data concerning the outcome of pregnancies may also not be altogether reassuring. While the first group's figures (women becoming pregnant not more than 20 weeks after oral contraception) corresponds to the normal expectation, the second group (women with blood vitamin A levels above 94 µg/100 ml) could show differences. Excluding cases with unknown outcome of pregnancy and counting only in the second group one baby which appeared in both groups, the proportions of all abnormal pregnancies would be estimated as 6.7% and 14.8% respectively in the first and second groups, and if abortions are excluded the figures are 2.4% and 7.8%. The relatively high incidence of urogenital anomalies in male infants (2.4% and 11.8% in the two groups) deserves attention because an influence of maternal progestogen therapy (from oral contraceptives) cannot be ruled out. Since the sex distribution of the infants was not given I was obliged to base the calculations above on a rough estimate of 50% for each of the sexes.

Vitamin A and synthetic sex hormones are teratogens in experimental animals and the teratological safety of these substances has never been proved in humans. It is well known that drugs can on occasion be powerful teratogens without giving symptoms of

	Total Cases	Distribution of Petechial Haemorrhages				
		Intrathoracic	Cardiac			
			Atrial Wall	A-V Node	Isthmus	His Bundle
Cot deaths (8 wks-9 months)	38	35	15	—	1	3
Controls (3 days-30 months)	30	3	—	—	—	2

maternal intoxication and therefore the lack of maternal clinical hypervitaminosis has little relevance to the discussion of the teratogenic potential of vitamin A.¹ It is evident that the conclusion of Mrs. Wild and her colleagues that "it seems unlikely that women who conceive soon after discontinuing oral contraception run any teratogenic risk from increased vitamin A levels" may yet be more definite than is warranted by their data and should not be accepted as final. Further work is required.—I am, etc.,

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- ¹ Briggs, M., Briggs, M., and Bennum, M., *Contraception*, 1972, **6**, 276.
- ² Gal, I., Parkinson, C. E., and Craft, I., *British Medical Journal*, 1971, **2**, 436.
- ³ Gal, I., and Parkinson, C. E., *Contraception*, 1973, **8**, 13.
- ⁴ Bubb, F. A., *British Medical Journal*, 1974, **1**, 391.
- ⁵ Bodansky, O., Lewis, J. M., and Lillienfeld, M. C. C., *Journal of Clinical Investigation*, 1943, **22**, 643.
- ⁶ Lund, C. J., and Kimble, M. S., *American Journal of Obstetrics and Gynecology*, 1943, **46**, 207, 486.
- ⁷ Gal, I., and Parkinson, C. E., *International Journal for Vitamin and Nutrition Research*, 1972, **4**, 565.
- ⁸ Gal, I., Joint meeting of the Blair-Bell and Clinico-Pathological Societies, February 1972.

Complication of Proximal Gastric Vagotomy

SIR,—About a year ago a death following proximal gastric vagotomy was reported in your columns.¹ It appeared that the lesser curve of the stomach had sloughed. I am writing to describe a very similar case.

The patient was a lean, 64-year-old man with a 10-year history of peptic ulceration who had developed pyloric stenosis. On 7 March 1974 proximal gastric vagotomy and Heinecke-Mikulicz pyloroplasty were performed. In the initial postoperative phase there was delay in gastric emptying and a nasogastric tube was not removed till 11 March. On 13 March he suddenly collapsed with generalized peritonitis. At operation 45 minutes later it was found that the whole of the lesser curve of the stomach had sloughed between the two lines of ligatures placed on the blood vessels entering it. A 0.5-cm strip of adjacent mucosa was purple and obviously had a very poor blood supply. The peritoneal cavity was cleaned out and the defect in the stomach wall was closed with interrupted black silk. A Maurice Lee tube was inserted and the abdomen was closed with drainage to the lesser curve of the stomach. Leakage of gastric contents from the abdominal drain occurred on 25 March but gradually ceased and eventually the patient recovered completely.

The sequence of uneventful proximal gastric vagotomy, dissolution of the lesser curve on the sixth day, and recurrence of leakage 12 days later is exactly the same as in the previously reported case. I wonder whether there may have been other similar cases which have not been reported. If this is so surgeons may have to alter their impression that proximal gastric vagotomy is an operation almost completely free from undesirable sequelae.—I am, etc.,

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- ¹ Newcombe, J. F., *British Medical Journal*, 1973, **1**, 610.

Drugs for Gastric Ulceration

SIR,—Your leading article on gastric ulceration (27 April, p. 186) asks "Where is the clinician to turn when faced with this variety of drugs?" May I suggest that he

has very little to do with any of them. But, instead of trying to influence the "gastric acid secretory potential," tries relying instead on the natural neutralization of the gastric acid that occurs with natural foods and which is so seriously interfered with (especially by refining the carbohydrates) in our modern civilization.

Epidemiological evidence from all over the world and from prisoner-of-war camps in the last war shows the very close correlation between such interference and the incidence of gastric and duodenal ulceration¹ and has recently been much strengthened.² The striking absence of peptic ulceration in those peoples who do not consume refined carbohydrates and the closely related reduced buffering effects of these foods on the gastric acid merit the closest attention.—I am, etc.,

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- ¹ Cleave, T. L., *Peptic Ulcer*. Bristol, Wright, 1962.
- ² Cleave, T. L., *The Saccharine Disease*. Bristol, Wright, 1974.

Pregnancy Associated with Copper-containing I.U.D.

SIR,—Pregnancy may occur in women with an intrauterine contraceptive device in situ.^{1 2} Usually the device stays embedded in the placenta or membranes and the thread tends to disappear as pregnancy advances. In one series,³ for instance, 33 patients using the Birnberg bow became pregnant. In only two cases the bow was expelled and in 31 it remained in situ. Out of 16 patients who became pregnant while using a Lippes loop the loop remained in situ in 13. In another series⁴ the contraceptive device was retained in utero in all 29 pregnant patients. However, in a study of an intrauterine contraceptive device containing copper wire (Copper-7, Searle) we noticed in 13 patients who became pregnant a feature that is uncommon with other devices. Twelve of the 13

patients had not expelled the device. In three of the 12 the Copper-7 remained in situ, as occurs with most devices, but in nine the device had descended and the stem lay with the tip just palpable within the external os.

Removal of a tailed intrauterine device has been reported to increase the abortion rate.⁵ In our limited experience, however, it would seem that when a Copper-7 device lies low in the cervical canal the placental site is not disturbed when the device is withdrawn. It is interesting to examine the possible reasons for this difference. One likelihood is that the shape of the device is such that it is pushed down by the conceptus instead of becoming trapped by it. The other possibility is that pregnancy occurs mainly when the device lies in such a way that much of the stem, which carried the copper, lies in the cervical canal. Though the exact mode of action of copper-containing devices is not fully understood the contraceptive action is probably effected by release of copper ions into the surrounding tissue fluids.⁶ The pregnancy rate is thus within certain limits dependent upon the surface area of the wire within the uterine cavity.⁷ It is therefore essential to ensure that when the device is inserted it lies right up against the fundus uteri.

We thank G. D. Searle & Co. for their help in this study.

—We are, etc.,

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- ¹ Tietze, C., and Lewit, S., *Intrauterine Contraception*. Amsterdam, Excerpta Medica, 1965.
- ² Wood, C., *Intrauterine Devices*. London, Butterworths, 1971.
- ³ Bolognese, R. J., et al., *Fertility and Sterility*, 1968, **19**, 294.
- ⁴ Wajtraub, G., *Fertility and Sterility*, 1967, **18**, 570.
- ⁵ Lewit, S., *Contraception* (Los Altos), 1970, **2**, 47.
- ⁶ Oster, G. K., *Fertility and Sterility*, 1972, **23**, 18.
- ⁷ Zipper, J. A., et al., *American Journal of Obstetrics and Gynecology*, 1969, **105**, 1274.

Clinical Public Health Doctors

SIR,—I welcome your leading article (18 May, p. 348) in stressing the current uncertainty of the future of the clinical medical officers but join issue with the statements, "Worse still the isolation of the school health service is to be perpetuated, since it is to be put in the charge of the area specialist in community medicine (child health)" and later, "The appointment is not consistent with the concept of a district department of child health and furthermore is seen by many experienced child health principal medical officers as the imposition of another doctor above them, thus degrading their own position."

In the former large local authorities there was the medical officer of health and principal school medical officer in titular charge of the service, usually supported by a senior medical officer for school health responsible for the day-to-day administration. In the new service the area specialist in community medicine (child health) is responsible to the local education authority, not the area medical officer, so that one community physician is responsible for the school health service. I cannot see where there is the

imposition of another doctor—the situation is the reverse.

I cannot understand how you can justify your statement about the perpetuation of the isolation of the school health service from the document "Child Health Services (Including School Health Services)" (HRC (74)5). One of the main functions of the whole sphere of operation of the medical aspects of community child health. Nothing in HRC(74)5 precludes the future creation of district specialists to help to provide a comprehensive district child health service and I would agree that the main role of the area specialist should be co-ordination, epidemiology, and medical education.

However, I agree with the main theme of your article. There is a feeling among clinical medical officers that the majority of administrators at area and district levels have come from the hospital services and have little knowledge of community child health. No career structure has been agreed, and to await the Court Committee's findings, possibly late in 1975, shows a lack of realism regarding the work to be carried out now. Experienced staff are leaving the