(13 January, p. 109). I wanted and still want to warn the others. I have my suspicions, too, about you, Mr. Editor, as the result of your putting out a special supplement on the royal malady complete with vivid purple-coloured cover!—I am, etc.,

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REFERENCE

- ¹ Waldenström, J., and Vahlquist, B., *Acta med. scand.*, 1944, 117, 1.

Steroids and Nephrotic Syndrome

SIR,—Dr. I. G. Wickes (16 March, p. 706) claims that the regimen he uses will identify those children with nephrotic syndrome who are "likely to relapse." He instructs the mothers to test their child's urine with Esbach's reagent and to record the result. However, this does not predict anything; it merely records the relapse or the degree of proteinuria. We know of no way of predicting which nephrotic child will run a relapsing course; neither renal biopsy nor the selectivity of proteinuria will do this. Dr. Wickes then advocates that the mother should titrate the dose of steroid against the amount of proteinuria, maintaining that in this way the increasing proteinuria that may be associated with colds and other infections is kept in check. We do not doubt that the proteinuria goes during this regimen, but believe that it would often disappear without any therapy. Our experience accords with that of Arneil and Lam.¹ Very often the proteinuria goes as the child recovers from the respiratory infection, and therefore it is better to withhold steroids for one or two weeks, provided that oedema does not develop. Withholding therapy avoids needless steroids, and parents agree to it willingly, since the child does not necessarily feel ill at the time of the proteinuria.

As he suggests, a simple record of proteinuria kept by the parent can be very helpful for outpatient supervision. However, Esbach's test seems a rather cumbersome one to inflict on parents, particularly as it takes 24 hours to read. Albustix is a simple and reliable test² which a parent or child can use readily at home, recording the result in a few seconds. When demonstrating the method we warn them that trace and transient one-plus readings are unimportant.—We are, etc.,

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REFERENCES

- ¹ Arneil, G. C., and Lam, C. N., *Lancet*, 1966, 2, 819.
² Rennie, I. D. B., and Keen, H., *Lancet*, 1967, 2, 489.

Detection of Bacteriuria

SIR,—Dr. J. M. Bradley and others in their paper (16 December, p. 649) have misquoted the results we obtained in our assessment of the triphenyl tetrazolium chloride reduction test for the detection of significant bacteriuria.¹ Their statement that in our series "only 44% of infections in the 10⁴–10⁹ organism per ml. of urine range were

detected" referred only to the ability of the test to detect infections with Gram-positive organisms, and this was made clear in our paper. As we also made clear, when patients on antibiotic therapy were excluded, the triphenyl tetrazolium chloride (T.T.C.) test picked up 12 of 24 infections with Gram-positive organisms, and these included, besides a number involving *Streptococcus faecalis*, infections with diphtheroids, micrococci, and *Candida albicans*, infections which the method described by Dr. Bradley and colleagues, depending as it does on the use of one MacConkey plate only, must inevitably miss.

When all urines with significant bacteriuria (10⁵ or more bacteria per ml.) encountered in a general hospital are considered the detection rate for the T.T.C. test was 182 of 258 specimens or 70.5%. Our paper further pointed out that in a selected population, such as in obstetrical patients, where 79% of all infections are due to *E. coli*, the detection rate of the T.T.C. test can be bettered, and can detect as many as 90% of cases of significant bacteriuria.

No chemical test, of course, can replace accurate quantitative culture of urine, but, as Dr. Bradley and colleagues point out, there is a real need for a simpler, less expensive screening method. We found, and reported, the T.T.C. test to be a much better screening method than the quotation of our paper would suggest. A direct comparison between the T.T.C. test and the method of Dr. Bradley *et al.* is not possible. Their graph (Fig. 2) shows a reasonable correlation between the number of colonies counted by their method, and a true quantitative culture in 20 specimens, none of which apparently had more than 10⁵ bacteria per ml. The graph (Fig. 4) showing the correlation between counts obtained with a standard loop and their screening method also shows a correlation, but in fact their paper does not state how many of a given number of positive urines can be screened out, nor how many false-positives are obtained, and the comparative values of the T.T.C. method (which is simpler to perform) and their screening method remain to be assessed.—I am, etc.,

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REFERENCE

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Acute Iron Poisoning in Children

SIR,—Drs. D. G. D. Barr and D. K. B. Fraser (23 March, p. 737) report further tragedies from swallowing ferrous sulphate. This is the salt of a weak base and strong acid. Its solution has a pH of 3.7 and readily damages any mucosa with which it is long in undiluted contact. Many neutral salts are now available which small children may swallow by the dozen without harm. Often these are damned because they cost 2d. a week more than ferrous sulphate, but with increasing use of desferrioxamine even this miserable economic argument may fail. Is it not time to abandon and to ban the use of ferrous sulphate, which is a lethal poison?—I am, etc.,

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Q Fever

SIR,—The account of Q fever in Northern Ireland by Dr. J. H. Connolly (2 March, p. 547) prompts us to report our own experience in the period 1960–7. During these eight years we have made a laboratory diagnosis of Q fever by serological tests or by isolation of *Rickettsia burnetii* from 24 patients (and confirmed it in two additional cases from other laboratories). They resembled closely those reported by Dr. Connolly in age and sex (see Table) and in seasonal distribution.

Age and Sex Distribution

Age Group	Male	Female	Total
10–29	4	1	5
30–49	6	4	10
50–69	5	2	7
70+	2	0	2
All ages	17	7	24

The acute illness of two patients began in the first quarter of the year, of 10 in the second, one in the third quarter, and six in the fourth. The types of illness were: pneumonia, 10 cases (one chronic with probable endocarditis); fever, eight cases (one severe with vomiting, dehydration, and rash; one post-partum); subacute endocarditis, four fatal cases, all male; pericarditis, two cases, one with pleuropericarditis, one with aortitis and syphilitic aneurysm. The last patient, the four with subacute endocarditis, and the patient with pneumonia and probable endocarditis all had high complement fixing antibodies to both phase 1 and phase 2 antigens, as had also an additional case of endocarditis in which we made confirmatory tests for another laboratory. Serious cardiovascular diseases have thus been more prominent in our series than in Dr. Connolly's, but pericarditis was found in both series. Tests for Q fever as well as for Coxsackie virus infection therefore seem appropriate in cases of acute pericarditis.

Contact with farm animals (mostly bovine) or their products was traceable in most cases. Six were farmers, three had driven trucks loaded with dung, one had cleared dung from cattle wagons, and three used dung as gardeners; three had consumed unpasteurized milk and one of these lived beside a farm; one office worker frequently visited a byre; an electrician developed pneumonia with serological evidence of both Q fever and a psittacosis-group infection after working in an abattoir¹; another case of pneumonia developed after working in fields near lambing sheep. No contact was traced in three Glasgow patients or three from the counties of Renfrew, Dumfries, and Kirkcudbright; in each of these counties Q fever is known to infect cattle. Infection of sheep and cattle in the west of Scotland has been recognized for more than a decade² and continuing surveillance tests in collaboration with Dr. T. F. Elias-Jones continue to reveal *R. burnetii* in samples of unpasteurized milk. We agree, however, with Dr. Connolly that inhalation of particles derived from farm animals or their products seems a more important source of infection than ingestion of infected milk.

The finding of false positive complement fixation antibody responses to Q fever in some children with infections due to adenoviruses types 7 and 21 was reported at the meeting of the Society for General Microbiology, Janu-

ary 1967, by Dr. J. van der Veen, Department of Hygiene, University of Nijmegen, Holland. We have recently encountered one such case in a boy aged four years with pneumonia from whose throat swab adenovirus type 7 was isolated. His sera on 9th, 21st, and 37th days of illness gave complement fixation titres for adenovirus of 1/256, 1/4,096, and 1/2,048 respectively; the titre for Q fever (phase 2) showed a rapid rise and fall, the corresponding figures being <1/8, 1/128, and <1/8 respectively.—We are, etc.,

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REFERENCES

- ¹ Grist, N. R., and McLean, C., *Brit. med. J.*, 1964, 2, 21.
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Pigmentation and Oral Contraceptives

SIR,—Some users of oral contraceptives show pigmentation¹⁻³ similar to that found in pregnancy. Many women find pigmentation so cosmetically disturbing that they prefer to give up the pills.¹ After discontinuation the pigmentation slowly disappears.

Against this background a patient is presented who developed a marked hyperpigmentation of a different type while taking Lyndiol (lynoestrenol, mestranol).

The patient was a 34-year-old woman in good health. She had had two full-term pregnancies during which no appreciable pigmentation of the skin had occurred. In autumn 1963 she started to take Lyndiol. At first she gained some weight. After six months the administration was interrupted for one month, during which her weight resumed its normal level. Repeated Lyndiol therapy did not affect her weight. In March and April 1965 she experienced indefinite abdominal pain, for which she was examined at hospital; an innocent appendix was removed and contrast medium radiography of the gall bladder and large intestine was carried out. No specific findings were made. Abdominal pain continued and she experienced sweating, tremor, and prostration. She also found that her skin was getting darker. Dark-brown pigmentation was seen on the face and on both upper and lower limbs. A few freckles were seen on her cheeks, forehead, and chin. The impression was Addison's disease, suggested by the patient's thinness, abdominal pain, and increased pigmentation, including pigmentation of the oral mucosa, operation scar, and in palmar lines.

Lyndiol was discontinued and a dexamethasone suppression test of the pituitary carried out with dexamethasone (0.5 mg. × 4) when the 17-OHCS fell from 15.7 mg. to 4 mg./24 hrs. and 17-KS from 8 mg./24 hrs. to 1.2 mg./24 hrs. The patient was treated with dexamethasone for a further two weeks (0.5 mg. × 2). During this therapy the patient's subjective abdominal pain ceased and the skin pigmentation diminished rapidly; after 3-4 weeks the skin had resumed its normal colour and the freckles were smaller. Lyndiol was tried again four months later, in December and January (this is the season when there is practically no sun at the latitude of Oulu). In one to two weeks the patient's skin began to darken; pigmentation appeared on the face, arms, and

feet. Laboratory tests gave normal results. The use of the pills was discontinued, and in a fortnight the pigmentation had almost disappeared. Subsequently the patient has been in good health, and no abnormal pigmentation has been observed.

Chloasma has been found to be one of the most common secondary symptoms in users of oral contraceptive pills.¹⁻³ The present patient had both chloasma and dark, smooth pigmentation in different parts of the body. The coloration of the skin was exceptionally dark for a Finnish woman, similar to the skin pigmentation seen in Addison's disease. Addison's disease was suggested by oral pigmentation, as well as by the patient's subjective symptoms, loss of weight, and indefinite abdominal pain. The normal secretion of adrenal cortical hormones into the urine and the normal levels of serum electrolytes were in contradiction with Addison's disease. The part played by the Lyndiol tablets was demonstrated by the fact that the use of the contraceptive for four months later during the dark season of the year distinctly increased the pigmentation again.

Apparently Lyndiol tablets had a stimulating effect on the secretion of pituitary melanocyte-stimulating hormone, with the result that unusually strong cutaneous pigmentation developed; the effect was reversible.—We are, etc.,

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REFERENCES

- ¹ Cook, H. H., Gamble, C. J., and Satterthwaite, A. P., *Amer. J. Obstet. Gynec.*, 1961, 82, 437.
- ² Esoda, E. C., *Arch. Derm.*, 1963, 87, 486.
- ³ Rice-Wray, E., Schulz-Conteras, M., Guerrero, I., and Aranda-Rosell, A., *J. Amer. med. Ass.*, 1962, 180, 355.

Asbestos Bodies

SIR,—The paper by Dr. T. Ashcroft "Asbestos Bodies in Routine Necropsies on Tyneside: A Pathological and Social Study" (9 March, p. 614) raises one or two points upon which I cannot refrain from commenting. This paper is merely another one of a number of similar studies which have been appearing from different countries, as enumerated by Dr. Ashcroft in his introductory remarks. His findings are similar in most respects to those previously published. In no studies so far published have there been supporting data to indicate whether all, most, or some of the bodies were asbestos.

The finding of "asbestos bodies" in only 63 cases in a necropsy series of 311 cases raises one very interesting question. Are all the "asbestos bodies" in fact due to retention of asbestos fibres in the lung? Dr. Ashcroft's findings in relation to age and sex indicate that occupational exposure of the male is perhaps more significant than urban contamination. No cases of severe asbestosis are recorded in this series, although one case of mesothelioma of the pleura is. In Table IV Dr. Ashcroft attempts to show the relationship between the incidence of malignancies in the series and the finding of "asbestos bodies," but has to conclude that there is no statistically valid association.

There does seem to be an association between asbestos exposure and diffuse mesothelioma if the bodies described are in fact "asbestos bodies." Dr. Ashcroft identifies "asbestos bodies" by examining their morphology under the light microscope. The term "asbestosis body" was discarded in favour of "asbestos body" some years ago, as bodies are found without necessarily finding asbestosis as well. Many workers believe that the term "asbestos body" should be used only when it can be shown by more exact techniques that the fibre present in the body is asbestos. Until a body can be shown to have been derived from asbestos, then a more suitable descriptive name would be "ferruginous body."¹ It is well documented that mineral particles of needle shape other than those of asbestos can produce structures which closely resemble "asbestos bodies."² Gough has said that the most important differential diagnosis is, however, from bodies having a similar appearance but formed from the elastic tissue of the lung. This condition is haemosiderosis of the elastic tissue, sometimes called elastosis. Talc exposure may result in the presence of bodies which are indistinguishable from "asbestos bodies," and may be due to tremolite, a form of asbestos in talc.³

The presence of "ferruginous bodies" in random necropsy material studied by means of the lung-smear technique has been adequately demonstrated. Whether these "ferruginous bodies" are all due to asbestos has never been adequately shown. Investigations, using low-temperature ashing techniques to recover inorganic material from lungs and then subjecting this material to electron diffraction, are now in progress. Animal experiments are also in progress which thus far show that "ferruginous bodies" can be formed after inhalation of talc, glass fibre, and aluminium hydroxide. There is ample reference in the literature⁴ that so-called "asbestos bodies" can be found in workers with exposure other than asbestos—for example, graphite workers, soft-coal miners, diatomaceous earth workers, etc.

I would like to make a plea to future investigators of this subject that they make every effort to demonstrate the presence of asbestos in "ferruginous bodies" before concluding on morphological grounds alone that this is a *sine qua non*.—I am etc.,

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REFERENCES

- ¹ Gross, P., *Arch. Path.*, 1966, 82, 195.
- ² Gough, J., *Ann. N.Y. Acad. Sci.*, 1965, 132, 368.
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- ⁴ Cooper, W. C., *Arch. environm. Hlth*, 1967, 15, 285.

Decreased Tolerance to Heroin

SIR,—The papers concerned with morbidity and mortality from heroin addiction by Dr. T. H. Bewley and others (23 March, pp. 725 and 727) must be of great interest to those who will shortly have to deal with heroin addicts at the new centres.

One cause of death in the case of heroin addicts is an overdose of heroin due to a decrease of tolerance following a period of abstinence in hospital or prison. After such