this field, after studying large numbers of subjects of all ages,[1-3] would probably consider them to be unrealistically high as a guide to the lower limit of normality. It is also difficult to know what importance can be attached to the findings of a low total bone value in a narrow trephine (0·3 cm) bone biopsy sample since it is known that there may be wide variations in the individual patient depending upon the site selected.[3]

Regarding the number of patients with osteomalacia, we are told that osteomalacia was diagnosed when the osteoid index was 0·8% or more, since the index was less than 0·7% in all normal subjects. It is well recognized that it is unreliable to diagnose osteomalacia merely on the basis of an excess of osteoid. This is particularly the case in azotaemic renal osteodystrophy where excess osteoid formation may be a feature of osteitis fibrosa associated with increased osteoblastic activity even in the absence of osteomalacia. The diagnosis of osteomalacia can be difficult in these circumstances and it is necessary to take cognizance of the amount of osteoid, the width of osteoid seams, and the nature and distribution of the calcification front.[4]

Regarding osteitis fibrosa, apparently this was diagnosed and graded on the number of identifiable areas of bone "scalloping." The main difficulty here is that resorption of bone in these sick patients may not necessarily be due to osteitis fibrosa, and it is desirable to take into account the number of osteoclasts and amounts of marrow fibrosis and woven bone formation.[4]

One final point concerning the time of onset of osteomalacia in azotaemic renal osteodystrophy. Recent published work[4] based on detailed quantitative histology of bone is in keeping with the view that the first bony abnormality to arise is usually osteitis fibrosa due to secondary hyperparathyroidism, with subsequent development of a mineralization defect and osteomalacia. This concept is also consistent with the results of studies of serum parathyroid hormone levels in patients with chronic renal failure.[5]—I am, etc.,

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Q-fever Endocarditis

SIR,—In Dr. Graham W. Hayward's Croonian Lecture (23 June, p 706 and 30 June, p. 764) on infective endocarditis *Rickettsia burneti* is mentioned as a causative organism in the section on vegetations but ignored in the sections on diagnosis and treatment. Though it is not considered to be a common cause of the disease, four cases have been diagnosed in this laboratory in the past six years. It is therefore our practice to look for antibodies to *R. burneti* in a specimen of blood collected during the time blood cultures are being taken from cases of infective endocarditis. The diagnosis is made by the demonstration, not of a rising titre because this is a chronic infection, but of high titres of antibody to both phase 1 and phase 2 antigens. Dr. Hayward's recommendation that "bacteriologically negative patients should be treated as if they had a resistant organism such as the enterococcus" implies that patients suffering from Q-fever endocarditis should be treated with the ineffective combination of penicillin and streptomycin, although evidence is accumulating that other antibiotics may at least arrest the progress of the disease. Tetracycline has been used successfully either alone[1-4] or combined with lincomycin,[5] co-trimoxazole[6] or chloramphenicol.[7]

In this connexion we are able to quote the outcome of case 6 of Kristinsson and Bentall,[7] the only patient in their series who was considered unsuitable for surgery. He terminated his tetracycline treatment in December 1967, after about 10 months, and had no more antibiotic therapy. He required digoxin and diuretics to control his cardiac failure but continued working intermittently as a car park attendant. His antibody titres to *R. burneti* fell during treatment and then appeared to stabilize, the results for the last two sera being:

Date	Phase 1	Phase 2
10 December 1968	1/160	1/160
5 December 1969	1/160	1/80

He died in August 1971 after a road traffic accident. At necropsy there was mitral stenosis but the other valves were normal. The vegetations on the mitral valve were fibrosed and no rickettsiae were seen in them. Some of this tissue was inoculated into guinea-pigs; they did not develop antibodies to *R. burneti*. We suggest that this patient's Q-fever endocarditis was cured by tetracycline and chloramphenicol.

In view of the successes claimed for both medical and surgical treatment we consider that the early diagnosis of infective endocarditis due to *R. burneti* is important. The delay involved in waiting until other forms of treatment have failed may result in serious valvular damage.

We are grateful to Drs. A. J. B. Edwards, M. George, and R. B. H. Tierney for information about this patient.

—We are, etc.,

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Epidemiology of Simple Hypospadias

SIR,—Professor H. Campbell and his colleagues (7 July, p. 52), using data relating to malformations notified to local health authorities in England and Wales in 1967-71, show that the incidence of hypospadias did not vary according to the month of birth. They point out that this finding differs from that reported by Dr. C. J. Roberts and Mrs. S. Lloyd (31 March, p. 768) from South Wales, where there were fewer cases than expected in pregnancies commencing in the period April-September (corresponding broadly to January-June births). Two earlier studies also gave conflicting results. One carried out by the College of General Practitioners in 1954-60[1] showed no seasonal variation, but in the United States Wehrung and Hay[2] reported an appreciable excess of cases among infants born in the first half of the year.

These inconsistencies prompted us to look at our records relating to hypospadias in Birmingham births. We examined two 10-year periods, 1950-9 and 1963-72, but since the two distributions were similar we have combined them into a single table. Like Dr. Roberts and Mrs. Lloyd we have excluded cases associated with other malformations. Expected numbers, based on the monthly distribution of all Birmingham births, represent the number of cases that would have occurred if the monthly incidence had remained constant.

Month of Birth	No. of Cases	
	Observed	Expected
January	26	26·9
February	21	24·8
March	20	28·1
April	31	26·9
May	31	27·7
June	26	26·5
July	24	27·0
August	30	25·8
September	25	25·9
October	27	25·5
November	29	23·8
December	24	25·1

The close agreement between observed and expected numbers leads us to conclude that in Birmingham, as in the country as a whole, there is no appreciable seasonal variation in the incidence of hypospadias.—We are, etc.,

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Lithium Toxicity in the Newborn

SIR,—Although reports to the Registrar of Lithium Babies (Dr. M. Schou and others, 21 April, p. 135) include two infants "floppy" at birth and one case of perinatal asphyxia out of a total of 113 liveborn infants, there have been no detailed reports published of problems encountered in the neonatal period in infants delivered to lithium-treated mothers whose serum lithium was below the toxic level of 2 mEq/l.[1] Toxic symptoms have been noted in one infant whose serum lithium level was 2·4 mEq/l. on the second day of life but whose mother's level, post-delivery, was 4·4 mEq/l.[2] Silverman *et al.*[3] noted no long-term effects in an infant whose serum lithium level was 1·1 mEq/l. at birth, but a degree of hypotonia was present for 48 hours.

We suggest that transient but significant effects of lithium toxicity may occur in infants born to mothers whose serum lithium levels are within therapeutic limits:

A 3·07-kg male infant was born to a 26-year-old A-negative woman, gravida 3, para 1+1. Because of manic-depressive symptoms lithium carbonate had been prescribed, 800 mg daily throughout pregnancy. Serum lithium levels at 26, 32, and 35 weeks were 0·22, 0·06, and 0·20 MEq/l. respectively. She had taken no other medication during pregnancy. The infant was delivered at 38 weeks gestation by lower segment caesarian section because of a previous section and a transverse lie. Anaesthetic agents used during this procedure were atropine 0·6 mg as premedication, 400 mg thiopentone, 100 mg suxamethonium, and 20 mg D-tubocurarine during induction, nitrous oxide, and oxygen. Delivery was effected within four minutes of induction.

The infant was in fair condition at birth with an Apgar score of 5 at one minute rising to 8 at 10 minutes with minimal resuscitative measures. At this time marked hypotonia was noted. The serum lithium level (cord blood) was 0·32 mEq/l. The child fed from a bottle at four hours but was still hypotonic. Circulation was adequate, but respiration was shallow and slightly laboured. Poor sucking, bradycardia (110/min), and continuing hypotonia were noted at 10 hours. The arterial glucose level at this time was 48 mg/100 ml and blood gas levels were satisfactory. A chest *x*-ray was normal and blood culture sterile. Intra-arterial supplementation of oral feeding was begun.

The child's condition slowly improved over the next 14 hours, with deeper respiration but poor response to handling. The serum lithium level at 24 hours was 0·38 mEq/l.; urea, electrolyte, and blood gas levels were satisfactory. During the subsequent 48 hours his sucking and other responses became normal but a mild degree of jaundice developed. He was returned to the postnatal ward on the fourth day when a minor degree of poor sucking recurred. The serum lithium levels on the fourth and sixth days of life were 0·24 and 0·05 mEq/l. respectively. He began gaining weight on his ninth day and his feeding then improved. He was fit for discharge on the 13th day, weighing 2·83 kg., feeding well, and behaving normally.

In the absence of specific evidence of other causes of hypotonia, poor sucking, and poor respiratory effort it is suggested that this infant suffered transiently from lithium intoxication.

It may be that alterations in sodium distribution due to altered intake sensitize the neonate to the effects of lithium as in adults,[4] though other unknown sensitizing factors may operate. It would seem wise to consider a diagnosis of lithium toxicity in all hypotonic infants born to mothers taking lithium and to ensure adequate fluid and mineral intake during the first few days of life.—We are, etc.,

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General Practitioners' Superannuation

We continue to receive many letters on this subject. Below we print extracts from a selection of them.—E.D., *B.M.J.*

Dr. W. L. TULLIS (Newburgh, Fife) writes: Having been in general practice for over 40 years I would like to express my agreement with Dr. G. D. J. Ball and his colleagues and others (14 July, p. 111) re general practitioners' superannuation as applied to those of us who are no longer young . . . . I trust that the authorities concerned will act speedily to remedy the wrongs perpetrated over many years.

Dr. P. W. G. BAXTER (Brixham, Devon) writes: I note with satisfaction that the three Plymouth doctors are asking for more letters in the journal on this subject (14 July, p. 111). I agree heartily, but I recommend that as a further step all those concerned should do what I have just done, which is to send a letter to their M.P. setting out the facts of the case. It should be further suggested that if (as the Secretary of State alleges) retrospective payments cannot be made on account of existing regulations, it is only reasonable that new legislation should be brought in to remedy the injustice and put us on the same footing as our younger colleagues. . . .

Dr. C. C. M. WATSON (Penrhyndeudraeth, Merionethshire) writes: . . . My generation qualified at the beginning of the war and will be retiring over the next 10 years or so. Most of us have literally spent the whole of our working life in government service, apart from the two years between being demobbed from the forces after the end of the war and the beginning of the N.H.S. in 1948. What I feel is so absurd is that our time spent in uniform counts for nothing towards our retirement pensions, though both were a part of government service. . .

I have been horrified at the small pensions my older colleagues get when they retire, and there is an uncomfortable feeling abroad that we have all been "conned." Doctors unfortunately tend to be a trusting lot when it comes to financial matters and the complicated method of calculating our pensions, which few of us really understand. In this era of galloping inflation if we are to have any chance of a decent standard of living when we retire (those of us lucky enough to live that long!) we must all agitate now for all we are worth to get a fair deal for our retirement. It must be obvious that once we have retired and grim reality stares us in the face there is little we can do about it. The moment of truth has arrived with a vengeance! I should be very interested to know if many of my colleagues feel as strongly as I do that our war service should count towards our retirement pension. . .

Dr. B. A. COWAN (Liverpool) writes: I retired from general practice on reaching the age of 70 in 1970. May I add my voice of protest to the growing expressions of disappointment at loss of value in our now very inadequate pension. Is it too late for anything to be done to help out during the remaining time that may be left to us to enjoy?

Dr. A. GOODMAN (Liverpool) writes: . . . One has only to compare the value of our contributions since 1948 and the value of the superannuation benefit we now receive to see how badly the older G.P.s have been treated.

Dr. R. GARDNER (Manchester) writes: I wish to state how strongly I support the views expressed by other G.P.s (more eloquently than I could) regarding the superannuation of the G.P.s who retired before March 1972. It would appear that the generation of doctors who attemped to make a success of the 1948 N.H.S. and whose health suffered as a result of 20 years of hard work are being penalized for their past endeavour. . . .

Dr. T. B. MCALEER (London W.2) writes: This iniquitous treatment of G.P.s who retired before March 1972 is a sin crying to heaven for vengeance. I have written to the Secretary of the B.M.A. and to my member of Parliament and have received courtequs, soothing replies with vague remarks about the possibility of the situation's being reviewed at some time in the future. At 70 years of age I am more interested in the present than the future. The younger members of the B.M.A. should feel ashamed to sit back and do nothing about our pensions, and they should remember that some of them might reach our age and be less able to fight for justice.

Dr. J. P. SENIOR (Filey, Yorks) writes: . . . The most worrying aspect of the matter, in my opinion, is how, when we die, our widows are going to exist, as their pensions then become very considerably reduced.

Redesign of Medical Records in General Practice

SIR,—Recent assessment of various size and format folders preparatory to the choosing of one best suited to N.H.S. general practice purposes has led, it seems, to the A4 format (210 mm × 297 mm) being the type most favoured at the present time. The disadvantages of a file of this size are very obvious: unwieldy to stack on table-top and to carry on visits, and requiring a completely new filing system taking up a great deal of valuable space.

What has been made of the merits of a folder size more of A5 dimensions (148 mm × 210 mm)? This is approximately twice the size of the current medical record envelope compared with the A4, which is about four times that of the medical record envelope. The personal medical folder used by the Services, their F.Med 4, is of this order of size (233 mm × 177 mm), and would be a far better compromise. It would be far easier to handle and to file, and will indeed fit into a standard N.H.S. filing cabinet, albeit when laid on its side rather than vertically. Large-sheet hospital letters will fit