Ultrasound is more valuable in prenatal diagnosis than in the treatment of urinary tract anomalies. Two thirds of our patients had no clinical evidence of urinary tract disease at birth, and those with obstructive lesions would not have been diagnosed and treated so early had it not been for prenatal ultrasonography. Even in this respect a note of caution: not all dilated urinary tracts are obstructed (and surgical intervention is usually contraindicated) while the natural history of the lesser degrees of obstruction now being detected by ultrasound is largely unknown.[2]

With our presently limited knowledge it would be unwise to make any extravagant claims for the benefits of prenatal ultrasound examinations in the treatment of congenital anomalies of the urinary tracts.

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[1] Kramer SA. Current status of fetal intervention for congenital hydronephrosis. *J Urol* 1983;**130**:641-6.
[2] Whitaker RH. Diagnosis of obstruction in dilated ureters. *Ann R Coll Surg Engl* 1973;**53**:153-66.

Standardisation of oral anticoagulant treatment

SIR,—Dr A M H P van den Besselaar and others (11 February, p 486) state that the use in Britain of Quick test reagents other than Manchester comparative reagent is insignificant. A survey carried out by the UK national external quality assessment scheme on behalf of the British Society for Haematology showed, in fact, that only 76% of inpatient tests and 72% of outpatient tests are performed with Manchester comparative reagent or Manchester capillary reagent, the remainder using Thrombotest or a mixture of reagents. As the survey also showed an estimated total of 2·3 million tests performed annually, the number of tests using Thrombotest is at least 500 000, not an inconsiderable number. It is therefore important that hospitals using Thrombotest should calibrate the reagent against British comparative thromboplastin and report results as British corrected ratio until the manufacturers of Thrombotest state the international sensitivity ratio of each batch. The international normalised ratio (INR) could then be calculated as (ISI = international sensitivity index): $INR = (\text{prothrombin ratio})^{ISI}$.

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Treatment of hypercalcaemia associated with malignancy

SIR,—Dr R Wilkinson was surprisingly dismissive about the role of cytotoxic chemotherapy or hormonal manipulation to control the underlying tumour and therefore the hypercalcaemia (17 March, p 812). He says that the correct approach is to use agents which inhibit calcium resorption from bone, but no mention is made about using the most effective treatment directed against the tumour, which is surely the best way in which to inhibit calcium resorption.

In moderate to severe hypercalcaemia, there is little argument about the need for rehydration, loop diuretics, and high dose glucocorticoids. The controversy arises once the serum calcium is controlled in the short term. The commonest causes of hypercalcaemia associated with malignancy are myeloma, carcinoma of breast, and small cell and squamous carcinoma of lung. Apart from squamous carcinoma of the lung these are chemosensitive tumours. Therefore, after rehydration and steroids, most oncologists would proceed to the best combination chemotherapy, oral or parenteral, for that particular patient's tumour rather than using calcitonin, oral phosphates, or mithramycin. The latter agent is notorious for causing unpredictable marrow suppression, which then precludes logical chemotherapy.

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Misinterpretation of toxocaral serodiagnostic tests

SIR,—Serodiagnosis for human toxocariasis is necessary as direct histological evidence of parasitic infection is rarely forthcoming. Dr G H Rée and others (25 February, p 628), however, quoting the work of Woodruff[1] and De Savigny *et al*,[2] state that over 2% of the population of Britain have been infected with *Toxocara* spp.

Woodruff and de Savigny *et al* indicate a seropositivity of over 2% in the adult population or blood bank sera detected by skin testing and supported by indirect immunofluorescence on formalin fixed second stage *T canis* larvae[3] or enzyme linked immunosorbent assay (ELISA) tests using the excretions-secretions of in vitro maintained second stage *T canis* larvae as antigen. Seropositivity can only be taken as synonymous with past or current infection if it is known that the diagnostic test is specific.

We have recently shown that parasite derived human A and B blood group like substances are present on the outer surfaces of in vitro maintained second stage *T canis* larvae.[4] This outer surface is dynamic, and turnover of antigens occurs along its entire length with the subsequent release of antigens into the environment. This phenomenon is dependent on both temperature and metabolism.[5] Doubt must be cast on the use of such larval microprecipitation tests for the diagnosis of human toxocariasis as the presence of A and B like substance will interfere with the specificity of the test. Moreover, the interpretation of microprecipitation tests which depend on the attachment of antibody to the outer (epicuticular) surfaces using living larvae could be misleading unless physical conditions such as temperature are controlled. Dr Rée and others state that the ELISA test using excretory-secretory products of in vitro maintained second stage *T canis* larvae "is both sensitive and specific." We are in agreement that the use of excretory-secretory larval products increase both sensitivity and specificity. The toxocaral ELISA test can, however, still be misinterpreted because *T canis* excretory-secretory products, produced by the method of de Savigny contain parasite derived human A and B blood group like substances, which might interfere with the sensitivity of the test when human sera with anti A or anti B isohaemagglutinins are tested. The human prevalence of these haemagglutinins must compromise the specificity of the test when employing excretory-secretory products as antigen. We have recently demonstrated such reactions using excretory-secretory products as antigen on ELISA. Further analyses and purification of secretory antigens are in progress in our laboratories in order to restore the validity of these tests.

We think that these comments endorse the importance of continued testing of sera from patients suspected of having toxocariasis. At present it is only by investigating such sera (including their blood group) in relation to the antigen(s) used for serodiagnosis that the true prevalence of human *Toxocara* spp infection can be discovered.

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[1] Woodruff AW. Toxocariasis. *Br Med J* 1970;iii:663-9.
[2] De Savigny DH, Voller A, Woodruff AW. Toxocariasis: serological diagnosis by enzyme immunoassay. *J Clin Pathol* 1979;**32**:284-8.
[3] Bisseru B, Woodruff AW. The detection of circulating antibody in human toxocara infections using the indirect fluorescent antibody test. *J Clin Pathol* 1968;**21**:449-55.
[4] Smith HV, Kusel JR, Girdwood RWA. The production of human A and B blood group-like substances by *in vitro* maintained second stage larvae: their presence on the outer larval surfaces and in their excretions/secretions. *Clin Exp Immunol* 1983;**54**:625-33.
[5] Smith HV, Quinn R, Kusel JR, Girdwood RWA. The effect of temperature and antimetabolites on antibody binding to the outer surface of second stage Toxocara canis larvae. *Mol Biochem Parasitol* 1981;**4**:183-93.

Early diagnosis and treatment of steroid induced avascular necrosis of bone

SIR,—Two points arise from Mr J E Nixon's review of steroid induced avascular necrosis of bone (10 March, p 741).

Firstly, no firm evidence exists that the lesions result from interruption of the blood supply. It has been assumed that since the lesions resemble those that do occur after such an insult—for example, after transcervical fracture of the femoral neck—that impairment of the blood supply also causes the bone necrosis that may be a consequence of steroid treatment. The correct term for this is steroid induced osteonecrosis.

Secondly, Mr Nixon gives the impression that osteonecrosis may be detected and treated at an early stage. This is misleading because the natural history of the condition is not completely understood. Studies of a similar condition, caisson disease of bone (dysbaric osteonecrosis), support this view.[1]

The study of serial long bone radiographs of deep sea divers has shown that over three quarters of those with juxta-articular lesions are asymptomatic and that the radiological appearances may remain unchanged for many years. There is also no certain method of determining which of them may progress to disruption of the joint surfaces. The information from the registry on both divers and compressed air workers suggests that symptoms only develop once disruption of the joint surface has occurred and therefore at a stage when progression to a secondary degenerative osteoarthritis is no longer preventable by conservative surgery.

Since dysbaric osteonecrosis closely resembles steroid induced osteonecrosis it would seem unwise to suggest that patients with early stage juxta-articular lesions should