incorrect assessment of fetal growth rate or risk. If labour has not occurred by 40-41 weeks of gestation, the cervix should be examined to assess its favourability and so help choose between a trial of labour and operative delivery. Obstetric guidelines must be tentative, because no one obstetric team has yet accumulated enough experience based on consistent obstetric practice to justify any conclusions. The difficulties of accumulating clinical experience can be overcome by referral of the problem to the local team with most skill and interest in the condition.[10]

The general practitioner and the paediatrician must be told that placental sulphatase deficiency was found in the pregnancy, so that when the skin changes develop they are treated with topical urea, an appropriate treatment for ichthyosis. Topical corticosteroid treatment is unnecessary and its continued use might reduce growth. The reptilian appearance of the skin in X-linked ichthyosis has been well illustrated[4 11] and may suggest the diagnosis. The onset of the condition in a boy in the first year of life and its relative constancy thereafter provide one clue, especially if about half of the other males in the family are affected in a fashion consistent with X linkage. Another clue should be the obstetric history. The correct dermatological diagnosis may be difficult to make on clinical grounds.

Between 25 and 100 patients with placental sulphatase deficiency or X-linked ichthyosis can be expected in a population of 250 000 to 500 000 served by one British district general hospital, so that there is a large potential load of diagnostic tests. Unfortunately, there is no validated easy diagnostic method for X-linked ichthyosis suitable for routine clinical biochemistry laboratories. Enzyme assays on skin or cultured skin fibroblasts[7] are relatively complex and expensive. Recently, cholesterol sulphate measurements have been used.[12] The relatively high incidence of this deficiency and its clinical features suggest that appreciable benefit should come from the provision of some means for precise diagnosis. This is already practicable in obstetrics but difficult in dermatology. What remain to be determined are the most effective means of improving the clinical care of affected patients. At very least, however, the documented experience available should help doctors reach a tentative diagnosis on purely clinical grounds.

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Renal failure after contrast radiography

Twenty years ago most doctors came to believe that patients with impaired renal function should not be investigated by intravenous urography. The procedure was thought unlikely to provide adequate detail for diagnosis and also to be possibly harmful. These doubts disappeared with the use of larger doses of better contrast medium, more rapid injection, and the use of tomography. Even in patients with severely impaired renal function or very raised blood urea concentrations intravenous urography can and often does give information of diagnostic or therapeutic importance.[1 2] In addition to nephrograms to show the kidneys and pyelograms to show the collecting systems even blood vessels can be shown if the contrast agent is injected into the vascular system quickly at high concentration. In part, this change in view is due to an increased awareness of the importance of the nephrogram.[3 4] Nevertheless, the earlier concern about whether contrast agents cause toxic renal damage seems to have persisted, especially in the United States, and several recent studies have reawakened that anxiety. Concern about toxicity may cause some clinicians or radiologists to delay an investigation that is likely to show the nature of a problem—and their patients may deteriorate undiagnosed and untreated.

So how dangerous are the contrast agents in current use? In any series of sick patients some will develop acute renal failure, either as an associated "idiopathic" event or from one of several types of adverse reactions to drugs.[5] The occasional patient who develops renal failure after examination with contrast medium does not prove that the agent caused the renal impairment, but the association should not be dismissed as coincidental simply because proof of causation is difficult to obtain.

A recent paper from the United States reports a prospective study of patients having angiography performed to visualise structures other than the kidney.[6] In some of these patients (mostly those with pre-existing renal dysfunction) the techniques and the doses used reduced renal function, and in some patients this renal dysfunction caused morbidity. The investigators looked at the plasma creatinine concentration, the urine deposit, and a late *x* ray film of the renal areas. (The plasma creatinine concentration was considered as reported and also as the logarithm but not as the more physiologically logical reciprocal.) Not surprisingly, those patients with initial high concentrations of creatinine proved

most likely to develop rises of more than 90 μmol/l (1·0 mg/100 ml), for relatively less damage will produce greater rises of plasma creatinine concentrations when renal function is already impaired. Nevertheless, a 33% incidence of rises of more than 90 μmol/l in those with high initial plasma creatinine concentrations (over 220 μmol/l (2·5 mg/100 ml)) certainly indicates a common problem. In contrast, only 2% of patients with less abnormal or normal initial function showed rises of the same order. The authors concluded that the contrast agent was toxic and the renal failure was a real event. Another recent review concluded that little useful was obtained from contrast studies in acute renal failure and that the risk was considerable and far outweighed the benefits,[7] and similar if less dogmatic recommendations have recently come from the British group at St Bartholomew's Hospital that were previously[2 3] enthusiastic advocates of contrast radiology.

In contrast recent British reviews that have reached publication suggest that it is diagnostic uncertainty that is dangerous and that any dangers of contrast studies are offset by the merits of knowing what condition a patient has got, or more often what he has not got—obstruction. Part of the explanation for recent changes in opinion in the United States and in relatively well funded London units, working with staff having a specialised interest in renal radiology, is the improvement in non-invasive techniques, such as ultrasound, radionuclide, and computed tomography scanning—but we should remember that facilities for these latter techniques are inadequate or unavailable in many parts of the world outside North America and London. Adoption of the extreme caution now seen in the United States would mean a major change in policy for most British units. Furthermore, uncertainty about obstruction may persist even when the renal tract has clearly been shown to be non-dilated,[4] and an abnormal nephrogram may be a diagnostic clue in cases of calculus anuria in patients in whom obstruction would be missed with ultrasound or computed tomography.

When contrast studies are considered important, whether for renal or non-renal indications, and especially if the patient has some impairment of renal function, how can any hazards be reduced? The rare cases of severe damage from an allergic vasculitis or a glomerulonephropathy are difficult to predict and therefore to prevent. Acute renal failure from direct toxic effects should be unusual if care is taken about dose.[8] Special care should be taken with children. Most deteriorations in renal function are probably due directly or indirectly to the massive solute loads induced during these procedures. The contrast acting as a solute causes intracellular dehydration and extracellular fluid expansion, which with the osmolar load causes a large diuresis.[9] This in itself may well create a renal crisis in a patient who already has just adequate hydration or mild dehydration. This risk may be reduced by replacing fluid losses before the procedure, by avoiding dehydration and "no fluid" regimens, and by giving extra fluids before, during, or after the investigation. Further studies are needed to establish both whether such precautions will reduce or prevent problems and to decide whether fluid replacement should be as water by mouth, intravenous 5% dextrose, or some other electrolyte containing supplement. The policy of not adding electrolytes to fluids given during the procedure and of removing them from contrast agents is debatable—for the diuresis is a solute diuresis and removes substantial amounts of sodium and other electrolytes.[9] For the time being, while the results of such investigations are awaited, the priority should be ensuring good hydration, but the choice of the most appropriate fluid to use will have to be a matter for individual judgment—perhaps water by mouth and 0·45% saline intravenously. No regimen can be recommended as a recipe for all patients. Evaluation of a patient's hydration requires clinical skills at the bedside. Some patients about to undergo contrast radiography will be dehydrated, others will have heart failure or have overhydration for other reasons. Only for the majority who will be correctly hydrated can anyone design a standard perioperative programme.

Finally, clinicians need to remember that contrast procedures are often expensive, may be uncomfortable for the patient, and are not without some risk. More thought and study are needed on how to reduce the risk, but as more equipment and staff become available for non-invasive investigative techniques there should be less need for angiographic and other more hazardous investigative procedures. In other patients the use of clinical skills and logical thinking before reaching for a radiology request form may avoid some procedures. After the request form is written the clinician still has a responsibility to take enough care with his patients' management before, during, and after the investigations if he is to keep any risk to a minimum.

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Which inguinal hernia repair?

Modern surgical repair of inguinal hernia is less than 100 years old. In that time the methods have passed through three phases. The original techniques were developed by pioneers such as Bassini[1] and Halsted,[2] who described the suturing together of the margins of the defect. There followed a second phase of imaginative solutions to the problem of closing the defect using flaps, darns, inserted fascia, skin, and tantalum gauze. The methods now in vogue all depend on the drawing together of tissues already present, the elimination of tension, and the use of non-absorbable sutures.

Three techniques are popular. Tension may be eliminated when using the original Bassini technique by making a relaxing incision[3] which allows the upper edge to be drawn down easily to the inguinal ligament. The relaxed upper edge is slid down in the Anson and McVay method,[4] to be sutured to the ligament of Cooper and the anterior femoral sheath.