attack on the origin of the dissection, reported a mortality of 21% in 179 patients. These results from Houston excited admiration and optimism but were not matched from other centres. Indeed, Hume[3] reported a mortality of 64% after surgery, though the surgical group did fare better than the unoperated patients, of whom no more than 4% survived beyond two months. If we leave aside questions of technical expertise (where the Houston group are certainly masters) this startling difference in the results crystallises the central problem in evaluating treatment for this condition—selection of cases. Hume's patients had consisted of all admissions to a general hospital, together with coroner's cases for the area; whereas the Houston series came from an enormous referral practice and contained three times as many dissections of the most favourable type—the postsubclavian or type III. The natural incidence of dissections is exactly the reverse—three times as many arch and ascending aortic lesions as descending—and Hume's series contained exactly that proportion of patients with the deadly proximal type of lesion.

Against that background, in the mid-1960s Wheat[4] argued that since surgery gave better results in chronic dissections than in acute ones (where the aortic wall is extremely friable) time should be bought by lowering the blood pressure in these often hypertensive patients and reducing the force of the cardiac impulse. Using a mixture of arfonad, guanethidine, and reserpine he reported success for up to 15 months in six patients, one of whom then required elective aortic root and valve surgery. This technique was adopted widely with minor modification of the drug regimen. Later reports, however, did not match the early promise, and Wheat next suggested[5] that the drug regimen should be used in all patients initially but that those with aneurysms of types I and II, especially with valve or pericardial complications, should be operated on urgently; whereas patients with type III dissections could be managed conservatively unless dire complications threatened. Appelbaum *et al*[6] reported that 26% of a series of 108 patients with type I and II lesions treated surgically died compared with 88% of those treated medically. Their early results for type III lesions were much the same whether the treatment was medical or surgical. In the long term, however, more of the surgically treated group survived, and they recommended that acute descending dissections should be treated medically in the first instance but then as soon as possible by operation. Using a combination of arfonad and propranolol both before and after operation Reul *et al*[7] had a death rate of 21% for dissections of the descending aorta.

A recent paper from Mills *et al*[8] has reported less happy results of surgical treatment. In both type I and type III dissections they found no difference in mortality between patients treated surgically and medically. Strangely, the best surgical results were in patients with type II dissections, 60% of whom were alive at one year. Mills's patients included a group treated non-specifically, and among these patients those with type III aneurysms did at least as well as the group treated with the Wheat regimen.

These recent studies, like that from Houston in 1965, were based on series with a high proportion of the favourable type III dissections. The true death rate in the community of ascending and arch dissections must be very high indeed, and treatment by vascular surgeons at present is only scratching the surface of the problem. Furthermore, none of these accounts mentioned the incidence of ischaemia of the legs or its management, though this may be a problem in as many as 30% of patients with dissections.

At present, then, the approach to treatment seems to favour surgery (resection and graft replacement, sometimes with valve surgery) for type I and type II dissections; but patients should be treated from the outset by hypotensive and inotropic drugs, and these should be continued after operation. Patients with type III dissections should be treated with drugs alone unless there are complications, when again resection of the aorta and graft replacement becomes the best form of treatment. When there is ischaemia of the legs immediate operation at the aortic bifurcation is needed as well as treatment of the dissection itself.

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[2] De Bakey, M E, *et al*, *Journal of Thoracic and Cardiovascular Surgery*, 1965, **49,** 130.
[3] Hume, D M, and Porter, R R, *Surgery*, 1963, **53,** 122.
[4] Wheat, M W, *et al*, *Journal of Thoracic and Cardiovascular Surgery*, 1965, **50,** 364.
[5] Wheat, M W, *Progress in Cardiovascular Diseases*, 1973, **16,** 87.
[6] Appelbaum, A, Karp, R B, and Kirklin, J W, *Annals of Surgery*, 1976, **183,** 296.
[7] Reul, G J, *et al*, *Archives of Surgery*, 1975, **110,** 632.
[8] Mills, S E, *et al*, *American Journal of Surgery*, 1979, **137,** 240.

Sex and intersex

Among the saddest of congenital anomalies are those leading to indeterminate or neutral sex. In its incomprehension society reacts with cruelty, and these unfortunate people have often had little compassionate support even from doctors—through a lack not of sympathy but of scientific understanding; the very term "hermaphrodite" suggested that here was the incomprehensible meeting place of clinical medicine and classical mythology. Recently, however, the intersexual states have gradually been defined and now the remaining major mysteries of sex differentiation seem to have been solved.

The advances started with Barr's discovery of the sex chromatin body when he was studying exhaustion of the cat's hypoglossal nucleus.[1] This was predictably followed by chromosome analysis and by 1970 the broad range of sex chromosome abnormalities had been established, with three patterns (XO, XXY, and XX/XY) representing sex chromosome neutrality. The concept of neutrality was important as it guided clinicians away from search for a *true* sex, which had so often proved futile, to an appreciation of sex differentiation as being a range without rigid division. The few XX/XY cases were the most interesting of the neutrals, for marker studies proved that most resulted from double acts of fertilisation, thus distinguishing them from mosaics.[2] The occurrence of dispermic conceptions raised the possibility of multiple paternity—though the mythological term "chimaera" was perhaps unfortunate.

Chromosomes, in man the Y particularly, control gonadal differentiation; but even when the chromosome abnormalities had been defined there remained a major group of puzzling intersexes in which the abnormality seemed to reside in the gonads. In some the gonads were contrary to the chromosome sex, and in others ovarian and testicular tissue coexisted—this is so-called "true" hermaphroditism, usually associated with an XX sex chromosome complement. Again, the crucial advance came from work on another topic: transplantation studies on inbred mice demonstrated a sex-linked rejection factor carried by males—the H-Y antigen, now known to be

present in man.[3 4] Hitherto the Y chromosome had been regarded as the prerequisite for testicular differentiation, but the H-Y gene and the antigen it controls now appear to be the active elements, probably capable of transfer to other chromosomes or other cell surfaces and possibly subject to control from other gene loci. Techniques are difficult and the picture is far from complete, but this approach seems likely to explain many of the incompatibilities reported between chromosomal and gonadal sex, including sex reversal and ovotesticular states.

Given that the chromosomal sex and the gonadal sex are normal and compatible with each other, there are only two basic types of intersex: inappropriate masculinisation of chromosomal and gonadal females and failure of expected masculinisation of chromosomal and gonadal males. The occasional masculinisation of females by abnormal androgenic hormones has been easy to understand, masculinisation by 21-hydroxylase deficiency (the classical "congenital adrenal hyperplasia") being the outstanding example. Much more puzzling have been the extraordinary cases in which, despite the presence of XY chromosomes and testes, masculinisation fails to occur—the "testicular feminisation" or "androgen insensitivity" syndrome. It is now established that the "incomplete" form of this syndrome, in which embryonic masculinisation fails but pubertal masculinisation occurs, is due to deficiency of another steroid enzyme, 5α-reductase.[5] Embryonic masculinisation requires dihydrotestosterone, which is absent; pubertal masculinisation occurs directly through testosterone and is therefore unaffected. The "complete" androgen insensitivity syndrome seems on circumstantial evidence to represent a receptor rather than an enzymatic failure, as both embryonic and pubertal masculinisation fail despite high plasma testosterone concentrations. Now that the various enzyme defects have been defined the imprecise terms should make way for precise designation of the chemical abnormalities.

Because of society's conventions the clinician must still assign an individual to one or other sex—not usually according to fundamental biological principles but merely from a practical standpoint. But he can now do this with relatively complete scientific understanding, which is important for effective psychological support. Thus elucidation of the scientific basis of sex differentiation can contribute to compassionate and humanitarian care for those with such aberrations, and it also suggests a possible chemicophysical explanation of some deviant forms of sexual behaviour.[6]

A source of confusion is sex change. "Correction" of the sex of registration can be made and individuals may change their social sex, but change of chromosomal and gonadal sex has not been reported in man or in other mammals,[4] though it is commonplace in molluscs. Various hormone influences can, of course, produce a crop of ambiguous secondary sex characteristics, the most recently documented and publicised[7] being those produced at puberty in boys with congenital 5α-reductase deficiency. In biological terms, however, this is merely a form of delayed manifestation of sexual identity.

The liberally educated doctor of today should have a broad understanding of sex differentiation and its failures. But our professional responsibilities for education extend beyond our own ranks; until society knows that sex is not a binary phenomenon but a set of characteristics of bimodal distribution, it cannot show understanding and compassion to those who appear freaks in terms of the usual conventions. Recent serious attempts to inform the lay public about these matters[8 9] are to be welcomed, supported, and given only constructive criticism.

[1] Barr, M L, and Bertram, E G, *Nature*, 1949, **163**, 676.
[2] Bain, A D, and Scott, J S, *Lancet*, 1965, **1**, 1035.
[3] *British Medical Journal*, 1979, **1**, 704.
[4] Short, R V, *British Medical Bulletin*, 1979, **35**, 121.
[5] Walsh, P C, *et al*, *New England Journal of Medicine*, 1974, **291**, 944.
[6] *Sex, Hormones and Behaviour. Ciba Foundation Symposium 62*, ed R Porter and J Whelan, p 382. Amsterdam, Excerpta Medica, 1979.
[7] Imperato-McGinley, M D, *et al*, *New England Journal of Medicine*, 1979, **300**, 1233.
[8] *Horizon*, 21 May 1979, BBC2.
[9] Goldwyn, E, *Listener*, 24 May 1979, p 709.

Surgical needles

Surgeons are first and foremost craftsmen, with just as much pernickety interest in their tools as the most obsessional carpenter. Any of the large catalogues of surgical instruments will show the almost infinite variations of size, shape, and length of, say, artery forceps or dissecting forceps, each eponymously named, to pander to their individual requirements. Surgeons will debate for hours over the size and properties of their sutures, the potency of their skin disinfectants, and the colour of their operating drapes. Even the surgical needle now comes in well over 100 different patterns —no doubt reflecting the personal requirements of the prima donnas of our hospitals.

The fascinating story of surgical needles has recently been reviewed by Trier.[1] Needles are among the most primitive of man's appliances, the earliest, eyed and made of bone, having appeared between 20 000 and 35 000 years ago. The use of needles for surgical purposes is mentioned in the Edwin Smith papyrus, now nearly 5000 years old.

Needles threaded with fine wool, silk, sinew, and other materials have been used by surgeons for centuries, and the first suture swaged into a needle was invented by a Mrs Ella Gaillard over 100 years ago. To the surgeon the eyeless needle has the great advantage of avoiding the need to pull a double-suture strand through the wound, and time is saved by not having to thread it. By being attached to a length of suture material the needle is also less easily lost in the depths of the chest or abdomen.

Most modern surgical needles are manufactured from stainless steel wire, but recently a metallised monofilament nylon suture serving as its own needle has been introduced for microsurgery. The variations in the characteristics of needles run into dozens of permutations of size, diameter, curvature, and cross-sectional shape. Needles may have a cutting edge, and may be round bodied or blunt (the latter type being used for friable tissues such as the liver). Since most surgical needles are now swaged to sutures they are disposable, and providing a new and undamaged needle with each suture strand makes little difference to the cost of the suture.

With such an overwhelming variety of needles to choose from how should the surgeon decide which to use? Perhaps in many cases he simply takes the needle his theatre sister passes to him. As Trier[1] points out, however, certain requirements must be met if we assume that the surgical needle should introduce no damage beyond that inflicted by the suture on the tissues. The needle should make a hole in the tissue only large enough to allow the suture material to go through, so