to design such models is already under way using bioengineers and moulding technicians as well as surgeons. Although it is reasonably straightforward to simulate anastomosis of hollow viscera, a much greater challenge faces design teams in creating simulated tissues for dissection and resection.

The structure of surgical training in Britain is undergoing considerable change at present. Entry to a surgical career in the future seems likely to be preceded by a more thorough assessment of the candidate's potential as a surgeon. Aptitude testing—including evaluation of psychomotor skills such as manual dexterity and eye-hand coordination—is already being used in Holland in selecting surgical trainees.[7] The structure of professional examinations for surgeons is also being radically altered, and discussion is taking place about the possibility of testing manual skills as part of the surgical fellowship examination. Simulation may well find a place in these assessments of trainees, and its importance in surgical training seems destined to increase.

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Genetic testing for Huntington's disease

Internationally agreed guidelines are being followed

Recent advances in mapping the gene for Huntington's disease have for the first time made accurate prediction possible for those at risk of carrying the gene for this disorder.[1] A test for the gene has been foreseen and its implications widely debated for some years,[2] but only now is it becoming possible to evaluate its use and the associated problems in practice. Over the past year a series of publications has begun to give us a clear picture of this difficult issue, which is in many ways a prototype for genetic prediction in other late onset genetic disorders, such as some of the hereditary ataxias and familial Alzheimer's disease.[3-5]

For several years after the Huntington's disease gene was first mapped to the short arm of chromosome 4 investigators wisely held back from using this information clinically, and the widespread discussion during this period among professionals and lay groups has resulted in an important set of guidelines, which have recently been published in the *Journal of Medical Genetics*[6] and the *Journal of the Neurological Sciences*.[7] Perhaps the most valuable aspect of these guidelines is that they represent the consensus of a working group of both family members (the International Huntington Association) and professionals (the World Federation of Neurology Research Group on Huntington's Disease). In Britain a multidisciplinary group of professionals concerned with predictive testing has drawn up recommendations for good practice that are complementary to the international guidelines.[8] These recommendations emphasise the need for expert counselling in association with the test; the need for freedom from pressure from relatives, employers, or insurance companies; and the importance of complete confidentiality. They advocate that children should not be tested.

How are these guidelines actually being observed in practice? Several series of predictions from major centres have now been published,[3-5] and probably at least 200 predictive tests have been carried out world wide. The World Federation of Neurology Research Group's meeting in Vancouver in July 1989 gave a valuable opportunity to examine problems and, most importantly, to hear first hand accounts of the experience from some of those at risk of Huntington's disease who had undergone testing.[9]

Even though these results are necessarily preliminary, we may already learn much from what has happened. In almost all cases so far testing has been preceded and accompanied by skilled counselling, which has been valued by those being tested and which may have contributed to the lack of serious adverse effects so far encountered. Clearly, counselling must be regarded as an integral part of the testing procedure, and in its absence testing would fall short of the expected standard of practice.[10] As laboratory aspects of testing for Huntington's disease become simplified and tests become available for other late onset neurological disorders this point needs to be remembered, especially if economic pressures were to be exerted to cut down on all but the minimal laboratory procedures.

The principal worry of those concerned in predictive testing for Huntington's disease has been the possibility of serious emotional and psychological effects in people whose result indicates a high risk of having the Huntington's disease gene. Although it is too early to determine long term results, there has in fact been a striking lack of such problems.[3] This probably reflects in part the cautious approach to testing and the provision of counselling and support by the centres concerned, but at least as important may be the resilience of those being tested, most of whom have waited many years for such a test and have already prepared themselves mentally for an adverse result. From surveys done so far,[11] particularly a study from Manchester,[12] it is clear that uptake of the test is low, even when those at risk are systematically informed of its availability. Those currently being tested are therefore a self selected group, and those who decline to be tested might be less able to handle the severe stresses entailed.

It would be misleading to imply that predictive testing for Huntington's disease has been completely free of problems. Most of the difficulties expected have occurred, as well as some that were not foreseen.[13] One study of these problems in a large series of people who requested testing reported several requests for testing of children (mostly by parents) and for testing before adoption[14]; all such requests were declined. The question of childhood testing for Huntington's disease and the powerful arguments against it have been discussed in detail in a review from the Vancouver group.[15] An important problem has arisen when individuals who have proved at first interview to be clinically affected request testing; telling such people that they not only have the gene for Huntington's

disease but that they already have the disease has been difficult. Hence, most of the problems encountered have been in the clinical and counselling sphere rather than laboratory related.

In general most of the reported studies of predictive testing have closely followed the principles laid out in the guidelines. Perhaps inevitably some recommendations have not been fulfilled, notably that testing should be available equally to people of all regions and from all countries. Many European countries have not begun testing, while in the United States problems of continued funding have led at least one of the original regional centres to stop offering the test. In Canada a nationwide network of testing centres has been set up, with a common set of procedures and regular communication among units.[16] A comparable network is now evolving in Britain: there are now around 12 such centres, all following a similar pattern of testing, counselling, and support, with a co-ordinating group and a common core protocol for service use.

These developments should allow continued audit of the process of predictive testing for Huntington's disease and will eventually provide information on how this process has affected the lives of those who have undergone testing. New advances—notably the detection of the specific mutation(s) for the disorder (which is likely in the near future)—will be able to be incorporated into the testing framework, and we should learn much that is relevant to prediction in other disorders. Whether the system that has evolved will be able to continue and develop will also be a test for the changes proposed in the NHS. So far, as with other developments in medical genetics, the system has proved flexible and effective; we hope that it will continue to allow the steady evolution of preventive measures such as testing for Huntington's disease and other serious genetic disorders.

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Pelvic inflammatory disease

A sexually transmitted disease with potentially serious sequels that is often treated poorly

Pelvic inflammatory disease in women is inflammation of the upper genital tract. The term embraces endometritis, salpingitis, and salpingo-oophoritis, together with spread to the peritoneum as peritonitis and along the paracolic gutters to cause the Fitzhugh-Curtis syndrome of perihepatitis. It is virtually always due to ascending infection through the cavities of the cervix, uterus, and fallopian tubes; on histological examination the endometrium is always affected.[1,2]

The natural barrier to pelvic infection is the cervix, where a downward flow of the mucus and the action of cilia are augmented by the production of a lysozyme. Aided by the presence of cervically secreted IgA the lysozome hydrolyses the peptiglycan links of micro-organisms, allowing osmotic destruction.[3] The cervical barrier may be compromised after miscarriage, abortion, childbirth, cervical surgery, and in the presence of an intrauterine contraceptive device. The risk of pelvic inflammatory disease after termination of pregnancy is of the order of 2%, but this increases up to 10-fold in women with asymptomatic sexually transmitted diseases.[4] Few centres, however, screen women undergoing termination for asymptomatic infections or give prophylactic antibiotics—which seem to be effective in these circumstances.[4] The risk of pelvic inflammatory disease in women fitted with intrauterine contraceptive devices has probably been overstated. There is a transient risk at the time of insertion,[5] but the increased relative risk (approximately 1·0-3·0) is probably due to a diagnosis bias.[6]

The most common cause of pelvic inflammatory disease is coitus. Many organisms that alone do not seem to be sufficiently mobile may ascend the genital tract on the back of spermatozoa. These include *Neisseria gonorrhoeae*,[7] *Ureaplasma urealyticum*,[8] *Chlamydia trachomatis*,[9] and several anaerobic species.[10] The uterine contractions that accompany orgasm may also possibly draw spermatozoa and micro-organisms into the uterus.[11]

Pelvic inflammatory disease has undoubtedly become more common. In the 1960s gonorrhoea seemed to be the main cause, and over a period of 20 years this infection became at least three times more frequent in most parts of the world.[12,13] Up to one fifth of women infected with gonorrhoea developed pelvic inflammatory disease.[14] In the 1980s *C trachomatis* has become a more important cause; it now accounts for up to half of the cases of pelvic inflammatory disease in Europe[15] and two fifths of the women treated in hospital in the United States.[16] Chlamydial disease (as judged by the rate of non-gonococcal urethritis in men) has increased fourfold in the past 30 years in England and Wales.[12,17] Overall, sexually transmitted diseases now underlie about three quarters of all cases of pelvic inflammatory disease. In industrialised countries the incidence of pelvic inflammatory disease is now 10 to 13 per 1000 women of reproductive age, with the peak incidence of 20 per 1000 occurring in those aged 15-24 years.[18,19]

Despite some problems of definition,[18] the varying criteria for diagnosis in published reports, and follow up that is often