Changes in the law on abortion

Won’t produce radical changes in practice

On 18 October the House of Lords finally passed, unamended, the Human Fertilisation and Embryology Bill, including the controversial change in the upper gestation limit for induced abortion. The bill received royal assent on 1 November. When the ground for termination is a risk to the physical or mental health of the pregnant woman or her existing children the law now requires that the pregnancy should not have exceeded its 24th week. This corresponds to the professional advice on viability. The 24 week limit does not, however, apply when there is risk of grave permanent injury or death to the mother or a substantial risk of serious handicap in the child. Does this then permit more liberal practice than hitherto, unconstrained by the Infant Life (Preservation) Act 1929?

No change is likely in respect of grave maternal illness as obstetricians have always been able to conclude a pregnancy prematurely when the mother is seriously ill. In the case of impending or actual eclampsia, for example, delivery is indicated but would not normally be notified under the 1967 Abortion Act as the intention is not to abort the fetus but to save the mother, thus maximising the chance of survival of the fetus.

The answer is more complex when the risk is of serious handicap in the child. Firstly, planned screening for malformation—by chorion villus biopsy, amniocentesis, fetal blood sampling, or anomaly scanning at 18-20 weeks—will usually provide a reasonably definite diagnosis by 24 weeks; everything possible should be done to streamline such services, but unavoidable delays will occasionally occur. Secondly, a structural abnormality may be discovered incidentally much later—for example, in the course of ultrasonography to monitor for fetal growth—and there may be associated chromosomal anomaly. Thirdly, although ultrasonic techniques are always improving, the exact nature of a malformation, or even whether it is present or not, may still be difficult to determine; certainly the extent of impairment that a child would experience may be impossible to predict accurately even in well documented and common conditions such as Down’s syndrome. Sometimes, as in the case of suspected microcephaly, the diagnosis will become more secure with advancing gestation, and unnecessary termination may be avoided by delaying the decision for a few weeks. For some conditions, such as hydrocephalus or non-immune hydrops, the prognosis will be worse the earlier in pregnancy the condition presents.

The description “seriously handicapped” encompasses a wide range of conditions. When the fetus has a lethal condition such as anencephaly, renal agenesis, or trisomy 18 there is no dilemma for the obstetrician in agreeing to a mother’s request to be delivered whenever the diagnosis is made, and, indeed, the obstetrician may advise prompt delivery in the case of anencephaly to avoid shoulder dystocia. At the other extreme, when a serious condition is discovered that may be amenable to corrective surgery in utero or, more often, after delivery, appropriate arrangements for delivery are required. It is less clear how to proceed in the event of incurable handicapping conditions that are not necessarily lethal, such as some osteochondrodysplasias, central nervous system malformation, inborn errors of metabolism, or chromosomal anomalies.

When such a condition is diagnosed in the second trimester it was and still is legal to terminate the pregnancy if the mother wants termination. In the third trimester the question of whether and when the obstetrician has the option, ethically, to withhold aggressive intervention, to hasten delivery, or, indeed, actively to cause the death of the fetus, has recently been discussed by two New York doctors. They have produced a set of recommendations taking into account the security of the diagnosis and the expected quality of life of the child in determining the ethical obligations of the mother and the obstetrician. Among other things, the authors discuss and advise against the use of cephaloectomy in the case of hydrocephaly. They admit that their proposals are arbitrary and record that their actual management (presumably influenced by the wishes of the mother) more often included termination than did their recommendation. It remains to be seen whether British women will wish to make the same choices or whether obstetricians will readily accede to their wishes. Relevant factors will be the expected severity of the physical and mental impairment, the life expectation of the child, the gestation at diagnosis, and the mother’s obstetric history. A mother who already has a seriously handicapped child usually has a firm view of whether or not she wants termination in the event of a recurrence. Including the paediatrician, the geneticist, or the surgeon in counselling will often be helpful and necessary. Decisions should never be hurried.

In Scotland, where the Infant Life (Preservation) Act 1929 never did apply, less than two terminations a year were performed after 24 weeks’ gestation in the years 1985-9.
relation to population these figures do not differ greatly from those in England, where only 23 terminations were performed after 24 weeks in 1989. They also suggest that obstetricians use Glover’s “ascending slope” view of the consideration to be accorded to the fetus, and that 24 weeks is near the top of the slope. This may well be due to the fact that though termination in the second trimester can be accomplished surgically or by use of hormones to cause the expulsion of a non-viable fetus, in the third trimester the obstetrician would have to take active steps to cause fetal death—for example, intra-cardiac injection. Liberalisation of the law will probably not be used extensively to increase the number of late terminations. Nevertheless, the knowledge that a carefully considered, well documented termination for fetal malformation diagnosed after 24 weeks will fall within the scope of the abortion legislation is welcome.

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Recurrent hereditary polyserositis

Not necessarily familial or Mediterranean

The first report of recurrent hereditary polyserositis—“recurring attacks of a peculiar nature”—was published in 1908 by Janeway and Mosenthal. They described a 16 year old Jewish schoolgirl “without special neurotic inheritance” who had had febrile bouts associated with abdominal pain—with prodromal, crescendo, and recovery phases—from the age of 2 weeks. Subsequent reports of recurrent hereditary polyserositis included large numbers of Jewish (from the United States and Israel), Armenian (United States), Turkish, and “fair skinned” Arab patients. Despite the now substantial number of accumulated reports and the growing list of terminologies many questions about this disease remain unanswered. (One is why this condition was not recorded in historical works from the Middle East and Mediterranean littoral or in the Bible or Koran, for the “attacks” are dramatic.)

The size of the problem is unknown. The ethnic groups mainly affected are clearly defined, but sporadic cases are widely spread—in, for example, Ireland, Holland, France, Germany, Sweden, the Soviet Union, India, Japan, New Zealand, and Australia, although in the last two countries there was evidence of Middle Eastern ancestry. Mendelian recessive inheritance seems likely, although a dominant form has been described. Significant male to female predominance is usual; incomplete penetrance might be more common in women. Amyloid AA deposition (widely distributed and, most importantly, affecting the kidneys) is certainly thought to be more common in affected Turks and Sephardic Jews, but many reports are from renal units and bias seems probable. With the exception of Israel, the high prevalence of recurrent hereditary polyserositis in areas that are heavily affected has resulted from natural gene drift; a selective advantage associated with the responsible gene therefore seems likely, but what is it?

An “infective” agent (operating against a suitable genetic background) remains a possibility; Is recurrent hereditary polyserositis related to Behget’s disease? Many investigators have concentrated on an immunological defect. A C5a inhibitor deficiency in joint and peritoneal fluids might be causally related to the acute attacks. Defective lipocortin protein(s) or abnormal formation and elimination of mono-hydroxy and dihydroxy fatty acids have been suggested; and abnormal metabolism of catecholamines is another possibility.

The most widely used clinical criteria are: firstly, more than four attacks (lasting 24-72 hours) of peritonitis or pleurisy, or both, in the presence of fever, often accompanied by arthropathy; secondly, absence of symptoms between attacks; and, thirdly, lack of a known causal or pathological factor. Clinically, the prevalence of the various symptoms and signs varies in different series. Abdominal pain and vomiting are common and diarrhoea is unusual. Over half of those with the condition experience pleurisy during an attack, 24-84% an arthropathy, and fewer a dermatological manifestation—most commonly erysipelas-like lesions affecting the legs, ankles, or dorsum of the feet. Many associated features have been described, and symptoms are often alleviated during pregnancy. The concentrations of several (non-specific) acute phase reactants are raised during an attack, but diagnostic tests—including neutrophil chemiluminescence, measurement of DNA antibodies (by an enzyme linked immunosorbent assay (ELISA)), a metaraminol provocation test, and measurement of plasma dopamine-β-hydroxylase activity—have all proved disappointing. Detection of amyloid deposition is usually based on Congo red staining of biopsy specimens (haemorrhage may complicate renal biopsy in amyloidosis), but encouraging results have recently been reported with a non-invasive technique using scintigraphy with serum amyloid P component labelled with iodine-123.

Colchicine (0-5-1.5 mg/day) was successfully used to prevent the attacks in 1972; its effectiveness was soon confirmed. Evidence that colchicine reduces the progression of renal disease (including amyloid deposition) came from a large prospective study (1070 “compliant” Sephardic Jews) in Tel-Aviv; proteinuria was used as the marker of amyloidosis (and the nephrotic syndrome). Other forms of chronic renal failure are also associated with recurrent hereditary polyserositis, and this should be taken into account in future studies. Nevertheless, amyloid deposition now seems preventable.

Further macroepidemiological and microepidemiological studies are required. Future clinical and molecular genetic studies should address the possibility that two or more separate immunological or metabolic defects are interwoven; Why is AA amyloidosis confined to only some affected groups? A simple non-invasive diagnostic test is also required. Preventive strategies might ultimately include genetic engineering, but in the immediate future an increased level of awareness of recurrent hereditary polyserositis among doctors and other health workers (especially in affected areas) must underly the most useful strategy: chemotherapy may then be initiated early. Colchicine (whose mode of action is not clear) has appreciable side effects, so a safe and effective alternative should be sought.

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