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Congenital anterior abdominal wall defects

See p 903

Environmental factors, improved prenatal screening, or poor data collection may explain changing incidence

Accurate epidemiological data about the incidence of congenital malformations is essential for studying underlying risk factors, implementing possible preventative measures, and providing services. The data reported by Tan *et al* in this week's *BMJ* (p 903) suggest that there have been substantial changes in the incidence of anterior abdominal wall defects in Britain, in particular an increase in gastroschisis (a paraumbilical defect with evisceration of abdominal contents) and a decline in exomphalos (a ventral defect with herniation of the intra-abdominal contents into the base of the umbilical cord and a covering peritoneal membrane).[1] Certainly, in our unit over the past few years there has been an increase in the number of fetuses with gastroschisis referred for prenatal diagnosis and delivery, from 24 in the six years to 1991[2] to 28 in the past three years.[3] Some, but not all, other reports have confirmed an increase in incidence of gastroschisis, with a small decline in birth prevalence of exomphalos,[4] but the substantial difference in incidence of the two types of defect reported by Tan *et al* is not supported by most other studies.

Tan *et al* obtained data from the Office of Population Censuses and Surveys (OPCS) and the National Congenital Malformation Notification Scheme. The figures quoted relate to the incidence of anterior abdominal wall defects at birth and do not include pregnancies ending in termination; this is because the OPCS data collection system on abortions does not allow for differentiation between gastroschisis and exomphalos. The authors note that there has been no change in the overall rate of therapeutic abortion for anterior abdominal wall defects over the period of their study and suggest that, because of the greater association with other abnormalities, pregnancies where the fetus has an exomphalos are more likely to be terminated, thus accounting for some of the difference in incidence reported.

Data from the North Thames West Congenital Malformation Register for the period 1990-3 gives an overall incidence of 4.3/10 000 for exomphalos and 1.6/10 000 for gastroschisis (L Abramsky, personal communication). The data included in this register are collected from multiple sources such that it is possible to differentiate between the types of abdominal wall defects as well as ascertaining pregnancies ending in therapeutic or spontaneous abortion. During this period, 55% of pregnancies with exomphalos and 23% of those with gastroschisis were terminated, the majority in both cases because of associated abnormalities. While the proportion of fetuses with gastroschisis and other abnormalities is unusually high (most other series reporting figures of around 5-10%[5 6]), these figures indicate that failure to ascertain accurate data on pregnancies ending in termination must account for a large part of the difference in incidence reported by Tan *et al*.

OPCS data about abortions are obtained from forms completed as part of the statutory notification process. This requires that diagnosis is reported, but it is not necessary for all the associated malformations to be recorded. Thus a fetus with an exomphalos and trisomy 18 may be recorded as trisomy 18 only. In North Thames West region from 1990 to 1993, 60 pregnancies in which the fetus had an anterior abdominal wall defect were terminated. According to Tan *et al*, the equivalent number for the whole of England and Wales for the same period was 163. It seems highly unlikely that in this period 37% of the terminations associated with an anterior abdominal wall defect occurred in North Thames West region, further demonstrating that underascertainment secondary to the method of statutory reporting contributes significantly to the difference in incidence reported.

While the difference in overall incidence of gastroschisis and exomphalos may not be as great as suggested by Tan *et al*, it does seem clear that, as reported by others, there has been a real increase in incidence of gastroschisis at birth.[4] There are several possible reasons for this. There has been an increasing use of routine scanning for fetal anomaly, so that most cases of anterior abdominal wall defect are now detected before birth.[7] Gastroschisis, unlike exomphalos, is most often an isolated abnormality with a good prognosis. Several factors—recognising the importance of close monitoring, ensuring delivery in a unit with easy access to paediatric surgical facilities, and general improvements in neonatal care—mean that the survival rate for fetuses with gastroschisis is usually reported as being about 90%.[2 5 6 8 9] Faced with a high chance of a good outcome, parents are therefore less likely to request termination of a pregnancy complicated by fetal gastroschisis, perhaps contributing to the increase in birth incidence.

Gastroschisis is usually sporadic, occurring most often in young, primiparous, socially disadvantaged women.[3 10] In addition, there are several reports describing a higher rate of smoking and drug misuse in women whose fetuses are found to have gastroschisis.[10 11] One view regarding the aetiology of gastroschisis is that it occurs as a result of a vascular accident in early gestation.[4 12] The reports of maternal cocaine ingestion associated with fetal gastroschisis support this aetiology, as there are several mechanisms by which this drug can potentially impair the circulation.[13] The epidemiological data support the theory that environmental teratogens may be

responsible for the increase in incidence of gastroschisis. Further structured prospective studies of pregnancies complicated by gastroschisis are required to define precise risk factors so that preventive measures may be taken. While this can be done on a relatively small scale in individual units, there is no doubt that countrywide epidemiological data would also be very helpful. It would seem that the statutory reporting system in England and Wales is currently inadequate for this purpose, and the development of more local (or perhaps national) congenital malformation registers such as exist in North West Thames region and some other regions should be encouraged.

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Intracoronary stents

Reducing restenosis after angioplasty

Coronary angioplasty has become an accepted treatment for coronary disease, with over 13 000 procedures performed in Britain in 1995 (410 000 in the United States). During the 1980s the overall primary success rate of angioplasty was 90%, with complications such as myocardial infarction, emergency surgery, and death occurring in up to 0.9%, 3.2%, and 1.2% of cases respectively, depending on the extent of coronary disease.[1] However, even with recent improvements in angioplasty technology, percutaneous revascularisation techniques have been limited by restenosis, predominantly through neointimal hyperplasia, but also through vessel shrinkage (adverse remodelling).[2] Angiographic restenosis occurs in 30-50% of cases by six months' follow up, with clinical (symptomatic) restenosis in most of these cases.[1] With no pharmacological treatment consistently reducing the restenosis rate in randomised studies, there has been an increased need for reintervention with further angioplasty or bypass surgery in 44% over the first three years of follow up, offsetting the initial savings in cost and morbidity.[3] Intracoronary stents, deployed by balloon dilatation at the site of coronary stenoses, were first developed as a mechanical means of preventing restenosis. They also allowed "bailout" from acute closure[4] (avoiding myocardial injury or emergency surgery) with preserved vessel patency at angiographic follow up.[4,5]

Intracoronary stents are deployed by balloon dilatation at the site of coronary stenoses. Balloon angioplasty works by a combination of stretching the vessel wall and rupturing the plaque. Depending on the complexity of the lesion, plaque rupture results in dissection of the wall to differing degrees in most cases. In the past, acute or threatened stenoses were treated either by dilatation with a perfusion balloon catheter or by emergency surgery, with high rates of myocardial injury and occasional deaths.

The use of intracoronary stents in both elective and emergency situations has reduced the rates of acute stenosis, emergency surgery, and myocardial infarction, as well as reducing the rate of restenosis by a third.[6,7] Early studies reported unacceptably high rates of stent thrombosis,[4,5] necessitating an aggressive antiplatelet or antithrombotic strategy.[8,9] Improved stent design and deployment has reduced the incidence of stent thrombosis, which occurred over the first 10 days after angioplasty in up to 18% of patients.[8,9] This occurred particularly where the stent was deployed in a bailout situation, although very rarely in vessels more than 3 mm in diameter.[9]

An aggressive antithrombotic strategy was associated with vascular and bleeding complications in up to 15% of cases.[8-10] Recent trials have suggested that formal anticoagulation is no longer necessary: the antiplatelet drug ticlopidine, along with aspirin, can be used to reduce the risk of subacute thrombosis.[11-13] Use of ticlopidine is restricted in some countries because of rare reports of reversible neutropenia. Thus, until recently, stenting required anticoagulation with intravenous heparin for up to 96 hours after deployment. One multicentre study of 246 patients who had undergone stenting confirmed the safety of using aspirin, ticlopidine, and 48 hours of intravenous heparin followed by low molecular weight heparin for one month.[12] The rate of stent thrombosis was as low as 1.2%, even though 12% were bailout stents. Vascular complications occurred in 11% of cases. Subsequent reports from this registry of 1183 patients receiving aspirin and ticlopidine alone have confirmed a rate of stent thrombosis of 1.2% with no vascular complications.

Initial observations suggested that in most patients the stent failed to expand completely after deployment.[14] As this may not be apparent at angiography, intravascular ultrasound has been used to guide adequate stent deployment with high pressure dilatation (more than 12 atmospheres) and larger balloon sizes to improve expansion and reduce the need for formal anticoagulation. In one study of 452 stented lesions, subacute thrombosis occurred in only 0.9% of lesions.[11] A comparison of aspirin alone against aspirin and ticlopidine in 226 patients undergoing stent implantation guided by ultrasound showed no difference in the rate of stent thrombosis (2.9% *v* 0.8%) or in major clinical events, confirming an advantage over studies also using warfarin.[13] The recent Benestent-II pilot study confirmed the safety of a regimen of aspirin and ticlopidine in a subgroup of 50 of 220 patients after stenting with heparin coated stents; there was no stent thrombosis, no major bleeding complications, and a restenosis rate of 6% in this subgroup.[15]

The first generation of heparin coated stents will become available this year, encouraging stenting without formal anticoagulation; with optimal deployment, a single figure restenosis rate can be expected, reducing the costs of reintervention. The exact indications for elective stenting remain relatively flexible given the