bovine internal mammary grafts, and prosthetic tube grafts. The results with all these are either dismal or unknown. One other option is the use of the free internal mammary artery. This method extends the range of the grafts and gives results with an enhanced long term patency similar to those with pedicled internal mammary artery grafts,¹³ but it imposes a technically awkward requirement for a proximal aortic anastomosis and still may not provide enough conduit for all of the vessels requiring grafting. Surgeons are looking for further possible second arterial conduits, and current interest has centred on the right gastroepiploic artery being left as a pedicled graft fed from the gastroduodenal artery.¹⁷⁻¹⁹ It should be possible to use double internal mammary artery grafts to the left anterior descending and circumflex systems supplemented by the right gastroepiploic artery to the inferior surface and so to achieve total arterial revascularisation.²⁰

What about those grafts that have failed? Balloon angioplasty or revisional surgery are possible options. Balloon angioplasty may be effective in opening up stenoses in internal mammary artery and saphenous vein grafts, although any instrumentation in old saphenous vein grafts carries a risk of distal emboli from atheromatous or thrombotic debris. The long term outcome after graft dilatation is unknown, but

reoperation is associated with 90% survival at five years and 75% at 10 years.²¹

In the early 1980s the concept of "complete revascularisation" was popular; it was based on the belief that grafts should be made to all vessels with stenoses tighter than 50%. We are now more realistic, taking account of the uncertain progression of underlying coronary artery disease. Given the likelihood that (for the near future, at least) saphenous vein grafts will continue in common usage, coronary surgery should be seen as a palliative rather than a curative procedure. While increased use of the internal mammary artery is indicated, particularly to the left anterior descending coronary artery, restriction of grafting to coronary arteries that have severe (>70%) stenoses and that will allow adequate (>40 ml/min) run off might represent the wise approach for the coming decade.

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Providing the fetus with calcium

Dietary supplements are needed for women in high risk groups

Necessarily, all of the calcium in the newborn infant has come from the mother. At birth the mineral content of the average mature baby is about 28 g of calcium and 16 g of phosphorus, with 99% and 85%, respectively, in the skeleton.^{1,2} The combination of progressive skeletal mineralisation and exponential growth means that some 70% of this calcium is deposited in the last trimester; the net flux of calcium into the fetus towards the end of pregnancy is about 250 mg a day.² The control of this process requires considerable integration and collaboration between the fetal and maternal endocrine systems, which meet at the interface of the placenta.

In women who are not pregnant circulating parathyroid hormone concentrations are controlled by the amount of ionised calcium in the serum; the hormone promotes gastrointestinal calcium influx principally by activating the renal enzyme 25-hydroxyvitamin D₁- α -hydroxylase. During pregnancy concentrations of parathyroid hormone measured by immunoradiometric assay (probably the most biologically

relevant method as it measures the intact molecule) are low.³ This strongly suggests that other factors—possibly lowered serum concentrations of phosphate—are responsible for the activation of the enzyme during pregnancy. Other possible candidates are prolactin; oestradiol; and placental lactogen, with its growth hormone-like effect. There is equally little agreement about maternal calcitonin, with studies reporting high, low, and normal concentrations.^{4,7}

1,25-Dihydroxyvitamin D plays a key part in calcium homeostasis. Vitamin D is produced in the skin by the action of ultraviolet light on 7-dehydrocholesterol, forming cholecalciferol (vitamin D₃), which in turn then undergoes 25-hydroxylation in the liver. The maternal concentration of vitamin D binding protein (which accounts for about 1% of the plasma proteins) is increased through the action of oestrogens on hepatic synthesis.⁸ Interpretation of total concentrations of the vitamin D metabolites is therefore complicated in pregnancy. The consensus seems to be,

however, that the concentrations of maternal free (biologically active) 1,25-dihydroxyvitamin D₃ are raised during pregnancy, increasing net calcium absorption^{8,9}; whether or not there is a concomitant reduction in the amount of calcium secreted in the gastrointestinal tract is uncertain.

Because of the limited access to the fetal circulation the endocrine influences on fetal calcium concentration are even more contentious. Probably quite early in pregnancy the placenta begins to pump calcium ions from the mother to the fetus against a concentration gradient. The ionised calcium concentration in the umbilical vein at term is around 1.10 mmol/l, compared with the maternal value of 0.90 mmol/l. What is the driving force for this calcium influx into the fetus? In sheep the fetal parathyroid glands seem to play a part as parathyroidectomy leads to fetal hypocalcaemia,¹⁰ but whereas the lamb maintains its high calcium concentration for several weeks after birth, the calcium concentration in the newborn baby falls rapidly in the first 48 hours by about 8-9%. This fall is greater in preterm or asphyxiated infants or those born to diabetic mothers.¹¹⁻¹³ Recent studies have raised the possibility that the newly discovered parathyroid hormone related peptide(s)—the cause of hypercalcaemia in patients with some malignancies—may be the placental calcium transport hormone.¹⁴ The suggestion is that in ruminants this peptide is formed in the parathyroid gland and in primates is either from another source—perhaps the placenta—or formed in the parathyroid glands, under the control of the placenta.

The practical implications of these research findings for clinicians are straightforward and important. The well nourished mother has a slightly positive calcium balance during pregnancy, and this is reversed if she breast feeds subsequently.¹⁵ Although the calcium requirement of the fetus is only about 2.5% of the maternal skeletal reserve, repeated pregnancies and prolonged lactation without adequate replenishment may lead to osteomalacia in the mother^{2,16} and neonatal rickets.¹⁷ These disorders are mostly confined to economically deprived communities¹⁷; those living at high latitudes¹⁸; and Asian immigrants in northern Europe,¹⁹ especially in winter.

Clinicians caring for pregnant mothers should, therefore, ensure that their patients receive a diet adequate in calcium

(1000-1200 mg a day) and vitamin D (400 IU a day), and they should prescribe supplements to women in the high risk groups. This is important not only in achieving the goal of a healthy mother and baby but also in ensuring the long term health of women—those with a low bone mass are more likely to develop early osteoporosis after the menopause.

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A revised consultant contract

Professional principles retained but more accountability

A remarkable feature of the National Health Service has been the wide measure of autonomy enjoyed by consultants in treating their patients and arranging their professional working practices. They have responded by showing exceptional dedication despite a chronic shortage of resources, the increasing intrusion of management, and repeated arbitrary interference with their pay awards. Indeed, doctors' contributions to the NHS have represented excellent value for what most countries would regard as comparatively modest resources, and the service has engendered widespread patient satisfaction. This has been achieved with remarkably low administrative costs, and the treasury has been able to contain the increase in costs to an extent which few if any other developed countries have been able to match. The aficionados of cost-benefit analysis can therefore be excused for wondering why the government has insisted on a reform of the consultant contract. The answer is that the existing arrangements do

not measure up to the government's policies to dismantle protectionism, promote competition, and strengthen management accountability. Furthermore, the politicians and the media have made much of those relatively few consultants whose work for the NHS has fallen short of their contractual commitments.

The government should not have been surprised that the survey carried out by the Office of Manpower Economics, which the review body took the unusual step of publishing as an appendix to its 1990 report, showed that consultants had been working on average 10 hours a week more than required under their contracts, not including on call commitments.¹ For their part consultants will not have been surprised that the government responded by refusing yet again to accept their review body's related recommendation for an increase in the top of the consultant incremental pay scale. The health service has not been alone in attracting unwelcome political