emergency operating is done has meant that much of it has been unsupervised.

This custom was established when the keynote of emergency surgery was to do the least necessary to ensure survival, but the past 20 years have seen growing recognition of the value of a major definitive operation performed at emergency surgery. This move was pioneered in the 1930s by such masters of surgery as Yudin and Finsterer, who successfully undertook emergency gastrectomy for bleeding and perforated peptic ulcers.^{3,4} In the 1950s primary resection of the obstructed and perforated colon was shown to be effective and safe^{5,6} and in the 1960s definitive treatment of the ruptured aortic aneurysm became the rule.

Contemporary senior registrars regard these operations as standard procedures. They have been trained by consultants who have come to recognise that (when carried out by experienced surgeons and anaesthetists) these definitive procedures offer patients a better prospect than the conventional lesser operations—for example, simple suture of perforated gastric ulcers and three stage treatment of neoplastic obstruction of the left colon.⁷ The corollary is that highly trained surgeons should be available to undertake emergency surgery at night. This may, however, be difficult to arrange, especially in the smaller district hospitals which employ only two consultant general surgeons, both of whom have a full daytime workload. (In this connection it seems strange that the argument, so strongly advanced, that junior staff should not be on duty more than one day in three does not seem to apply to consultants.)

Surgeons are a tough race and may consider loss of sleep to be a small price to pay for the sight of a patient making a good recovery from a serious emergency, but the interests of patients must be reconciled with the need for the surgical team to undertake reasonable hours of work.

So what may be done to ease the emergency load at night so that experienced surgeons have the time and energy to undertake such work in addition to their routine during the day? The arguments in favour of increasing the number of consultants (to improve among other things the care of emergencies) have been deployed for long enough.⁸ A better level of junior staff training and experience would come from adopting a more rational arrangement for the distribution of registrars from both home and overseas.⁹

Expansion of the consultant grade is still delayed despite the support of the Social Services Committee of the House of Commons² and of the royal colleges.^{10,11} To provide better emergency consultant cover it is unnecessary (and undesirable) to go to the lengths of introducing a shift system⁹ or expecting consultants to sleep in while on call.¹² These suggestions, which can only deter consultants from pressing for expansion, must be dropped. Furthermore, only 30% of patients admitted as surgical emergencies need urgent surgery, and, as Sherlock and his colleagues have shown, as many as one third of these urgent operations, currently performed at night, may safely be left until the next day (p 170). Staff should not have to work at night only because an operating theatre is not available for emergency work during the day.

One of the tasks to which the National Health Service, as opposed to private medicine, is particularly committed is the provision of a high level of emergency care, and all the necessary skills in clinical, laboratory, and radiological services are concentrated in district hospitals. Many emergency operations are relatively simple and play an important part in training junior surgeons, but recent developments in resuscitation, anaesthesia, intensive care, and in major

definitive operations for surgical emergencies require a higher level of skill. Provided that this is available gravely ill patients, especially the elderly, may make a quick and full recovery from clinical states that were previously fatal or the cause of long stays in hospital. It is not reasonable to expect willing but relatively inexperienced surgeons in training to undertake this type of surgery.

There are, therefore, compelling reasons for expanding the number of surgical consultants, not the least being the length of waiting lists and the very long hours of work required of many consultants. A major additional reason is that many skilled surgical registrars and senior registrars are ready to move into positions of greater responsibility. They are attuned to the recent developments in emergency surgery and could bring to out of hours emergency work the level of skill which it often requires.

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Metabolic consequences of bilateral nephrectomy

Bilateral nephrectomy is seldom performed routinely nowadays but it may be indicated for some patients with end stage renal failure who are having maintenance dialysis. Current dialysis techniques take over the excretory role of the kidneys and help in the control of salt and water balance but they cannot replace the renal endocrine functions entirely. Erythropoietin, for example, may be secreted by the kidneys even in advanced uraemia, and anephric patients often become severely anaemic and respond poorly to treatment with haematinics.¹ These patients may require frequent blood transfusions—with the consequent risk of iron overload.

The influence of bilateral nephrectomy on calcium and vitamin D metabolism has been more difficult to determine. The recent development of sensitive radioimmunoassays for vitamin D and its metabolites has shown that the kidney is of crucial importance in the conversion of vitamin D₃ to its active metabolites. Vitamin D₃ may be obtained from the diet or from photoconversion of 7-dehydrocholesterol in the skin and it undergoes several metabolic steps before its full biological potential is achieved.² Firstly, it is metabolised by a hepatic microsomal enzyme to 25(OH)D₃. This step is substrate limited, so that serum concentrations of 25(OH)D₃, broadly reflect the vitamin D in the diet. The

seasonal variation in exposure to sunlight, more definite in temperate climates, brings about a seasonal fluctuation in serum concentrations of $25(\text{OH})\text{D}_3$.^{3,4}

$25(\text{OH})\text{D}_3$ is considerably more active than vitamin D_3 itself in promoting absorption of calcium from the gut, but it is not the final active metabolite.² Further metabolism takes place in the kidney, where the dihydroxylated metabolites $1,25(\text{OH})_2\text{D}_3$ and $24,25(\text{OH})_2\text{D}_3$ are synthesised.^{3,5,6} $1,25(\text{OH})_2\text{D}_3$ is produced by an enzyme, $25(\text{OH})\text{D}_3$ -1-hydroxylase, in the renal proximal tubular cells; this enzyme is inhibited by hypercalcaemia, hyperphosphataemia, low serum concentrations of parathyroid hormone, vitamin D repletion, and by $1,25(\text{OH})_2\text{D}_3$ itself.^{2,8} Synthesis of $1,25(\text{OH})_2\text{D}_3$ is stimulated by hypocalcaemia, hypophosphataemia, high serum concentrations of parathyroid hormone, vitamin D depletion, and $24,25(\text{OH})_2\text{D}_3$.^{8,9}

$24,25(\text{OH})_2\text{D}_3$ is produced by a similar enzyme, $25(\text{OH})\text{D}_3$ -24-hydroxylase, again found in the renal proximal tubular cells¹⁰; this enzyme is inhibited by hypocalcaemia, hypophosphataemia, high serum concentrations of parathyroid hormone, vitamin D depletion, and $1,25(\text{OH})_2\text{D}_3$.^{8,11} Thus in normal people replete with vitamin D_3 , $24,25(\text{OH})_2\text{D}_3$ is the major circulating dihydroxylated metabolite of vitamin D_3 . At times of vitamin D deficiency production of $1,25(\text{OH})_2\text{D}_3$ is increased as an adaptive measure aimed at maintaining a normal serum calcium concentration by mobilising calcium from gut and bone.⁸

Against this background not surprisingly renal failure has potentially serious effects on the production of active metabolites of vitamin D. When the glomerular filtration rate falls below 25-30 ml a minute the serum concentration of $1,25(\text{OH})_2\text{D}_3$ often falls substantially owing to a combination of a reduced mass of functioning nephrons and inhibition of $25(\text{OH})\text{D}_3$ -1-hydroxylase by the hyperphosphataemia that commonly accompanies renal insufficiency. In end stage renal failure and in patients undergoing dialysis, serum concentrations of $25(\text{OH})\text{D}_3$ are variable¹² and of $1,25(\text{OH})_2\text{D}_3$ and $24,25(\text{OH})_2\text{D}_3$ uniformly low.^{13,14} Patients in renal failure of this severity often have histological evidence of a defect of mineralisation.¹⁵ Osteomalacia is not, however, invariable—and indeed is uncommon in patients having dialysis who are replete with vitamin D. Improvement of the mineralisation defect has been observed after the start of dialysis even in an anephric patient not treated with vitamin D.¹⁶ Although some investigators have found increases in the serum concentration of $25(\text{OH})\text{D}_3$ after the start of dialysis, there is no generally accepted explanation for this apparently spontaneous histological improvement and for the absence of osteomalacia in bone obtained from some anephric patients.⁴

Recently, however, several investigators have reported low but detectable concentrations of the dihydroxylated metabolites of vitamin D in sera from anephric patients with normal or raised serum concentrations of $25(\text{OH})\text{D}_3$. Initially these observations were confined to the presence of $24,25(\text{OH})_2\text{D}_3$,^{17,18} and, despite concern that other vitamin D metabolites (principally $25,26(\text{OH})_2\text{D}_3$ ¹⁹ and $25(\text{OH})\text{D}_3$ -26,23-lactone²⁰) might interfere with the sensitive assay techniques, the results have been interpreted as indicating an extrarenal site of synthesis of $24,25(\text{OH})_2\text{D}_3$. Further studies from different laboratories have not only confirmed these data²¹ but have reported detectable concentrations of $1,25(\text{OH})_2\text{D}_3$ in sera from some anephric patients.²²⁻²⁴

The concentrations of the dihydroxylated metabolites detected are well below normal values and lie at the lower limits of the currently available assays. Moreover, most

reported data concern measurement of total $1,25(\text{OH})_2\text{D}_3$ and not specifically the biologically active metabolite $1,25(\text{OH})_2\text{D}_3$. Nevertheless, though obtained from only a few patients, the results may indicate an extrarenal site of production for both $24,25(\text{OH})_2\text{D}_3$ and $1,25(\text{OH})_2\text{D}_3$. The origin of these metabolites is unknown, but both may be synthesised from $25(\text{OH})\text{D}_3$ by bone cells in culture.²⁵ It remains only to be determined whether such small quantities of active vitamin D metabolites help to prevent osteomalacia in some anephric patients.

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Delayed psychological effects of perinatal deaths: the next pregnancy and the next generation

Until about 15 years ago the psychological sequelae of stillbirth or neonatal death attracted few publications and were sadly neglected in clinical practice.¹ Recently, however, awareness of the psychological problems in the families concerned has increased.^{2,4}

The mother of a stillborn baby feels bewildered and sad, and such feelings may develop into chronic depression,