

long journey as a means of achieving "complete rest," particularly when the diagnosis is uncertain or when the patient has recently started on drug treatment. The acute stress of the journey may cause a relapse, and occasionally acute psychotic states may result from treatment with psychotropic drugs.¹

The risks of travel can be reduced by careful planning of journeys, which takes account of time zone changes and ensures adequate rest periods, preferably away from the seat or compartment used during the trip. The intake of food and fluid must be adequate, and that of alcohol or drugs should be maintained at or near their usual levels. The effects of isolation can be reduced by social contact with travelling companions and by purposeful activity during the journey. Prevention lies mainly in reducing the stresses of the journey rather than in identifying individual vulnerability, but the motivation for the journey is important—few of us will break down as we speed to our holiday island.

Vascular Aspects of Cholecystitis

The aetiology of cholecystitis is complex. While the condition sometimes develops in a previously normal organ as a result of bacterial infection, in the great majority of cases it is superimposed on a gall bladder harbouring calculi.¹ Experiments have shown that concentrated bile has a destructive action on the gall bladder mucosa, especially if the circulation is impaired,² and it is probable that acute cholecystitis usually starts as a chemical inflammation. In one series bacterial cultures were found to be positive in only 35% of cases during the first 24 hours, but the incidence was 65–80% by the third day.³

The precipitating factor is often a stone impacted in the cystic duct. As the mucous glands continue to secrete, the gall bladder becomes distended, while the entrapped bile damages the mucosa. The inflammatory oedema and the general distension gravely impair the blood supply of the wall, and, if the process continues, patchy infarction occurs, especially of the fundus, where the blood supply is poorest.⁴ The tension on the devitalized wall leads to its rupture. It has frequently been noted that in necrotizing ("gangrenous") cholecystitis the main histological features are vascular obstruction and infarction.^{1 2 4 5} Inflammatory change becomes marked only in the later stages, when bacterial infection is superadded.

While the importance of vascular damage in the pathogenesis of acute cholecystitis is apparent, it is less certain what effect it has in causing the condition. K. C. D. Gordon⁶ has studied the gross arterial architecture of the gall bladder by injecting its arteries with an opaque solution, filling the lumen of the organ with a translucent resin, and finally ren-

dering the wall transparent with a clearing agent. This technique shows the arteries clearly against the translucent gall bladder. Gordon has made comparative studies on many animals, and has noted species differences in the vascular arrangement. Gall bladder disease is rare in the lower animals, yet the differences in vascular architecture between some of these and man are insufficient to account for the high incidence of disease in the human gall bladder.⁶

Gordon has extended this work by investigating the vascular architecture of diseased human gall bladders.⁷ As the technique does not allow later histological examination, the diagnosis of chronic cholecystitis was made on macroscopic grounds. He found that in chronic cholecystitis the vascular pattern was grossly distorted and that the vascularity was patchy and greatly reduced. Even in apparently early cases these changes were well marked. By contrast, mucocoele and cholelithiasis without cholecystitis were associated with essentially normal vascular patterns.

That chronic cholecystitis is associated with vascular occlusion is not surprising, since endarteritis obliterans is a prominent feature of chronic inflammation. It is exceedingly likely that these vascular changes predispose towards repeated attacks of acute cholecystitis. The normal pattern in mucocoele and uncomplicated cholelithiasis shows that mechanical obstruction of the cystic duct and the presence of stones do not necessarily lead to vascular occlusion. Moreover, these conditions are not related to vascular abnormalities, though both predispose to cholecystitis. It is therefore doubtful whether vascular lesions have a significant role in the aetiology of acute cholecystitis, though they are probably crucial in leading to recurrent attacks in a diseased organ.

Nurses' Pay

The general public looks kindly on nurses, and there is not likely to be an outcry about the recent pay award by the Prices and Incomes Board.¹ It gave roughly a 14% salary rise, 4% backdated to October 1967 being payable immediately. This is not the only result of the Board's investigations, for it has coupled its findings with approval of the Salmon Committee's suggestions on management-structure in nursing, with which the Ministry of Health is only just beginning to experiment, and recommended a change in the age of entry, which is the province of the General Nursing Council.

The report is written with great clarity in basic English, and begins with an excellent description of the constitution of the nursing service. Chapter 4 describes the training of nurses, and notices that at present there is no shortage of recruits. But in anticipation that there may be a fall in the number up to 1975 the Board proposes that the entry age should be lowered to 17. The professional organizations have often reviewed the entry age, and found reasons for not changing, and since later the report notices that students are sometimes left alone in a ward at night the suggestion is a controversial one.

The Board was especially asked to consider the position of tutors, since the number of registered tutors in post has not

¹ Hallendorf, L. C., Dockerty, M. B., and Waugh, J. M., *Surg. Clin. N. Amer.*, 1948, 28, 979.

² Thomas, C. G., and Womack, N. A., *Arch. Surg.*, 1952, 64, 590.

³ Goldman, L., Morgan, J. A., and Kay, J., *Gastroenterology*, 1948, 11, 318.

⁴ Strohl, E. L., Diffenbaugh, W. G., Baker, J. H., and Cheema, M. H., *Int. Abstr. Surg.*, 1962, 114, 1.

⁵ Eliason, E. L., and Stevens, L. W., *Surg. Gynec. Obstet.*, 1944, 78, 98.

⁶ Gordon, K. C. D., *J. Anat. (Lond.)*, 1967, 101, 351.

⁷ Gordon, K. C. D., *Gut*, 1967, 8, 565.

¹ National Board for Prices and Incomes, Report No. 60, *Pay of Nurses and Midwives in the National Health Service*, 1968. H.M.S.O. (8s. 6d. net).