Clinic staff who have influenced its work substantially and who have contributed to its reputation include John Bowlby, who, with James and Joyce Robertson, studied patterns of maternal care and the effects on young children of temporary separation from their mothers; Henry Dicks, who made important advances in marital disorders; Michael Balint, who introduced new ideas into general practice—his particular approach to training seminars has been carried all over Europe; Wilfred Bion, who made a massive conceptual contribution to the theory of group dynamics and was one of the pioneers of group therapy; and David Malan, whose studies have had a major impact on the practice of brief psychotherapy throughout the world.

The Cassel Hospital in Richmond, Surrey, is predominantly an inpatient hospital but since it came into the NHS its staff have become increasingly concerned in outpatient, community, and liaison hospital work. T A Ross, who was medical director from its establishment until 1934, pioneered brief psychotherapy for the neuroses. After the last war T F Main introduced more direct psychoanalytic psychotherapy, developing a nurse-training programme that teaches psychosocial techniques embodying psychoanalytic findings, and creating a hospital structure to support and reinforce formal psychotherapy as one form of the "therapeutic community." Training for medical and other therapists and for nurses has gradually become more extensive and detailed, while at present efforts are being made to give the training programmes formal institutional shape and independent finance. The Cassel Hospital's experiments in structuring a therapeutic community have become widely known and copied in Britain and in other countries. Training now extends beyond the staff of the hospital in the form of seminars for various professional groups and as courses and placements for numerous visitors.

Before the war the hospital treated a wide range of neurotic and psychotic disturbance. Since the war it has increasingly treated more severely neurotic and borderline patients. It pioneered the inpatient treatment of puerperal mental illness and out of this grew the admission and treatment of whole families. Now there are units for "families and adolescents" and for "single adults." The presence of children and adolescents has led to the provision of a school as well as facilities for infant care.

Earlier concepts of treatment—individual psychotherapy carried out by doctors (usually psychoanalytically trained or in training), supported by a practical environment organised by nurses—have given way to concepts of combined individual, group, and community treatment, in which the hospital routine and structure integrate these different treatment aspects into a coherent whole. Evidence is accumulating that such a structure is a powerful therapeutic instrument which can effect substantial changes in severely disabled personalities relatively quickly.

A primary aim in treatment is to give patients responsibility for themselves and their illnesses. To the extent that the hospital has been able to do this may provide an encouraging model for other small specialised institutions faced with similar problems.

Though the work of the Tavistock Clinic and the Cassel Hospital is different, they share a common ideology and their endeavours complement each other. Over the past 60 years their efforts, while remaining rooted in the discoveries of the consulting room, have become increasingly concerned with the patient in his human environment. Psychiatry stands in their debt for their unswerving devotion to the psychosocial aspects of mental health and mental illness.

Evolution of colonic polyps

Both long-standing familial polyposis and chronic ulcerative colitis predispose to cancer of the large bowel, but most cases arise from one of the many different types of polyps found in the colon and rectum—the polyp-cancer sequence.[1] Surgeons need to be able to differentiate these varying types of polyps and to recognise and deal with those that are likely to be precancerous.

Juvenile polyps occur mainly in children and there is no evidence to suggest that they become malignant. Metaplastic or hyperplastic polyps are common, especially in the rectum, occurring with increasing frequency with age, being found in 95% of rectums removed for carcinoma; but, again, they are thought not to be precancerous. The colonic polyps found in patients with Peutz-Jeghers syndrome have little or no disposition to develop into carcinomas. All these polyps are either inflammatory, hamartomatous, or allergic, and are not truly neoplastic.

Adenomas of the colon are (after the metaplastic polyps of the rectum) the most common type of polyp encountered and are classified as tubular, villous, or tubulovillous. The tubular type is far more common (75%) than the typical villous tumour.[2] In gross appearance these polyps may be smooth and lobulated, shaggy, sessile, or pedunculated. Most villous adenomas occur in the rectum, and 85% cause symptoms—bleeding, passage of mucus, and watery diarrhoea—and often intussusception. Some villous tumours excrete large amounts of potassium and sodium chloride in the mucous discharge, leading to hypokalaemia, hypochloraemia, and peripheral circulatory failure.[3] The incidence of invasive carcinoma is said to vary between 20% and 40% in the villous adenomas, especially in the larger tumours, but in a recent unpublished series from the General Hospital, Birmingham, the incidence was as low as 11%. Treatment of villous adenomas should be by local excision with later treatment planned after conventional histological examination—not by frozen section.

About one-third of all colons resected for carcinoma will have one or more adenomas in addition. These adenomas tend to be found more frequently near the carcinoma, and patients with adenomas are at an increased risk of developing more than one carcinoma—developing at different times.[4] Patients found to have an adenoma with a focus of carcinoma are at a high risk, perhaps as high as 20%, of having an independent cancer either synchronously or later.[5] Most adenomas containing focal carcinoma are seen in older patients, and most in the sigmoid colon and rectum. The risk of cancer varies with the size of the polyp, its histological type, and degree of dysplasia. The risk of cancer is low for tumours with diameter of 1 cm but rises to 50% when this is over 2 cm.[2]

Whenever adenomatous polyps can be seen through the sigmoidoscope they should be removed by diathermy excision. Use of the flexible colonoscope permits removal of polyps above the reach of the sigmoidoscope without subjecting the patient to laparotomy.[6] Tumours under 5 mm in size, high in the colon, may be left with reasonable safety provided the patient is carefully followed up.

A recent report from New York[7] presented experience with over 7000 endoscopically removed polyps from the colon and rectum. After analysis of the results with 5786 neoplastic or adenomatous polyps—the authors advocate the routine endoscopic excision of all colorectal polyps. The presence of over 100 adenomas in the colon is considered to be a case of multiple polyposis; this condition is inherited as a Mendelian

dominant and should be treated by total colectomy and ileorectal anastomosis. Regular follow-up examinations of the rectum are needed, with any polyps being removed by fulguration.

While not all adenomatous polyps become malignant every opportunity should be taken to remove them, especially those over 2 cm in diameter. Because they are larger, and epithelial dysplasia is more common, villous adenomas have considerably greater malignant potential than tubular ones. Almost certainly most carcinomas of the colon and rectum develop from pre-existing adenomatous polyps. On average the polyp-cancer sequence takes 10 to 15 years, but the interval may be as short as five or as long as 25 years.[2]

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Chronic active hepatitis

Chronic active hepatitis is defined as inflammation of the liver continuing without improvement for at least six months, with the histological findings of round-cell infiltration of the portal tracts and moderate or severe piecemeal necrosis of liver cells extending outwards into the parenchyma.[1] Understanding of the pathogenesis and natural history of this condition has come a long way since it was first recognised in the early 1950s. Subgroups have been recognised with different mechanisms of damage to the liver, and these have important implications for treatment.

The two main causes of chronic active hepatitis, chronic infection with the hepatitis B virus and autoimmune disease, differ in their geographical distribution, sex ratio, serum markers, prognosis, and treatment. Hepatitis B virus infection is an uncommon cause in Britain but the incidence may be increasing, particularly among male homosexuals.[2] It is, however, the most common cause of chronic active hepatitis in the Middle East and Asia, where the chronic carrier state is more prevalent than in Europe—men are affected more often than women. The hepatitis B virus is usually detected by a finding of HBsAg in the serum, but in some cases the amount of viral antigen in the serum may be below the limits of detection by current methods. In these circumstances viral antigen can be identified in the liver cells by immunological methods, and there are usually high serum titres of antibodies to the core antigen (anti-HBc).[3]

In contrast, the autoimmune type of active hepatitis is more frequent in women and often affects other systems, being associated with the sicca syndrome, arthralgia, thyroid disease, haemolytic anaemia, inflammatory bowel disease, and renal tubular acidosis.[4] Smooth muscle and antinuclear antibodies are typically found in the serum, and there is usually a greater degree of hypergammaglobulinaemia than in patients with chronic viral infection. These immunological abnormalities and the mechanisms of hepatic damage have been reviewed recently.[5-7]

The prognosis of patients with chronic active hepatitis carrying HBsAg is uncertain: claims have been made for both a better[8] and a worse[9] outlook in the long term than in patients with the autoimmune type. What is clear is that the disease often progresses slowly to cirrhosis.[9] The treatment of the autoimmune type has been established in three prospective controlled trials,[10-12] each of which showed that treatment with corticosteroids (with or without azathioprine) considerably reduced the mortality in the early active phase of the disease; the benefits of treatment have recently been confirmed by the long-term follow-up of patients in the Royal Free Hospital trial.[13] On the other hand, in patients with viral disease the use of corticosteroids may not influence the outcome; indeed immunosuppression may allow more viral replication, thus increasing the patients' infectivity. The results of formal controlled trials are, however, still awaited. A more logical approach might be to try to eliminate the virus either by boosting the body's immune mechanisms or by using an effective antiviral agent; at present interferon is showing the greatest promise.

Recent reports have highlighted the difficulty of differentiating chronic active hepatitis from Wilson's disease[14]—a vitally important distinction since the conditions are susceptible to different specific and effective treatments. The patients with Wilson's disease may lack typical features such as Kayser-Fleischer rings and neurological changes, and serum concentrations of both caeruloplasmin and copper may be normal, especially if there is severe hepatocellular necrosis. In such patients the diagnosis of Wilson's disease may be delayed, and once cirrhosis is established the prognosis may be poor despite treatment with D-penicillamine.

Drugs may be responsible for the full range of both acute and chronic liver damage.[15] Chronic active hepatitis has been described after treatment with the laxative oxyphenisatin, with methyldopa, and with isoniazid.[15] Occasional reports have incriminated repeated exposure to halothane[16] and treatment with dantrolene.[17] Recently several cases of chronic active hepatitis have been attributed to the long-term use of nitrofurantoin.[18,19] Recognition of the offending agent is all important, for withdrawal usually halts progress of the disease. Drugs are probably not an important cause of chronic liver disease in Britain (where oxyphenisatin is not available) but physicians should be alert to the possibility.

Alcoholism may be accompanied by liver disease with the histological picture of chronic active hepatitis,[20] when the histological changes considered characteristic of alcoholic liver disease (fatty change, alcoholic hyaline, and polymorphonuclear infiltration) are either minimal or completely absent. Possibly alcohol damages the liver in these cases by initiating an abnormal immune response.

Some patients with liver disease associated with deficiency of alpha-1-antitrypsin show features of chronic active hepatitis on liver biopsy.[21] The hallmark of this disease is the presence in the liver cells of globules of alpha-1-antitrypsin, but these may be missed unless the appropriate stains are used. Primary biliary cirrhosis is another condition that may sometimes be distinguished with difficulty from chronic active hepatitis because of the overlap of clinical, biochemical, serological, and morphological features between the two diseases. The patients with chronic active hepatitis, however, can usually be picked out by their good response to corticosteroids.[22]

We are left with a nucleus of patients with chronic active