gall bladder, it has been for mucous fistula depending on stricture of the cystic duct following on gall stones, and all the cases were completely and permanently relieved.

Cholecystectomy can seldom be advisable or necessary as a primary operation in gall stones, and extremely rarely possible in malignant disease. In cholecystotomy, where it is impossible to bring the margins of the incised gall bladder into the wound, and where the parietal peritoneum cannot be tucked down to meet the edges of the opening, I have made a tube of the omentum, but in such cases no hesitation need be felt in trusting to a drainage tube, as the peritoneal cavity soon becomes occluded around the drain, and there is little or no tendency to the passage of bile among the viscera, so that a suprapubic drainage opening is quite unnecessary. With very few exceptions I have found a vertical incision along the upper part of the right linea semilunaris to give ample room, but if required I have not hesitated to get further room by a transverse cut in addition.

Suture of peritoneum, aponeurosis, and skin by separate stitches effectually guards against ventral hernia, if the patient be kept recumbent for from 21 to 28 days, and if a firm oval pad be worn under a belt for a few months subsequently.

In all cases strict antiseptic precautions have been observed, and the abdomen has been left as clean and dry as possible.

In conclusion, I would emphasise that with due skill and adequate care operations on the gall bladder and bile ducts are among the most successful of the major operations, but as many of them are extremely difficult, and as it is impossible to say beforehand whether any case may not prove so, I think such surgical work should be undertaken only by those who have had experience in abdominal surgery, and who have witnessed or helped in several operations of this kind. As soon as this is the case we shall cease to witness the varying rates of mortality in the hands of different operators, of from 50 to almost 0 per cent., and shall probably find that, excluding cases of malignant disease associated with jaundice, the all-round mortality will not exceed 5 per cent. I hope the time is not far distant when it will be fully recognised that though cholelithiasis, so far as its causes and its early treatment are concerned, is distinctly a condition for medical treatment, it is both unjust to the patient and unfair to the profession to continue medical treatment until serious complications supervene, or the patient is almost, if not quite, past relief, before the aid of surgery is invoked.

PERNICIOUS ANÆMIA AT 21.

By W. R. GOWERS, M.D., F.R.S.,

Consulting Physician to University College Hospital; Physician to the Hospital for the Paralysed and Epileptic.

PERNICIOUS anæmia is not often met with during the first 25 years of life, and the malady is still so mysterious that any unusual case deserves record, even if it is possible only to give a brief note of it.

A young man, aged 21, was brought to me on February 19th, who presented precisely the aspect which is seen in extreme chlorosis. The skin had the characteristic tint, and the gums and conjunctivæ were very pale. He was feeble, and readily rendered short of breath. The condition was said to have come on gradually during the previous six months. I have since learnt from Dr. Nicholls, of Croydon, that he had seemed well before that time, and in good health, although delicate as a young child.

When I saw him, examination of the blood showed that the hæmoglobin was only a little over 30 per cent., and the percentage of red blood corpuscles was scarcely 25. This of course at once showed the nature of the case. Had it been the common "chlorosis," such as is frequent in girls, with that percentage of hæmoglobin the red blood corpuscles would have been at least 85 or 90 per cent. (It is not unusual in pernicious anæmia for the hæmoglobin to be a little in excess of the corpuscles; each corpuscle has even more than the normal amount.) An examination of the eyes showed numerous flame-shaped hæmorrhages in each retina, and one or two, of more irregular shape, close to the disc. I subse-

quently learnt from Dr. Nicholls that hæmorrhages had been found a month before, by Mr. C. Wray, of Croydon.

A very grave prognosis was obvious, and the course of the malady corresponded. A fatal ending is almost inevitable with such a reduction in the number of the corpuscles. In spite of careful treatment he steadily failed. There was some elevation of temperature for a few days, and pyrexia returned on March 19th, when a large hæmorrhage occurred in the right eye. Vomiting set in, and the patient died on March 21st.

The course of the disease was rapid, but the significance of the later rapid failure may easily be misconceived. The blood-making tissues depend for their nutrition and functional power on the blood which they have formed; when this has sunk to a low percentage, its effect upon them must be to increase, with augmenting rapidity, their incapacity.

Such a case lends itself to the current tendency to associate all sorts of diseases with specific organisms. But another hypothesis deserves consideration. The failure of tissues soon after they complete their development is not unknown in other structures. We see it, for instance, conspicuously in the so-called "hereditary ataxy" and in some late cases of pseudo-hypertrophic paralysis and allied maladies. We must ascribe these to an inherent defect of vital endurance on the part of the tissues concerned. It may be that a similar defect of vitality or of vital endurance in the blood-making tissues is the cause of such cases of pernicious anæmia.

THE ATROPHIC PHENOMENA OF RHEUMATOID ARTHRITIS.¹

By JOHN KENT SPENDER, M.D. LOND.,

Lately Physician to the Royal Mineral Water Hospital, Bath.

IN the first volume of the *International Clinics*, second series, is a plate professing to represent the advanced and final stage of shaking palsy. We are told that in this disease there is no gross lesion, but obscure functional disorder; and that the stability of the nervous system is upset by molecular disturbances in the cortex of the brain. Whether this be so or not we must be struck with the resemblance of the figure on this plate to the last stage of rheumatoid arthritis—that stage, I mean, which implies final wreck and ruin, when joints are disorganised and muscles are wasted beyond all help and hope.

The study of atrophic paralysis has received a new impulse from the exhaustive paper read by Dr. Ferrier before the annual meeting of the British Medical Association at Newcastle-on-Tyne. We welcome and appreciate his lucid grouping of the atrophic palsies into (a) those dependent upon primary lesion of the spinal cord (a myelopathy); (b) those dependent upon lesion of the nerves (a neuropathy); and (c) those dependent upon lesion of the muscles themselves (a myopathy). It is not imagined that these forms of paralysis can always be diagrammatically separated and diagnosed. The order and development of the symptoms belonging to one type of paralysis may merge into those belonging to another; or one type may slide into another, so that the original landmarks become confused and obscured.

I shall try to prove one proposition: that the phenomena of muscular atrophy in rheumatoid arthritis are myelopathic, and capable of definition as a strict spinal paralysis. The argument for this proposition rests upon the fact that the purely rheumatoid phase of the atrophy now and then extends upwards, and that what was originally a myelopathy of spinal nuclei becomes a myelopathy of bulbar nuclei. In plain words, a person suffering from rheumatoid arthritis with much atrophy gradually finds that she cannot masticate or swallow well, nor protrude the tongue; and the expression of her face becomes stolid and almost blank. And we infer the pathology of the former condition from the known pathology of the latter. The atrophy of rheumatoid arthritis is not to be considered as a sequel of the arthritis, but as a concurrent fact. Both are sequences of a common antecedent

¹ Read before the Bath and Bristol Branch of the British Medical Association, on November 29th, 1893.