At thirty-five of the autopsies blood-agar cultures from the hepatized lung were made. Colonies of influenza bacilli were obtained in 15 cases (43 per cent.), staphylococci in 19 cases (54 per cent.), streptococci in 19 cases (54 per cent.), pneumococci in 2 cases (6 per cent.), *M. tetragenus* in 1 case (3 per cent.), and Gram-positive bacilli in 1 case. Influenza bacilli and staphylococci were obtained in 3 cases, influenza bacilli and streptococci in 5 cases, influenza bacilli, staphylococci, and streptococci in 2 cases, influenza bacilli, staphylococci, and pneumococci in 1 case, staphylococci and streptococci in 8 cases, streptococci and *M. tetragenus* in 1 case. In one case no growth was obtained.

The blood-agar medium was made by adding to peptone meat extract agar (previously melted at 100° C. and then cooled to 50° C. to 45° C.) 10 per cent. of a solution of blood (warmed to 40° C.). The latter solution was prepared by mixing 5 c.cm. of human blood, withdrawn from a vein, with 95 c.cm. of sterile 0.85 per cent. sodium chloride solution, and then laking by the addition of 10 c.cm. of ether. Colonies of influenza bacilli were isolated by plating out.

In several instances influenza bacilli could not be obtained in culture though present in the lung exudate; the reverse was also observed. In three cases influenza bacilli were found in the sputum, but subsequently at the autopsy could not be recognized in the lung exudate on microscopical examination of a smear preparation; in one of these cases however, colonies of influenza bacilli were obtained on culture from the lung exudate.

If all observations are grouped together (sputum, smear from lung exudate, culture from lung exudate) the following figures are obtained: Influenza bacilli were found in 56 per cent. of the cases; staphylococci in 51 per cent.; streptococci in 49 per cent.; pneumococci in 18.5 per cent.; *Diplococcus catarrhalis* in 7 per cent.

The cases first occurring were of a severe type rapidly terminating fatally; subsequent cases were of longer duration, and were frequently complicated with pleural effusion and empyema.

CHRONIC TRENCH FEVER.

BY

GORDON WARD, M.D.

DURING the last few years many cases of trench fever have passed through the hands of medical officers, and the disease is likely to trouble us and our patients for some years to come. For this reason I am prompted to send a note of two cases which exemplify somewhat unusual symptoms, although such as the nature of the disease would lead us to expect.

CASE I.

Pte. C. W. B., Royal Warwicks, was admitted to hospital as a N.Y.D. case. He complained of extreme pain in the head and legs, and of sleeplessness due to the pain. He presented the following signs, namely, frequent sweating, irregular temperature, never above 100° at night, and showing no definite periodicity, occasional vomiting, and more frequent nausea; pulse rate varying from 76 to 116, and the typical hyperalgesic

areas (C8 and D1; D 7, 8, 9; all lumbar) described by Carmalt-Jones. A diagnosis of trench fever seemed fully justified. It was thought that cases of trench fever with small evening pyrexia might show a much larger swing than normal below the normal line. A few cases and a few controls from other

cases were therefore put on special charts. This case was the best example obtained of the phenomenon expected. Chart herewith shows this. It was not found to anything approaching the same extent in any case not trench fever. Possibly this may be of some assistance in diagnosis.

It was very difficult to obtain accurate records of pulse rate in all cases, but it may be stated that this followed the temperature pretty accurately except that the prevailing rate was lower after he had been in bed a week. Case II is an example of vasomotor symptoms. These have not attracted much notice but are mentioned in the American report. Analogy with malaria led me to look for them, and some degree of "dead fingers" and "dead feet" was noted in several cases. Cases were not included unless the "dead areas" were obvious to sight and touch. Other symptoms of the same order resembled giant urticaria and erythromelalgia.

CASE II.

Rfn. A. E. B., K.R.R.C., was admitted with typical acute trench fever. In the first seventeen days he had four bouts of pyrexia. After this the temperature became irregular. On the twenty-sixth day he had acute pain in the legs, which were observed to be very red (the feet only). This was much increased when the legs were allowed to hang over the edge of the bed. This erythromelalgic syndrome was not observed again. On the twenty-ninth and thirtieth days he had slight haematuria, the urine contained a few granular blood casts. On the thirty-second day the urine was free from albumin and casts and remained so. At the same time as the haematuria there was some oedema of the calves of the legs, there was none of the feet. The oedema was very slight. There was no other oedema. His temperature was 101° on the two days of haematuria, afterwards it did not exceed 99°. His pulse was usually between 60 and 80; it was not recorded in the acute stage of his illness except on admission, when it was 116. He was up on the fortieth day, and sent to England on the forty-second.

Case II did not suffer from dead fingers, and the erythromelalgic symptoms were only present on one occasion. I consider that the haematuria was not significant of a kidney lesion but rather analogous to the occasional haematuria of malaria. It may have been haemoglobinuria, and this seems probable on theoretical grounds. It was not reported until the thirty-ninth day, and the next specimen obtained did not contain blood cells but only casts containing yellow pigment. The quick disappearance of urinary signs is also significant. I have seen oedema of the calves also in malaria in the absence of albuminuria. This note does not profess to be complete; it was very difficult to carry out detailed investigations in France, but I hope that, in spite of this, the mere mention of such symptoms may be of use to others and of interest from the theoretical side.

April 20th, 1919.

Kettsomian Lectures

ON

JAUNDICE: WITH SPECIAL REFERENCE TO TYPES OCCURRING DURING THE WAR.*

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LECTURE III.—PART II.

SPIROCHAETAL AND ACHOLURIC JAUNDICE, Etc.

INFECTIVE JAUNDICE IN SPIROCHAETOSIS ICTERO-HAEMORRHAGICA.

THIS form of jaundice was first differentiated by two Japanese workers, Inada and Ido,¹⁰ in November, 1914, who made a brilliant investigation into the etiology, symptomatology, and pathology of the disease.

In September, 1916, Captain A. Stokes, R.A.M.C., and Captain J. A. Ryle, R.A.M.C., published¹¹ an account of several cases they had carefully investigated amongst our troops in Flanders, and later, in January, 1917,¹² they published a further account of this work. Major-General Sir Bertrand Dawson, Lieut.-Colonel Hume, R.A.M.C., and Captain S. P. Bedson, working independently, published¹³ a very complete account of their observations on cases

* Delivered before the Medical Society of London, April-May, 1919.

occurring in the army in France, and in November, 1918,¹⁴ at the Royal Society of Medicine, London, Sir Bertrand Dawson gave a further more detailed account of this disease.

During the past three years this form of jaundice has been closely studied in various parts of the world and most careful investigations have been made. Thus in the French army the disease was found by Martin and Pettit, who published¹⁵ an account of it in October, 1916, and have made several further contributions. Investigations on similar lines have been made in the Belgian and Italian armies.

The occurrence of the disease in rats has also been closely studied on the Continent, in Japan, America, and elsewhere, and in March, 1918, Dr. A. E. Coles¹⁶ found on examination of rats at Bournemouth that 9 per cent. were infected with the specific spirochaete.

Accounts so full and careful have been published recently of this disease that it will be unnecessary to give a detailed description.

The Clinical Manifestations.

An excellent account of these was given by Sir Bertrand Dawson,¹⁴ which I cannot do better than quote:

The disease has more often a sudden than a gradual onset, and manifests itself by shivering, head and body pains, great prostration, vomiting, and diarrhoea. The temperature rises quickly to 102° or higher. During the succeeding three or four days the conjunctivae become injected, and herpes, which is liable to become haemorrhagic, appears on the lips in about 40 per cent. of the patients. Bleeding occurs in most of the severe cases, but uncommonly in the mild cases. It may come from the nose, lungs, stomach, bowel, or as a purpuric rash. Early in the illness a slight haemoptysis is a valuable diagnostic sign. It is to be noted that haemorrhage often precedes jaundice in order of appearance.

The jaundice usually appears on the fourth or fifth day, but may be as early as the second and as late as the seventh day. It reaches its height about the tenth to twelfth day. In some cases it is intense, and the skin takes the greenish hue met with in complete obstruction of the common bile duct. Constipation is marked. The stools may become clay-coloured, though in most cases a small quantity of bile gives them a light brown coloration. Tenderness in the upper abdomen is usual. The tongue is furred and brown. The liver is frequently enlarged to the extent of two or three fingerbreadths below the costal margin. The spleen is not palpable. The glands in the axillae and groins are sometimes enlarged and shotty.

In severe cases there are evidences of an acute bronchitis. The respiration rate may rise to 28 or 30, and when a fatal result is impending may resemble that met with in uraemia. The pulse is slow in proportion to the pyrexia, a rate of 75 to 85 being quite usual.

In this respect the disease resembles enteric fever, though differing from the latter in that the heart does not lock with atropine. The systolic blood pressure is about 120 mm., being higher than that of enteric fever.

The early weakness and prostration are characteristic of the disease. Frontal headache and aching behind the eyeballs are distressing symptoms of the commencement, but diminish as the days pass. The muscular pains last longer and are at times intense. Twitchings and convulsions may precede or accompany the coma of the fatal cases.

The urine contains bile in abundance, which may persist for four to five weeks. Albumin is usually present, and may reach a sixth; casts—hyaline, epithelial, and granular—are common. Some French authors lay stress on the evidence of renal insufficiency.

The course of the illness varies with the severity of the disease. In the acute case an irregular type of pyrexia persists for ten to fourteen days and ends by lysis. In some instances there is a secondary rise of fever about the beginning of the third week which is difficult to explain, since there is no accompanying exacerbation of symptoms or increase of jaundice. The jaundice will reach its height about the tenth day, and this often coincides with the fall of the temperature. In other cases the temperature will fall earlier while the jaundice is still deepening. Convalescence is slow, but recovery is complete.

It is interesting to note that spirochaetosis icterohaemorrhagica may occur without jaundice. Stokes¹² stated that in 100 cases examined by him jaundice occurred in only 60 per cent.

Morbid Anatomy.

In three cases described by Sir Bertrand Dawson, marked evidence of duodenitis and intense inflammation in the area round the ampulla of Vater were found. In one of his cases associated with this duodenitis was a plug of mucus impacted in the ampulla. He says that it would seem clear that in some cases the spirochaetal infection localizes in the duodenum. Stokes did not mention the occurrence of duodenitis in his fatal cases. Both observers describe the bile ducts as being free from inflammatory

changes to the naked eye. Usually the liver showed few abnormal changes macroscopically, but on microscopical examination evidence of cellular degenerative changes was found in some instances. In one case described by Dawson the liver was diminished in size, its capsule wrinkled, and microscopically there were numerous areas of cell necrosis—in other words, a condition of acute yellow atrophy. Hart quotes¹⁷ a case in which extensive replacement fibrosis had supervened on acute yellow atrophy, just as in Case 1 of my tetrachlorethane cases.

The condition of acute yellow atrophy in fatal cases of spirochaetosis icterohaemorrhagica is apparently uncommon; death usually results from the toxæmia of the disease rather than from auto-intoxication consequent on acute yellow atrophy.

In all cases the kidneys showed some degenerative changes in the epithelial cells. In some there was evidence of a nephritis which in a certain number was haemorrhagic. In none of Dawson's or Stokes's cases was there found any enlargement of the spleen.

Clinical Pathology.

The scarcity in number of organisms present in infected patients makes it necessary frequently to resort to guinea-pig experiments in order to discover the spirochaete. The guinea-pig is extremely susceptible to the disease; the introduction of infected material into the intraperitoneal cavity is followed, after an incubation period, by a rapid development of typical symptoms associated with a heavy infection, so that it is easy to demonstrate the spirochaete.

In man the blood infection is of short duration, and after the fifth day of the disease the chances of a positive result diminish. The infection, as already mentioned, is not heavy, so that blood film examinations are usually negative. Introduction of some of the blood (3 c.c.m.) into the peritoneal cavity of a guinea-pig is likely to give a positive result up to the seventh day of the illness.

The urine from the tenth day to the fifth week usually contains spirochaetes, and the centrifugized deposit may yield a positive result on microscopical examination, or a portion injected into a guinea-pig may reproduce the disease in it. The spirochaete obtained from the urine is agglutinated by the blood of the patient after the fourteenth day of the disease.

After the fourteenth day of the disease protective substances conferring immunity are found in the blood, and an immunity test with guinea-pigs was described by the Japanese workers and has been confirmed by Stokes and Dawson.

Mortality.

The Japanese workers found a high mortality (about 30 per cent.). Stokes put the mortality in his cases at less than 6 per cent. Dawson estimates it as not more than 4 to 5 per cent. in his series.

A comparison of the symptoms of spirochaetosis icterohaemorrhagica with those of epidemic catarrhal jaundice renders it clear that the two diseases are quite distinct. In the latter the low mortality, the premonitory period of several days before the onset of pyrexia, the absence of herpes and of haemorrhagic symptoms, the absence of symptoms of severe toxæmia, the common occurrence of splenic enlargement, are prominent distinguishing features.

Weil's Disease.

In this type of jaundice, described by Weil in 1886, the symptoms resembled in many respects those of spirochaetosis icterohaemorrhagica, but important points of difference are the high mortality (30 per cent.) and the marked splenic enlargement. The German workers strenuously deny that Weil's disease is identical with spirochaetosis icterohaemorrhagica, either clinically or bacteriologically.

It is, in my opinion, most desirable to abolish the term "Weil's disease" entirely. For cases of that type, in which no specific spirochaete or other infection are found, it would be far better to use the term "haemorrhagic infective jaundice."

Kartulis described¹⁸ a form of haemorrhagic infective jaundice which he studied carefully in Egypt. It has been called by various writers Mediterranean yellow fever, Weil's disease, spirochaetosis icterohaemorrhagica, and by other names. Such a method of nomenclature is, to

say the least of it, most unscientific. Until the cause of a particular type of disease has been conclusively proved, some general term such as "infective haemorrhagic jaundice" should be given it.

JAUNDICE IN ANAEMIA.

In chlorosis jaundice is almost invariably absent.

In pernicious anaemia a waxy, lemon yellow tint of the skin is common, but a definite yellow tint affecting both skin and conjunctivae is rare. In a series of 1,200 cases described by Dr. R. C. Cabot,¹⁹ in only 12 was the yellow tint so distinctive that the term jaundice could be applied. Marked jaundice scarcely ever occurs in this disease.

In Banti's disease, or splenic anaemia, jaundice is a rare symptom. If the splenomegaly is accompanied by progressive cirrhosis of the liver, jaundice, usually slight, is present.

ACHOLURIC JAUNDICE.

The congenital type of acholuric jaundice was first described by Minkowski in 1900, and since then numerous cases have been recorded. Its characteristic features are:

1. A family history of the disease.
2. Chronic splenomegaly.
3. Maintenance of fair health for a long period.
4. Chronic slight jaundice.
5. Presence of urobilin but absence of bile pigment in the urine.
6. Increased tendency to haemolysis of the red blood corpuscles.
7. Bile pigment in the blood serum.

I have recently examined, at St. Mary's Hospital, a very typical case of this disease which is under the care of Dr. F. S. Langmead, and it is by his kind permission that I quote the notes of the case.

CASE.

H. S., a man aged 42, was sent to hospital with a very accurate and detailed medical history by Dr. R. Craik.

The patient came into medical hands because he had been called up for active service in October, 1917; though his spirit was willing he felt his strength to be weak, and he wisely sought medical advice. He complained of breathlessness on exertion and inability to do heavy work, though his health had been fairly good and he had for some time done light work. At this time he was anaemic; the heart was somewhat dilated, with a haemic systolic bruit at the apex. The spleen was enlarged, but definite jaundice was not present. There was a history of previous "yellowness," especially following cold or exertion. In 1918 slight jaundice was observed on several occasions, but its depth varied from time to time.

When examined there was definite slight jaundice affecting both the skin and conjunctivae, pallor, marked splenic and some hepatic enlargement, and dilated heart, with a haemic systolic bruit at the apex region. There was no history of syphilis or alcoholic indulgence. The family history, however, was striking. Of his four children, two girls, aged 12 and 10 respectively, were jaundiced, one girl aged 5 was healthy, a boy had died "delicate," aged 3. H. S. had two sisters who died at the ages of 18 and 8 respectively of jaundice and dropsy, another sister died at 29 from "heart disease," a fourth sister died at the age of six months, and his only brother died at birth. The patient's mother and grandmother both suffered from jaundice.

Blood examination at St. Mary's Hospital:

Red blood corpuscles ...	2,600,000 per c.mm.
Haemoglobin ...	42 per cent.
Colour index ...	0.8 per cent.

Haemolysis of red blood corpuscles commenced in 0.5 per cent. salt solution, as compared with 0.4 per cent. for normal red cells. The leucocytes numbered 14,000 per c.mm.; there was nothing noteworthy as regards their variety. The urine contained a large excess of urobilin, but no bile pigments.

In this form of jaundice the icterus is not always present at birth, but may develop in childhood.

Cases with no family history have been described by Osler, Eason, Parkes Weber, Rowland Hill, and others.²⁰ The jaundice and other symptoms have the same characters as the congenital form.

Hereditary icterus occurring at birth has been studied by Dr. John Thomson,²¹ who has given classical examples of its hereditary nature. He attributes the disease to a congenital narrowing of the bile duct, coupled with an inflammatory condition set up by the altered bile. The congenital acholuric jaundice already described comes under this head.

ICTERUS NEONATORUM.

Jaundice in the newborn may be physiological, a fairly common mild form which soon passes away, or a severe type due to (a) congenital absence of the common bile duct; (b) congenital syphilitic hepatitis; (c) an infective

form set up by septic infection from the umbilicus, or more commonly by an infection of intestinal origin.

In this, unlike other forms of jaundice, the central nervous system often shows bile staining.

ACUTE YELLOW ATROPHY OF THE LIVER.

Acute yellow atrophy, described in the textbooks as a separate disease, is a condition of common occurrence after many chemical poisons and many infective processes. Its not infrequent occurrence in pregnancy, due no doubt to the production of autogenous poisons, is of special interest. It is a morbid anatomical condition of the liver due to some toxic cause, and with increased knowledge will cease to be regarded as a separate entity.

The morbid anatomical condition of the liver known as "acute yellow atrophy" causes loss of hepatic function, and this gives rise to the "symptom complex" known as "icterus gravis." It is in this sense that I have used the term "icterus gravis" throughout these lectures, in the course of which it has been amply demonstrated that the loss of function of the liver, symptomatically expressed by "icterus gravis," may result not only from a condition associated with shrinkage ("acute yellow atrophy") but also, as in delayed chloroform poisoning, from an extensive and rapid degenerative change of the liver cells followed by loss of function, no diminution in size or "atrophy" of the organ occurring.

CONCLUSION.

The above review of the subject of jaundice emphasizes the paramount importance of observation and experiment in determining the cause of disease and gaining exact knowledge. Down to 1881 knowledge had only enlarged upon that of Hippocrates as the result of the observations through *post-mortem* examination in fatal cases; this could go so far, but no further. In 1881 came the great advance due to animal experiments, of which those on dogs were of essential importance. Stadelmann, and in this country Hunter, by these experiments advanced our knowledge enormously. Since then progress has been steady and has been very great during the period of the war; every step has been the result of observation and experiment, and without the bacteriological, chemical, and physiological investigations in the laboratory we should have made little, if any, advance.

The history of jaundice is an object lesson showing the grounds on which scientific medicine should be based in the future.

In the study of disease clinical observation is of first importance, but it can carry us only a certain distance towards the goal of full knowledge. Clinical observation by the physician must go hand in hand with scientific experiment by the experimental worker if progress is to be sure and rapid. Neither alone can achieve the result. The physician and the scientific worker in the laboratory should be familiar each with the other's work and aspirations, and should ever lend a guiding hand to one another. The medicine of the future will attain that perfect advancement and full knowledge which all desire by the association of the physician and the scientific worker, not only in the laboratory, but also at the bedside.

ACKNOWLEDGEMENTS.

For very valuable help in the work on which these lectures are based I am especially indebted to Dr. B. H. Spilsbury, Dr. T. M. Legge, and to Mr. J. Webster, F.I.C., for their great assistance in the investigations relating to toxic jaundice.

For the work done during my period of military service I am very grateful for the help and encouragement always afforded me by Major-General Sir Francis Treherne, K.C.M.G., D.M.S. of the Mesopotamian Expeditionary Force (1916-1917), and to Major-General A. P. Blenkinsop, C.B., C.M.G., D.M.S. of the Mesopotamian Expeditionary Force (1917-1919). I am specially indebted to General Blenkinsop for the statistical figures quoted relating to epidemic jaundice. I desire especially to thank for very valuable help Lieut.-Colonel J. C. G. Ledingham, C.M.G., R.A.M.C., Lieut.-Colonel J. D. Graham, C.I.E., I.M.S., Major F. P. Mackie, C.I.E., I.M.S., Major R. S. Archibald, R.A.M.C., Major T. H. Gloster, I.M.S., Major Dunbar Walker, R.A.M.C., Captain M. A. Nicholson, I.M.S., Captain R. A. Woodhouse, R.A.M.C., and Captain Jarvis, R.A.M.C.

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Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

ACRIFLAVINE IN THE TREATMENT OF GONORRHOEA. 798

THE recent paper by Davies and Harrell,¹ of the Urological Institute, Johns Hopkins Hospital, an abstract of which appears in the *BRITISH MEDICAL JOURNAL* of March 22nd, 1919, is further evidence, if any be needed, of the discordant views of different observers. The writers point out that acriflavine is highly diffusible, and that it penetrates the mucosae of the urethra and bladder, that it is an antiseptic of high power, and inhibits the development of the gonococcus in dilutions of 1 in 300,000. There is no doubt that in the laboratory these results can be, and have been, obtained. But I am certain few who have had any large experience in the treatment of gonorrhoea will agree with the writers regarding its action on the living subject. They state the most satisfactory concentration is found to be 1 in 1,000; that the smarting is almost negligible, and that organisms disappear from the urethra after two or three injections. I have used this preparation, selecting 23 cases of ordinary uncomplicated gonorrhoea, carrying out the exact régime laid down by the writers. Of these 23 cases, 6, or 26 per cent., showed that at the end of three weeks Gram-negative diplococci, morphologically indistinguishable from gonococci, and purulent beads, were present within twenty-four hours after stopping the injection. Of the remaining 17, 13, or 76 per cent., showed pus cells in their smears, and their anterior washes, when centrifugalized, had a purulent deposit. The remaining 4 showed mucus and epithelium. Far from causing only slight smarting at the strength recorded, the patients treated complained at the end of ten days of severe smarting and dysuria, which has never been a symptom in any cases treated with ordinary solutions of protargol. I have found that in cases of submucous infiltrations or in soft strictures associated with gonorrhoea the remedy is apt to produce retention of urine. The fact of the urine remaining yellow for hours and the leucocytes being well stained is of little clinical significance. The remedy, I believe, has had an extensive trial at Rochester Row under the very careful observation of Colonel Harrison, and his views on the treatment of gonorrhoea have been stated before the Royal Society of Medicine, but I doubt if he even referred to this remedy.

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London, W.

HYPERPYREXIAL HEATSTROKE.

THE article by Mr. K. G. Hearne on hyperpyrexial heatstroke, in the *JOURNAL* of April 26th, leads me to record a case which occurred in the hospital to which I was attached in Mesopotamia. It supports the view that the condition is due to cessation of sweating, as in the case in question the condition was accidentally caused by a hypodermic injection of atropine.

¹ *Journal of Urology*, vol. ii, 1918.

It was the custom on the surgical side to give every patient before operation a hypodermic injection of morphine gr. $\frac{1}{4}$ and atropine sulphate gr. $\frac{1}{100}$ unless specially contra-indicated. The injection was given by the sister in charge of the ward. In 1917 the intense heat of summer commenced suddenly, and owing to a misunderstanding the injection of atropine was not countermanded. The anaesthetist noticed that one of the first cases to be operated upon after the heat wave broke was unduly unconscious, and that the patient's skin felt hot and he was not sweating. The thermometer showed the temperature to be 108° F.; the operation was suspended and an ice pack brought the temperature down within the limits of safety. On succeeding days the temperature showed no tendency to rise, thus differing from ordinary cases of heatstroke. This was the only case which occurred in the theatre; as it was built of brick the temperature in it was usually 12° to 15° lower than that in the wards, which were constructed of mud.

I entirely agree with Mr. K. G. Hearne that hyperpyrexial heatstroke is preventable among hospital patients, but in my experience the "incubation period" before the onset of delirium, unconsciousness, etc., should be allowed a wider margin than from one to forty-eight hours. I have seen sweating men while accomplishing a necessarily forced march during the heat of the day struck down with hyperpyrexia of 110° and accompanying symptoms within a few minutes. Doubtless the combination of external heat and heat derived internally from muscular exertion caused the abruptness of onset in these cases. On the other hand, one case gave a clear history of premonitory symptoms—sense of oppression, marked restlessness, frequency of micturition, etc.—extending over a period of four days.

Devonport.

R. J. McNEILL LOVE, M.B., B.S.Lond.

RUPTURE OF BRANCH OF THE CORONARY ARTERY.

ON April 18th Mrs. E. H., aged 80, was admitted to the infirmary with a diagnosis of arterio-sclerosis and bronchopneumonia. The heart was rapid but regular, and there was no bruit. There was a history of a fall off a chair a few days before admission, and there were some bruises and abrasions on the back. A history of chronic bronchitis and occasional attacks of giddiness was elicited.

About an hour and a half after admission I was urgently sent for. I found the patient suffering from syncope; the pulse at the wrist could not be felt, though the heart sounds were faintly audible. She rallied a little under the influence of cardiac stimulants, but died in about three hours afterwards. She never complained of any pain in the chest.

As there was a distinct history of a fall, the matter was reported to the coroner, and I made a *post-mortem* examination by his order. There were evidences of general arterio-sclerosis; there was oedema of the cortex and the whole of the brain, and its blood vessels were very anaemic. The kidneys showed signs of an early stage of arterio-sclerosis; other abdominal viscera were normal. There was evidence of chronic bronchitis and emphysema with commencing bronchopneumonia, and old adhesions between the right lung and the pleura. The pericardial sac contained a large clot of blood weighing over 3 oz.; its source was found in a rupture of a branch of the right coronary artery. The muscles of the heart, though flabby and exhibiting fatty degeneration, were quite intact. The aortic valves showed a certain amount of thickening. The aorta and the coronary vessels showed atheromatous patches. Possibly a fit of coughing accounts for the sudden rupture of the degenerated coronary vessel.

In searching the literature on this subject, the only reference that I have come across is by Sir Clifford Allbutt in his book on diseases of the arteries, page 368, where he says: "... as I have twice observed, haemorrhage into the pericardium from a ruptured coronary was quite painless." This being so, I have no hesitation in recording this very rare occurrence.

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THE General Association of French Medical Practitioners, at its annual meeting on May 4th, unanimously elected Dr. Darras president for a term of six years.