concerned, shows no change of practical importance or detectable by any other method.

The passage of impulses along a nerve cannot be detected directly in any other way than by the electrical effect. Einthoven has been able to show the passage of impulses in the vagus nerve when the lungs are distended or collapsed, and also in the depressor nerve when the blood pressure rises in the aorta. Piper has determined the rate of discharge of motor innervation. The method is capable of further extension to reflexes, and possibly the vexed question of the analysis of sounds by the cochlea may be decided.

The electrical changes in secreting glands are more complex than in nerve and muscle, and are not yet completely understood. The main component is undoubtedly connected with the flow of water, as shown by Bradford and myself, and to explain it we may consider briefly the nature of the secretory process in general. The cells possess the same kind of membrane as muscle has, but their impermeability appears to be to certain cations of the cell contents, not to anions, as judged by the sign of the electrical effects. This is, however, a matter of detail. During rest there is formation of complex substances to be used in the act of secretion. This is an automatic process, requiring the supply of energy by oxidation, and comes to an end by mass action of the products. When secretory activity occurs, two things happen. The stored products are split into smaller molecules, so that the osmotic pressure of the cell contents rises and water is attracted. But simultaneously that end of the cell in relation to the duct becomes permeable. Water flows out, carrying the secretory products. The electrical effect is accounted for in a way similar to that in the case of muscle. But there is also another component of the electrical change, smaller in magnitude and opposite in sign to that associated with the permeability effect. This is seen when the sympathetic supply to the submaxillary gland of the dog is stimulated. Its origin is unknown.

Sweat glands in the skin show the phenomenon of an electrical change. They are readily excited reflexly and, as Tarchanov and Wells and Forbes have shown, also by psychical events. It seems practically certain that the "nerve-leaks" of Baines are due to local activity of sweat glands. These may possibly be of use in the location of pathological processes in the central nervous system, but there are simpler means of detecting local sweating.

Waller has investigated the changes of resistance in the skin, which are associated with mental states, and proportional to the degree of the emotion. The phenomenon is called by him the "emotive response," and he is inclined to doubt its origin from sweat glands. But the evidence derived from atropine is not very satisfactory. The way in which the decreased resistance is produced in active cells has been described above. The reaction may be found useful in the process of "psycho-analysis" and has been used by Wells and Forbes in psychological investigations.

Photochemical reactions are associated with electrical changes, and the phenomena in the retina have been the object of much attention. They are complex in the vertebrate retina, probably on account of its mixed structure. In the cephalopod the effect of light is simple. When better understood, they will doubtless help to explain the retinal processes. The action of light on the green leaf has been shown by Waller to be accompanied by electrical effects. This again requires further work.

Finally, the process of inhibition in heart and smooth muscle may be referred to. Since increased activity means increased negativity, it is clear that decreased activity should be associated with decreased negativity. This was shown by Gaskell to occur in the tortoise auricle on stimulation of the vagus. In the ureter, it appears that a wave of inhibition precedes that of contraction, which propels the drops of urine downwards, a process similar to that described by Starling and myself in the case of the intestine. This would naturally result in a double electrical effect at each electrode and a complicated curve of the total effect, such as is described by Orbeli.

At the twenty-ninth annual meeting of the Association of Medical Colleges in Chicago a new constitution and by-laws were adopted, the principal modifications being in the high school and college requirements for admission to medical schools.

Lettsomian Lectures

ON

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

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LECTURE III.—PART I.

EPIDEMIC CATARRHAL JAUNDICE.*

This disease was very frequent in the Dardanelles campaign, in Egypt, and in Mesopotamia, and there is no doubt that it occurred frequently also in other war areas. The symptoms and course of the disease form a characteristic clinical picture, and undoubtedly constitute a special type of jaundice the exact pathological cause of which has not been ascertained.

I have had the opportunity of studying closely a very large number of cases, particularly in the Dardanelles and Mesopotamia, and the advantage of the co-operation of distinguished bacteriologists who have made the most painstaking and thorough examinations. From my own clinical observations, and from the results of these bacteriological examinations, I have not the slightest doubt that the epidemic catarrhal jaundice was a special entity, entirely distinct from *spirochaetosis ictero-haemorrhagica*.

The first published clinical description of the disease was by Lieutenant E. B. Gunson, R.A.M.C., and Lieutenant I. W. C. Gunn, R.A.M.C.,¹ who gave an interesting and accurate account of a series of cases in British troops at Alexandria in June, July, and August, 1915, nearly all from a single camp. In July, 1915, at a general hospital in Alexandria, the late Lieut.-Colonel Lister, R.A.M.C., who was in command of the Medical Division, kindly showed me a number of these cases, and I had the opportunity of examining them. I should like to take this opportunity of expressing the great admiration and regard of all who knew him for the skill, sympathy, and devotion shown by Lieut.-Colonel Lister in the splendid medical work which he carried on unremittingly in Egypt even when stricken himself by an incurable malady which later called him from us.

On February 26th, 1916, an account of the epidemic jaundice in the Dardanelles was published by myself.²

In 1916 Major R. S. Archibald, R.A.M.C., and his fellow workers, Captains Hadfield, Logan, and Campbell, in their bacteriological reports³ from the Mudros laboratories, gave a full account of a great deal of careful work carried out on the cases of jaundice at the Dardanelles. On April 7th, 1917, a paper on the pathology and etiology of this disease was published⁴ by Lieut.-Colonel C. J. Martin, A.A.M.C. Hurst also refers⁵ to the disease in his book on the *Medical Diseases of the War* (1917).

In Mesopotamia I have studied closely a very large number of cases of this type of jaundice, and the most thorough and exhaustive bacteriological investigations have been carried out by Major Mackie, I.M.S., Major Gloster, I.M.S., Major Stevenson, I.M.S., and Captain Short, I.M.S.

SYMPTOMS.

The patient had usually some abdominal discomfort for a few days, associated with anorexia, nausea, perhaps occasional vomiting, and either diarrhoea or constipation. Sometimes there was a history of diarrhoea or dysentery during the previous few weeks.

Pyrexia of a mild type would now generally set in, associated with some chilliness and occasionally some shivering. The tongue was moist and often furred, but might be quite clean. Vomiting often occurred at this stage, and slight headache was common. "Pains all over" were sometimes mentioned in the early stage of pyrexia. Abdominal discomfort was usually present in the hepatic and epigastric regions. The pyrexial period in some cases lasted only one or two days, but was generally

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

from two to four days; rarely it lasted longer. (See charts.)

About the third or fourth day from the onset of pyrexia jaundice occurred; it was slight at first, but gradually deepened and became of the ordinary obstructive type, with pale stools and bile-stained urine. The jaundice usually attained a maximum in about ten days and then gradually subsided, but the duration and intensity of this symptom varied.

Definite uniform enlargement of the liver could be made out by palpation as the jaundice developed, slight tenderness being present. Some cases showed definite swelling in the gall bladder region, and in about 10 per cent. of cases palpation afforded indications of enlargement of the gall bladder.

Enlargement of the spleen accompanied that of the liver, and the organ could usually be felt extending on inspiration for half an inch or more below the left sub-costal border. With the subsidence of the jaundice the enlargement of the liver and spleen gradually diminished.

CHART 2.—Jaundice was present on September 24th; on the 26th, liver and spleen enlarged, right heart dilated.

During the pyrexia the pulse was quickened to 80 or 90, but with the onset of jaundice it became slow—about 50 in some cases.

With pyrexia lasting three or four days or more a definite increase in the deep cardiac dullness could be made out about the third day, and the interesting feature was that the dullness was mainly on the right side; on percussion in the fourth intercostal space to the right of the sternum the dullness extended one or two inches to the right of the sternal border. The late Dr. D. B. Lees, to whose masterly teaching of physical signs so many of his pupils will ever be indebted, pointed out this phenomenon in cases of acute rheumatism many years ago. I know of no acute disease other than these two in which specific dilatation of the right side of the heart occurs without any lung involvement or mechanical causes to produce back-working. This cardiac condition in epidemic jaundice was first pointed out to me by Lieut.-Colonel Lister in Alexandria, and I have since confirmed it by observation in a large number of cases both in the Dardanelles and Mesopotamia. With rest in bed no symptoms of cardiac embarrassment occurred. The cardiac dilatation usually lasted for four or five days only, and then subsided.

The urine in the jaundice stage showed the presence of bile as in obstructive jaundice. Albumin and casts were absent, save in those exceptional cases developing icterus gravis. The duration of the obstructive jaundice stage was not sufficiently long for such skin complications as pruritus, boils, etc., to appear. Anaemia, herpes, congestion of the conjunctivae, and haemorrhagic symptoms were not observed, and special nervous symptoms other

than asthenia and depression did not occur in the ordinary type of case. A secondary rise of temperature in the course of the illness was very uncommon.

It is important that a correct perspective should be obtained of the disease "epidemic catarrhal jaundice." I have very careful notes of sixty-four consecutive cases at the Officers' Hospital, Baghdad, made by Captain Woodhouse, R.A.M.C., who was in charge of them. I saw these patients in consultation with him many times, and can confirm the careful accuracy of his records. Of these cases sixty-three were of the mild type described above. I will quote the notes of three of them taken at random.

CASE I.

Lieutenant F. Diarrhoea and abdominal discomfort for four days, then slight fever, reaching 102.5° F. on the sixth day, and since then normal temperature. On the eighth day tongue lightly furred; icteric tint of skin and sclerotics now noticed. The deep cardiac dullness was increased to right, liver enlarged, tenderness over gall bladder. Urine contained a considerable amount of bile, stools clay coloured. He made an uninterrupted recovery and was convalescent on the twenty-sixth day.

CHART 3.—Jaundice appeared five days before admission to hospital. V. Vomited.

CASE II.

Lieutenant G. Nausea and feeling of illness with slight diarrhoea for seven days; he passed some mucus on the seventh day, but no dysentery organisms were found on examination. Jaundice appeared on the sixth day. On examination on the tenth day liver enlarged and tender with tenderness over gall bladder, spleen palpable, urine contained bile, and stools were pale. Marked anorexia, depression, and pain and tenderness over the liver continued up to fourteenth day; on this day he vomited after an enema. The liver and spleen now gradually diminished to normal size and he made an uninterrupted recovery, being transferred down stream on the twenty-sixth day of the illness.

CASE III.

Captain J. Nausea and occasional vomiting, with constipation for four days, then slight fever. Jaundice appeared on seventh day. The liver and spleen were slightly enlarged (tenth day). Heart not dilated. Jaundice commenced to disappear on the thirteenth day and on the twenty-third day patient was convalescent.

Usually cases of epidemic catarrhal jaundice gave rise to no anxiety, though I have occasionally seen much hepatic enlargement in which most persistent vomiting occurred from the eighth to the twelfth day, and in one case rectal feeding had to be adopted.

In patients with marked hepatic and splenic enlargement convalescence was slow, and they were not usually fit for duty within two or three months.

TREATMENT.

The treatment of the ordinary type of case consists of rest in bed and light diet, with saline aperients and an alkaline mixture of potassium citrate and sodium bicarbonate. It is advisable to give oranges, and drink made from fresh lemon juice as antiscorbutics. In the treatment of such conditions as jaundice, the enteric group, or dysentery requiring special diet for a long

CHART 5.—Jaundice before admission. Patient vomited on December 31st; the following day he was dull and morose; was delirious on January 2nd; coma supervened on January 3rd, and he died on January 4th. Post-mortem: Acute yellow atrophy of liver.

* All the charts are of cases of epidemic catarrhal jaundice.

period in hot climates, the rapidity with which scurvy or vitamin deficiency diseases will develop must always be borne in mind.

In the Dardanelles I called attention⁶ to the development of multiple neuritis of beri-beri type in several cases of epidemic jaundice. This was undoubtedly due to the rigid dieting on sterilized foods, no fresh food being available for such cases. Calomel in small repeated doses should never be given in cases of epidemic jaundice, because, as was, I think, first pointed out by me,² there is here a great liability for the development of severe mercurial stomatitis. I saw several such cases in the Dardanelles. A single dose of calomel may be given without danger, but it and other powerful purgatives are best avoided.

Development of "Icterus Gravis" Symptoms.

Although, as already described, epidemic catarrhal jaundice usually runs a mild course without any serious symptoms, one can never be quite free from anxiety as regards a patient. In rare cases at some period of the jaundice stage acute toxic symptoms may develop exactly as in "icterus gravis," and almost invariably death ensues within about two or three days of the onset of the grave symptoms. I have seen several such cases and have notes of fifteen, on most of which careful *post-mortem* examinations were made.

The grave symptoms usually occurred a few days (about ten) after the appearance of the jaundice, in one case two days, in another four, while in one case the interval was as long as twenty-nine days.

There appeared to be no relation between the depth of the jaundice and the development of the grave toxæmia. Some cases were not deeply icteric at the onset of the toxæmic symptoms, but the jaundice increased as these developed. The preliminary symptoms of the illness were exactly like those of the ordinary type of the disease and gave no warning of the future grave development. In one case the symptoms of the preliminary stage of the jaundice were so slight that the patient did not report sick.

CASE I.

In January, 1917, when our troops were busily engaged in the fighting around Kut, preceding the crossing of the Tigris at Shumram and advance to Baghdad, an Indian soldier was sent from the front line to a field ambulance because of the development of curious mental symptoms. He had been found wandering about naked though the weather was very cold, and his mental condition was quite abnormal. I saw him on admission when he was markedly jaundiced; he had slight delirium and the liver dullness was considerably diminished. He rapidly became comatose and died in forty-eight hours, from typical symptoms of acute yellow atrophy of the liver.

This was undoubtedly a case of epidemic catarrhal jaundice in the course of which icterus gravis developed. I quote three other very interesting cases on which careful pathological examinations were made:

CASE II.

Gunner R. had had jaundice for eight days with the usual symptoms of the epidemic catarrhal type, temperature normal.

At 10 a.m. on the ninth day, coffee-ground vomit with streaks of blood, tremors, and drowsiness. At 5.30 p.m. he was delirious and shrieking; at 6 p.m. comatose, Cheyne-Stokes breathing, knee-jerks present, extensor plantar reflex, and incontinence. The liver dullness was much diminished, measuring only 2½ in. vertically; deep coma persisted. On the morning of the eleventh day the temperature rose suddenly from normal to 104°, and death occurred.

Post-mortem Examination.—Liver small, yellow, with small dark-reddish areas. Kidneys: epithelial degenerative changes. Petechial hæmorrhage in the pyloric portion of the stomach, and small hæmorrhages on the wall of the intestine, in the pleura, pericardium, and lung tissue. Major Gloster, I.M.S., made careful microscopical examinations and animal experiments for spirochaetes, with negative result.

CASE III.

Lieut. S., taken ill December 24th, 1917, with slight fever and the usual symptoms of epidemic catarrhal jaundice. On December 28th temperature normal, liver enlarged and tender, spleen palpable and slightly tender, tongue furred, constipated, definitely jaundiced, urine bile-stained, mental condition normal. Several attacks of vomiting without blood occurred during the next three days, but on January 2nd, 1918, the vomit

contained streaks of blood and was bile-stained. Jaundice was now deep. He was very restless and maniacal during last night and this morning. On January 3rd the vomit was the same. Epistaxis in morning, liver dullness much diminished, being only 2 in. vertically; knee-jerks increased. Very restless and noisy. Next morning he became comatose, urine contained albumin, bile, and a few red corpuscles; no spirochaetes found in urine or blood. At 5 p.m., Cheyne-Stokes breathing, extensor plantar reflex; at 10 p.m. the temperature rose suddenly to 109°, and death occurred at 10.30 (Chart 5).

Post-mortem Examination (by Lieut.-Colonel Ledingham, R.A.M.C., and Major Gloster, I.M.S.).—Liver 39½ oz., stained yellow. Spleen enlarged and soft; kidneys yellowish. Small hæmorrhages in pleura, pericardium, omentum, mesentery, and in lungs. The wall of stomach and duodenum contained petechiae, and their contents were blood-stained. The liver showed almost complete destruction of hepatic cells in blocks; only isolated islets of cells remained; in parts total necrosis of all but trabecular tissue. The kidney showed extensive degeneration of tubular cells; the pancreas, early but definite cell necrosis, with shrinkage and loss of outline of the cells that remained; in some places great reduction in number of cells.

Microscopical examination and animal experiments for spirochaetes of liver, kidney, and spleen were negative.

CASE IV.

I am indebted to Captain F. B. Ambler, I.M.S., for the clinical and *post-mortem* notes of this case.

An Indian was admitted to hospital on October 6th, 1917, with jaundice of the usual type; temperature remained normal until just before death. On October 10th he was constipated, the liver enlarged, tongue furred. Next day he had incontinence, was noisy and irritable, and vomited some blood-stained fluid; pulse 92; blood culture negative; became unconscious in the evening; knee-jerks now increased. Ankle clonus and extensor plantar reflexes present. On October 12th the jaundice was deeper; liver dullness very much diminished; coma continued. Temperature rose to 101° at 6 p.m., pulse 140. Death occurred at 7 p.m.

Post-mortem Examination.—Petechial hæmorrhages in gall bladder, mesentery, and omentum. Spleen slightly enlarged. Liver very small; the surface on section was of dirty khaki colour, with small raised reddish patches like cirrhotic areas scattered through. Some larger reddish patches were also present.

Cases per 1,000.

CURVE 1.—Showing the incidence rate of epidemic jaundice and diarrhoeal diseases in the Dardanelles, per 1,000 troops in one division, for the period August 7th to December 19th, 1915.

CURVE 2.—Showing incidence rate of epidemic jaundice and dysentery in Mesopotamia, 1917-18. A, Jaundice. B, Dysentery.

From these accounts it will be seen that the terminal fatal symptoms in epidemic catarrhal jaundice are closely identical with those of delayed chloroform poisoning and of the other toxic liver poisons. The *post-mortem* signs also are exactly identical. Atrophy of the liver or extensive degeneration are the pathological results of either condition.

Etiology.

Epidemic catarrhal jaundice had its maximum incidence in Mesopotamia in December and January; in the Dardanelles in October. In both areas there was a close association with the incidence of "dysentery"—that is, enterocolitis affections. Thus the charts for the two diseases in both areas indicate that the jaundice curve attained its maximum about three weeks after that of dysentery. (See Curves 1 and 2.)

There must be an etiological association between the two diseases—for example, food or water infection—and it is likely that the enterocolitis resulting from a "dysenteric" affection in some cases prepared the way for the infection causing epidemic catarrhal jaundice. This view is borne out by the symptomatology of the disease, for, as we have seen, there was commonly a previous history of diarrhoea or dysentery in the jaundice cases, and also it was not uncommon for jaundice to develop in patients actually suffering from dysentery in hospital.

The epidemic character of the disease is beyond doubt, and in the Dardanelles and in Mesopotamia there were numerous instances of a large percentage of cases occurring in one unit. The danger of infection from person to person does not seem to be greater than in enteric fever, for instances of infection from patient to patient in hospital were not common. The epidemic character appeared to be due to a common cause rather than a spread from person to person. No evidence of air-borne infection was obtained from the study of the disease in the Dardanelles and in Mesopotamia.

The association of the curves for epidemic catarrhal jaundice with those for dysentery, diarrhoea diseases, and enteric group infections, indicate an intestinal origin for the infection.

In Mesopotamia jaundice was wisely made a notifiable disease, and records have been kept for 1917 and 1918. In 1917 there were 1,538 cases with 0.6 per cent. deaths in British troops, while in Indian troops there were 2,634 cases with 0.3 per cent. deaths, the gross mortality being 0.41 per cent. These low mortality figures are in striking contrast to the similar figures from spirochaetosis icterohaemorrhagica, or the so-called Weil's disease.

It is interesting to note that an epidemic of jaundice closely identical with the epidemic catarrhal jaundice above described was reported⁷ by Captain G. C. Gray, R.A.M.C. In the Cambridgeshire area between April and November, 1917, seventy cases were investigated. In one instance four cases occurred in the same house, in another three, and there were four instances of two cases in the same house. No deaths occurred. Also Dr. S. Harthill described⁸ a similar epidemic occurring in Hertfordshire between October, 1917, and March, 1918. He gives details of fifteen cases, none of which were fatal.

Bacteriology.

In the Dardanelles cases Lieut.-Colonel C. J. Martin, Major Archibald, and Captain Campbell, R.A.M.C., made numerous blood cultures in cases of jaundice. In no case of clinical "epidemic catarrhal jaundice" was any specific organism found. Several cases yielded enteric or paratyphoid organisms, but these subsequently ran the typical clinical course of these diseases. Examinations of the faeces and urine gave no definite results. Similar investigations were carried out in Mesopotamia by Major Mackie, I.M.S., Major Gloster, I.M.S., and others, with like result.

Martin and Hurst investigated some of the Dardanelles cases by withdrawing fluid from the duodenum with Einhorn's evacuator, but no definite results were obtained.

In six out of eight cases of jaundice a bacillus of the *faecalis alkaligenes* group was obtained, but this organism was obtained also in three out of four from healthy patients. Some of the cases showed other bacilli and cocci in scanty numbers.

In the Dardanelles Sarraihé and Clunet⁹ found on blood culture organisms of the paratyphoid group in the jaundice cases. In some of these paratyphoid B was

found; later they found an organism which did not give the agglutination reactions for paratyphoid A or B, and which they named paratyphoid D. These observations have not been confirmed by our bacteriologists.

In the Dardanelles Martin made numerous examinations for spirochaetes with negative result, but was unable to carry out animal experiments as guinea-pigs were not available.

In Mesopotamia the most careful observations on cases of epidemic catarrhal jaundice in all stages of the illness were made by Lieut.-Colonel Ledingham, R.A.M.C., Consulting Bacteriologist to the Force, Major Mackie, I.M.S., Major Stevenson, I.M.S., and Captain Shortt, I.M.S.

The blood was examined in the early stages, and the urine also in the later stages. Liver punctures were made during life, and the organs were examined *post-mortem*. In no case was the *Spirochaeta icterohaemorrhagiae* found, though guinea-pig experiments were freely made in addition to the other methods of investigation. In two cases during life Mackie found organisms of the *B. coli communis* type—in one from liver puncture, in the other from a catheter specimen of urine. In fatal cases organisms of *B. coli communis* type were frequently found in the liver, but may have been *post-mortem* contaminations.

It has been conclusively proved that in the epidemic catarrhal jaundice of Mesopotamia the disease was not caused by a spirochaetal infection, and the exact similarity of the Dardanelles cases to those in Mesopotamia justifies the same conclusion for them.

Neither in the Dardanelles nor in Mesopotamia was there any association whatever between occurrence of the disease and the presence of rats. Indeed, in Mesopotamia numerous cases of jaundice occurred in camps in the desert where no rats existed for miles around. Major Mackie collected with some difficulty a number of rats from towns in Mesopotamia, and conducted careful guinea-pig experiments with them, but in no case did he find any evidence of a spirochaetal infection.

A specific organism causing "epidemic catarrhal jaundice" has not yet been found. The symptoms and etiology point to an infection by the intestinal tract, and there is, no doubt, a general blood infection the duration of which is probably very short.

It is not surprising that blood culture examinations have been negative, because, as has been pointed out, when jaundice develops—that is, the earliest time at which a diagnosis can be made—the temperature is either declining or has fallen to normal, and a blood culture examination would be expected to prove negative. In enteric it is well known that positive results from blood culture are usually yielded only in the early period (first eight days or so) of the pyrexia, so that in these cases of jaundice with a short pyrexial period it is probable that blood cultures have been taken too late for a positive result. So few cases prove fatal that opportunities of *post-mortem* examinations are rare, and in the few cases in which death occurs it is usually at such a late period of the illness that bacteriological examinations are rendered difficult by death agony and *post-mortem* contaminations.

I believe that epidemic catarrhal jaundice is probably due to an intestinal infection causing duodenitis and a catarrh of the bile ducts. The area around the ampulla may be mainly affected, and in some cases there may be produced an ascending catarrh causing cholangitis. In either case obstructive jaundice may result.

The above train of events is no doubt associated with a transient general blood infection, of which the causal organism has yet to be proved. It is possible that, in some cases at any rate, the catarrhal condition of the duodenum and bile ducts may be caused by organisms which are found in health in the intestinal tract, of the type of *B. coli communis* or *B. faecalis alkaligenes*.

It is well known that a common "cold in the head" or "nasal catarrh" may be caused in an individual by organisms which are found in his nasal passages in conditions of health. Thus one of the organisms present—for example, the pneumococcus or the influenza bacillus—may become for a time pathogenic and set up nasal catarrh and cause nasal obstruction. It is quite possible that a similar condition of things may occur in epidemic catarrhal jaundice.

Epidemic catarrhal jaundice as above described is not a new disease. It has probably occurred in most campaigns.

Thus in the American war 22,509 cases of jaundice occurred with 161 deaths, and in the South African war 5,648 cases with a small mortality. It is probable that these cases were mainly of the epidemic catarrhal type.

REFERENCES.

- ¹E. R. Gunson and I. W. C. Gunn: An Epidemic of Jaundice occurring at Alexandria, *Lancet*, December 11th, 1915.
- ²Willcox: The Epidemic Jaundice of Campaigns, *BRITISH MEDICAL JOURNAL*, February 26th, 1916.
- ³Archibald, Hadfield, Logan, and Campbell (1916): Reports of the M. and H. Laboratories dealing with Diseases affecting Troops in the Dardanelles, *R.A.M.C. Journal*, 1916, vol. 1, p. 695.
- ⁴Lieut.-Colonel C. J. Martin, F.R.S., A.A.M.C.: Concerning the Pathology and Etiology of the Infectious Jaundice common at the Dardanelles, 1915, *BRITISH MEDICAL JOURNAL*, April 7th, 1917.
- ⁵Hurst: *Medical Diseases of the War*, 1917.
- ⁶W. H. Willcox: "Beriberi," with Special Reference to Prophylaxis and Treatment, *Lancet*, March 11th, 1916.
- ⁷Captain G. C. Gray, R.A.M.C.: Outbreak resembling Epidemic Catarrhal Jaundice in England, *Lancet*, April 6th, 1918.
- ⁸S. Hartill: Epidemic Catarrhal Jaundice in England, *Lancet*, April 20th, 1918.
- ⁹Sarrailhé et Clunet (1916): "La jaunisse des camps et l'épidémie de paratyphoid de Dardanelles," *Bull. et mém. de la Soc. Méd. des Hôp. de Paris*, 1916, 3 Sér., 32^{me} Année.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

TRAUMATIC RUPTURE OF THE HEART WITHOUT FRACTURE OR EXTERNAL LESIONS.

A GURKHA rifleman, aged 19, previous chest measurements 32 to 34, height 5 ft. 1 in., was brought in dead to hospital on account of the following accident: Whilst engaged in fire practice in the regimental lines he was pulling the wheeled fire pump when, coming downhill, it ran away with him, and he was crushed between a stone wall and the cross-bar handle of the pulling shaft, which apparently caught him across the middle of the chest. He was seen to fall dead at once.

Post-mortem Examination.—Externally there was some congestion of the right eye, and a small cut and contusion on the right side of the forehead. No other contusions or lesions were found anywhere

and no fractures or haemorrhages, though the saliva was tinged with blood, possibly from some undiscovered cut in the mouth. The base of the skull was not fractured; there was marked pachymeningitis along the superior longitudinal sinus. The abdomen was natural; the ribs were unfractured, but were very pliable, so that the chest could be easily compressed. The

lungs were natural. The pericardium was full of blood but undamaged.

The heart was very small, being only about 3½ in. long and 3 in. broad. No scales were available at the time for weighing. The valves and heart muscle were apparently natural, and there was no trace of aneurysm or old disease. In the wall of the right auricle, between the right coronary artery and the entry of the inferior vena cava, was a small rent measuring about ¾ by ½ in., as shown in the diagram.

The only explanation I can offer of this curious accident is that at the moment of impact the man may have taken a deep inspiration, engorging the right auricle, which then burst on sudden compression through the costal wall by the cross-bar of the shaft of the fire pump.

A. C. L. BILDERBECK,
Captain I.M.S.

INTRAUTERINE CRYING.

A FEW days since I was called to attend a case of labour in a multipara. Progress was slow, and I injected pituitary extract twice—1 c.cm. doses two hours apart. I then dilated the os as fully as possible, and as the head still remained above the outlet of the brim I decided to use forceps.

Having punctured the membranes the fluid escaped

freely, and a short time afterwards I was surprised to hear muffled but unmistakable crying. The patient being uncovered and already prepared and under chloroform, I applied forceps, but the head being high and moving freely the blades slipped off. During the interval before the forceps were reapplied the crying was again heard quite clearly, and was remarked upon by the husband and nurse.

It was about six minutes from this time that the head was finally delivered, the blades having been previously removed to prevent laceration. The cord encircled the neck twice. The child—a girl—gasped, was easily resuscitated, and cried lustily. Later considerable fluid was removed from the air passages. The child did well.

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Reports of Societies.

THE BACTERIOLOGY OF PYORRHOEA.

At the meeting of the Section of Odontology of the Royal Society of Medicine on May 26th Mr. J. G. TURNER, F.R.C.S., and Mr. A. H. DREW, D.Sc., gave an account of an experimental inquiry into the bacteriology of pyorrhoea. It was described as a continuation of a previous communication on the general microbiology of pyorrhoea, and the further results were now reported because Dr. Drew was about to resume his duties as bacteriologist to the tuberculin department of the Federal Serum Institute, Melbourne. The methods by which the microscopical preparations were made during the present research were fully described, and the results were stated under the following headings:

1. *Living Pulp.*—The living pulp appeared to become readily infected, and such infection was not necessarily associated with caries in the ordinary acceptance of that term. A preparation from the pulp of a bicuspid was demonstrated, showing a diphtheroid infection; in this case there was associated chronic pyorrhoea, but it was thought improbable that it was concerned in the pulp infection. In another case infection by two distinct types of diphtheroids was demonstrated, in others mixed infections by diphtheroids, streptococci, and at times staphylococci, and spirochaetes. Mixed infections were always associated with caries to a greater or less extent, and in at least one instance the vessels of the pulp were found to contain organisms (diphtheroids and cocci).

2. *Dental Tubules.*—It was certain that the dental tubules were frequently infected from the pulp cavity. While probably any organisms in the mouth could infect pulp, those most usually associated with dental infections seemed to be comparatively few in number—namely, diphtheroids, cocci, and spirochaetes.

3. *Cementum.*—Infection of the cementum was frequent, and three routes could be distinguished—(a) from the pulp cavity and tubules; (b) through the periodontal membrane; and (c) from surface caries. There was evidence that cementum could be infected from a living but infected periodontal membrane. No section showing actual invasion of the cementum by bacteria from the dental tubules had been obtained, but one showed its near approach. Another showed surface caries of the cementum, with a thick growth of bacteria on the surrounding cement surface. This layer of bacteria was always found on the denuded roots in pyorrhoeal pockets, and explained the difficulty of treating pyorrhoea without thorough cleaning of all such denuded roots.

4. *Gums.*—In chronic cases bacteria were invariably present in the gums; the most frequent were diphtheroids, streptococci, and staphylococci. In one instance a heavy infection with a sporing bacillus was observed; the gum showed chronic fibrous thickening; there was no pocketing by destruction of the alveolar dental membrane but only by swelling of the gum, nor was there any ulceration; apparently the bacteria gained entrance through a merely inflamed surface. A section from the gum flap overlying an erupting wisdom tooth in a man of 48 showed dental sepsis at its earliest moment—namely, just as the gum uncovered the tooth and left the crypts open to infection. Here a pure diphtheroid infection was to be seen running up the lymphatics.