was first Lieutenant of the *Malabar* at the date of the duel, but had succeeded to the command by the time of the trial, was also indicted, having been Passmore's second. Lieut. Irwin, who seconded Key, does not seem to have been put on trial. The evidence consisted chiefly of the statement of Lieut. Searight, which was to the effect that Passmore had been the aggressor. The jury acquitted both prisoners. Key, who was stated to be very ill at the time of the trial, died a few days later on 22nd January.

Asst.-Surgeon John Porter Malcolmson, Bombay, was indicted in the Bombay Supreme Court, on 25th September 1835, before the Chief Justice, Sir Herbert Compton, and Sir John Awdry, Puisne Judge, for the murder of Captain Alexander Urquhart, at Poona, on 18th July, by shooting him in a duel. Mrs. Malcolmson appears to have been the cause of the duel. Malcolmson was wounded in the hand, got tetanus, and recovered. The two seconds, Major Foster Stalker and Lieutenant James McDonnell, and Asst.-Surgeon James Don, were also put on trial. The prisoners offered no defence. When Awdry began to deliver the charge to the jury, the foreman stated that the jury had made up their minds to acquit all the prisoners. In accepting this verdict, Sir John Awdry remarked that Don, who had not been present at the duel, but had been summoned after it was over, should never have been put on his trial. Don rose to be a Member of the Bombay Medical Board twenty years later on 26th January 1855.(17)

No case appears to be on record in which any member of the I. M. S. has been brought for trial before a Court-martial for cowardice. And the three cases quoted above, seem to be the only ones in which a member of the service has been cashiered for fraud.

Only one case of heinous civil crime committed by an officer of the I. M. S. appears in the records. On 19th June 1821, a Madras Surgeon of over twenty years' service was sentenced to transportation for fourteen years for forgery. He died in New South Wales before the end of the year.(18)

Madras G. O. of 23rd October 1840, notifies that an Asst. Apothecary of the Madras Service had been sentenced to death for the murder of another Warrant Officer of the same rank. The Commander-in-Chief commuted the sentence, on account of extenuating circumstances, to penal servitude for life.

It is curious that these two last cases, Blackwall's treason, Brown's case of poisoning,(19) Martin's trial for murder, and the execution of La Forge, all occurred in Madras.

REFERENCES.

(1) John Caillaud, (not Calliaud), arrived in India in 1753, was appointed in 1759 to command the E. I. Co.'s troops in Madras, and to the Command of the Bengal Army on 25th February 1760; was in command during the war in Bihar in 1760; succeeded as Commander-in-Chief by Major John Carnac on 31st Dec. 1760. Brigadier General, 1763; retired 1775; died 1810.

(2) Orme's MSS, India, Vol. XIII, pp. 3444-3447.

(3) Word *if* omitted in copying MSS.

(4) The actual Proceedings of the Court are not given in the MSS.

(5) *Alexander Boswell*; a medical officer, mentioned also in Orme's *History*, Vol. II, Book X, p. 437, was for many years physician to the Nawab of the Carnatic. Retired 12th February 1776.

(6) Tellicherri Records, Vol. VII, 27th March 1787.

(7) M.P.L. No. 333 of 14th Feb. 1794, Mily. Cons., Vol. CLXXXIII, pp. 764—768.
————No. 380 of 18th Feb. 1794, *Ibid*, pp. 796—834.
————No. 424 of 21st Feb. 1794, Mily. G. O., Vol. X, p. 81.
————No. 715 of 26th March 1794, Mily. G. O., Vol. X, p. 100.

(8) M. P. L., No. 5094 of 26th Dec. 1793, Mily. Miscell., Vol. XXXIX, pp. 428—431; No. 627 of 18th March 1794; Mily. Cons., Vol. CLXXXIV, pp. 1151—1218; No. 1170 (144) of 15th—31st July 1795, Mily. Miscell., Vol. XLVII, pp. 93—190; No. 1411 of 4th Sept. 1795; Mily. Cons., Vol. CXCVII, pp. 2724—2786.

(9) C. P. L., 4th, 19th, and 20th June 1777, pp. 282, 288, 295; and 5th, 7th, and 27th Dec. 1777, pp. 346, 347, 360; O. C., 8th Jan. 1778, No. 26.

(10) Another Surgeon who entered the Madras Medical Service in the same manner as Martin was John Carere, who deserted from Muhammad Yusuf at Madura, along with Captain Riquet, on 26th Feb. 1764, and was appointed Asst.-Surgeon in Madras Military Consultations of 13th Aug. 1764. John Castarede, who entered the Madras Medical Service on 2nd June 1790, had formerly been a Surgeon in Tipu Sultan's service. (M. P. L. No. 1767 of 21st July 1789; Madras Mily. Cons., Vol. CXXX, pp. 2034—2073).

(11) M. P. L., No. 1652 of 9th Aug. 1786; Vizagapatam Cons., Vol. IX, p. 182.

(12) M. P. L., No. 1961 of 31st Oct. 1787; Vizagapatam Cons., Vol. X, pp. 306—320.

(13) The original memorial must have been in French, as that in the Public Proceedings is certified by the French translator to be a correct translation.

(14) Orme MSS., various, Vol. LXXVI, p. 145.

(15) Orme MSS., various, Vol. LXXVIII, pp. 59, 60.

(16) Orme MSS., various, Vol. LXXVI, pp. 244, 245.

(17) This case is reported at considerable length in the *Asiatic Journal* for April 1836, Asiatic Intelligence, p. 268.

(18) This case also is reported in the *Asiatic Journal* for Jan. to June 1822, p. 491; and that of the Asst. Apothecary in the same journal for March 1841, p. 235.

(19) For Blackwall and Brown *see* Chapter VII, Early History, Madras and the Coast.

RECENT RESEARCH ON CHOLERA IN INDIA.*

BY E. D. W. GREIG, M.D, D. SC.,

MAJOR, I.M.S.,

On special duty for the Cholera Enquiry of the Indian Research Fund Association.

IN my investigations on the etiology of enteric fever in India I dealt with the relation of the B. typhosus to the biliary passages and its important bearing on the "carrier" question.

That the cholera vibrio is limited to the alimentary tract was the generally accepted view in regard to its distribution in the tissues of man when I commenced my research on cholera this year; thus Rogers, 1911, states, "The absence of infection of the gall-bladder and the bile ducts by the comma bacillus places the disease in quite a different position from that of typhoid in this respect."

* A summary of 4 papers on Cholera read at the All-India Sanitary Conference held at Madras in November, 1912.

OBSERVATIONS ON THE OCCURRENCE OF THE CHOLERA VIBRIO IN THE GALL-BLADDER.

The "carrier" question is of vital significance in the prevention of cholera, and, therefore, the elucidation of the exact mode of production of the cholera "carrier" is a problem of fundamental importance.

At the Jagannath festival at Puri in 1912 I examined bacteriologically the bile in 271 fatal cases of cholera and cultivated the cholera vibrio from the bile in 80 cases. Distinct pathological changes, both macroscopic and microscopic, were found in the gall-bladder in 12 of the 80 cases. Examination of serial sections of the gall-bladder showed the comma bacillus not only on the surface of the mucous membrane but, also, deeper in the submucous tissue in some of the specimens.

BEARING OF THESE OBSERVATIONS ON THE "CARRIER" QUESTION IN CHOLERA.

The conditions met with in the alimentary canal of man are not favourable for a prolonged intra-corporeal existence of the comma bacillus after the acute attack of cholera is recovered from. Because the delicate cholera vibrio is liable to be crowded out in the struggle for existence by intestinal and putrefactive organisms. On the other hand, if it gains access to the gall-bladder the comma bacillus finds there ideal conditions for a prolonged life, namely, absence of other competitors, and a suitable alkaline medium, indeed Ottolenghi has recently recommended bile as a selective medium for enriching the cholera organism. My researches have demonstrated that the cholera vibrio can enter and live in the bile, and thus afford an explanation of the mode of production of the "Chronic Carrier." An apparently healthy person, whether convalescent or "contact" harbouring the cholera vibrio in the gall-bladder would be obviously a great source of danger to the community, because he is a reservoir of the virus and liable to produce fresh cases and epidemics of cholera wherever he may go.

BEARING OF THESE OBSERVATIONS ON THE CAUSATION OF PATHOLOGICAL LESIONS AND COMPLICATIONS IN CHOLERA.

In some cases in which a pure culture of the cholera vibrio was present in the bile pathological changes were observed in the gall-bladder wall itself giving rise to a cholecystitis; and, further, the toxine liberated by the vibrios as they die will pass from the gall-bladder into the system, and some of serious complications of cholera, *e.g.*, uræmia and pneumonia, cardiac failure, etc., may be brought about by the action of the cholera poison from this source on the kidneys, lungs, etc. The administration of permanganate by the mouth would not affect the toxine in the gall-bladder, and thus certain of the fatal cases which occur in spite of its use may be accounted for by this observation. In a case of cholera, in which the acute stage was recovered from and death occurred after 13 days from pneumonia and nephritis, I found comma bacilli in large numbers in sections of the consolidated areas of the lung. The bacilli were lying free chiefly between the cells in the cellular exudate in the alveolus. The gall-bladder of this case contained a pure culture of the cholera vibrio and the wall showed marked pathological changes. This observation was interesting and suggestive: the cholera vibrio must have gained access, also, to the blood stream in order to reach the lungs.

A great advance in the prevention of cholera would be made if a means of destroying the organism in the tissues of man could be found.

PLACES OF PILGRIMAGE AS CENTRES FROM WHICH CHOLERA IS EXPORTED THROUGHOUT INDIA.

At the great festival of Jagannath at Puri in 1912 I made a series of observations which included

BACTERIOLOGICAL EXAMINATION OF PILGRIMS CONVALESCENT FROM CHOLERA.

At Puri I examined a number of patients just before they left the Cholera Hospital to embark on the train, and I found that about 36% were excreting the cholera organism in their stools. The full significance of this becomes apparent when it is remembered that these convalescent pilgrims embark on the train and are carried in a short time to their homes, which, I ascertained, were scattered all over India. Convalescent cholera "carriers" are in a position to cause epidemics of cholera in these various localities. More than a lac and a half of people arrived by train from all parts of India at Puri in July 1912, and about the same number left. These facts give some idea of the extent of the exportation of cholera virus from this pilgrim centre. Further, in view of the observation on the occurrence of the cholera vibrio in bile, it is probable that a number of these convalescents will continue to act as reservoirs of the virus for long periods and they cannot be detected except by careful bacteriological examination. In two convalescents I found the cholera vibrio being excreted in the stools 30 days and 44 days after the acute attack; it is difficult to say when they will become bacilli free.

In addition to the cholera convalescents I made a

BACTERIOLOGICAL EXAMINATION OF CHOLERA "CONTACTS" AMONGST PILGRIMS.

When a population is infected by the cholera vibrio, as at Puri in 1912, a number of

individuals (non-immunes) develop symptoms of cholera and many die, but in addition to these and of far greater importance from the preventive point of view, a certain number of persons (immunes) harbour and excrete the cholera vibrio in their stools without showing any apparent signs of disease. The latter group ("contacts") increase enormously the practical difficulties in dealing with the problem of the prevention of cholera.

Examination of 27 persons at Puri, who had been in close contact with cholera cases, showed that 6 were executing the cholera vibrio in their stools and were apparently quite healthy at the time. Like the cholera convalescents these healthy "contact" (cholera immunes) are extremely important agents in the transportation of the cholera organism to different parts of India.

The discharge of the cholera vibrio in the stools of convalescents is very intermittent, as is the case in enteric convalescents, and this fact introduces a serious difficulty, as regards the detection of the "carrier" and the determination of his freedom from bacilli.

It is interesting to note that convalescents excreting the cholera vibrio in their stools for a prolonged period showed cholera agglutinins in the blood, and, therefore, the Widal reaction may be a means of detecting them in the first instance. Convalescent cases which did not continue to excrete the cholera vibrio did not show agglutinins in the blood.

A Cholera Convalescent Causes Epidemic of Cholera in a Jail.

It might be urged that the "carrier" was incapable of producing an epidemic of cholera. I had an opportunity of making very careful observations on this point in connection with the investigation of an epidemic of cholera in Puri Jail. The epidemic was brought about in the following manner:—A patient, who had been attacked by cholera on the 6th July 1912, was discharged from Puri Cholera Hospital, he wandered about until July 23rd, 1912, when he was arrested and sent to jail. A few days after his entrance to the jail cases of cholera commenced to occur: there were 17 cases and 5 deaths in all. The strength of the jail was 222 at the time. On July 28th, 1912, a bacteriological examination of his stools was made by me, and he was found to be excreting the cholera vibrio in large numbers on that day, that is, more than three weeks after the attack. The first case occurred in the under trial ward in which the cholera convalescent was located. The man was isolated: but bearing in mind the fact that, in addition to the actual cases of cholera, there must, also, have been an uncertain number of healthy individuals excreting the cholera vibrio (immunes) resulting from the importation of the virus; consequently, a very careful disinfection of all fresh night-soil had to be adopted also to arrest the epidemic: these means were quite successful. Without a bacteriological examination it would have been impossible to have detected the source of this epidemic and to control it. Further, this observation proves that the "carrier" is capable of causing an epidemic of cholera, and demonstrates the danger to the community of infected pilgrims returning from these festivals.

Flies as Transmitting Agents in Cholera.

The organism of cholera passes from the infected human host, who may be either actually ill or passively acting as a reservoir of the virus, by various channels, *e.g.*, direct contact, water, flies, etc., to uninfected individuals. During the cholera epedemic at Puri in July and August 1912, flies were extremely abundant amounting almost to a plague; an interesting point in this relation was that the appearance and disappearance of the flies synchronised with the arrival and departure of the great mass of pilgrims: consequently, temporary breeding places must have existed at Puri. Bacteriological examination of flies caught in the neighbourhood of collections of cholera cases at Puri showed that the cholera vibrio was present on the external appendages, and also in the contents of the alimentary tract of the flies demonstrating that the fly was a channel by which the virus was being conveyed from the infected to uninfected individuals at Puri. Water I think could be excluded.

Observations on Disinfection in Cholera.

Healthy "carriers" and flies were the main channels by which the infection of cholera was transmitted to the uninfected at Puri. I give here a brief account of the procedure adopted by the local authorities, on my suggestion, for dealing with, in the first place, the epidemic in the jail, and, nextly, the more difficult problem of combating the disease in the town itself which was severely infected.

As already described by me in this paper the epidemic of cholera in the jail was caused by a convalescent cholera "carrier." Having regard to the channels by which the infection was spread, the object to be attained, in this particular case, was the disinfection of night-soil at the earliest possible moment before flies could gain access to it and, possibly, become infected with the cholera vibrio. To have determined the particular individuals excreting the cholera vibrio it would have been necessary to have examined bacteriologically the whole jail population; to do this thoroughly would have occasioned very considerable delay, accordingly it was decided to disinfect the fresh night-soil of

all the inmates of the jail. Each person in the jail passed his stool into a receptacle filled with a solution of cyllin of suitable strength, in this way the stool was rapidly disinfected and the access of flies during the process of disinfection was prevented, the fæces being submerged in the solution. Cases of cholera were occurring in the jail, but 4 days after (13th August), the commencement of the systematic cyllination of fresh night-soil (9th August), the outbreak ceased.

Encouraged by this observation the same plan of disinfection of fresh night-soil was extended to the town of Puri itself, but for the purpose a solution of fresh chlorinated lime was used, because (1) it is cheap, (2) it is a very good bactericide, (3) its pungent odour effectually keeps the flies away. The difficulties in carrying out the systematic disinfection of fresh night-soil in the town were very great owing to the extremely defective state of the private latrines and the absence of proper access to them. But in spite of these difficulties it was followed by a marked drop in the number of attacks and deaths from cholera. Thus on the 13th August, 3 days before systematic chlorination was commenced, there were 39 deaths from cholera. On 20th August, 4 days after it was begun, the deaths were 18. On 24th August, 4 deaths; and by the end of August the epidemic ceased entirely.

A Mirror of Hospital Practice.

PYORRHŒA ALVEOLARIS IN SYLHET JAIL.

BY L. BODLEY SCOTT, B.A., M.D. (CAM.), D P.H. (OX.), CAPT., I.M.S.

PYORRHŒA ALVEOLARIS is well known to be an extremely common complaint among the natives of Bengal and Assam and probably of other parts of India also.

It appears to take the place in this country of dental caries in England. Caries is comparatively rarely seen in Bengal but Pyorrhœa is universal and is even more effective in causing loss of teeth in those of advanced age.

It is the exception to meet a man past middle life with healthy gums, and old men almost invariably have the disease in an advanced stage with recession of gums and loosening or loss of teeth. Even below the age of twenty the disease is extremely common.

I think it may be said that beyond these generalities very little is known about the disease in Bengal. Its exact distribution and prevalence, its causes, effects on general health, relation to other diseases and amenity to treatment all need investigation.

This paper will throw no light on the most important question of all, namely, the cause of Pyorrhœa. So far as I know we are entirely ignorant of this. Much valuable work has been done in Europe and America on its pathology, bacteriology, etc., but as to its cause, or the reason why one person gets the disease and another does not, we are still, I believe, quite in the dark.

As regards the other questions much useful information could be collected by observations on prisoners in jail. I started to record observations in Sylhet jail about a year ago, and this paper gives the results of a year's records.

If similar statistics of all the jails in these provinces could be collected, I think our knowledge of Pyorrhœa in Bengal and Assam could be made very much more definite than it is.

The ideas prevalent among jail medical officers regarding the relations between Pyorrhœa and Dysentery, Pyorrhœa and Scurvy and other diseases such as beri-beri, epidemic dropsy, etc., are extremely vague and divergent. One man will say that jail dysentery depends entirely on the condition of prisoners' gums. Another will deny that there is any connection.

The condition used to be, and still is, often erroneously talked of as "spongy gums," and almost every jail disease has at one time or another been put down to "spongy gums." A great deal of loose talk and many dogmatic theories have been the result of this confusion of terms and the supposed connection between Pyorrhœa and Scurvy.

As a matter of fact the gums in Pyorrhœa are never spongy and bear no resemblance to the fungoid hæmorrhagic gums of Scurvy.

All this confusion could soon be cleared up by the collection of a few years' statistics in the jails, and the connection between Pyorrhœa and Dysentery, Scurvy, and other complaints definitely settled.

The first question which arises is whether the disease so common in Bengal is the same as the Pyorrhoea Alveolaris of European writers. There seems to be no reason to think otherwise. Its symptoms on the whole correspond very closely with the English descriptions. The following are the characteristics of the disease as seen here.

CHARACTERS OF THE DISEASE.

In the earliest cases the gums on inspection often appear normal, or they may have a rather deeper colour than natural, a purplish hue instead of a light pink. They are never swollen, but the margins may be slightly thickened and do not lie so flatly in contact with the teeth as they should do. On pressure a little blood wells up between the gums and teeth, but no pus. In this stage in fact the disease is not a Pyorrhœa.