Thesis,
for the Degree of Doctor of Medicine.

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(1). Typhoid Fever, Its nature, Mode of Spreading, and Prevention.
by William Budd M.D., F.R.S. London. 1873.

(2). Micro-organisms and Disease. E. Klein London 1886 Page 121.

(3). British Medical Journal Vol I. 1882. Page 421.

(4) Manual of Pathology. Joseph Coats London 1883 Page 255.

(5) British Medical Journal Vol II. 1884 Page 579.

(6) A Treatise on Continued Fevers London 1873.

Notes of Cases occurring in a Country Practice.

The question of the Etiology of Enteric, or Typhoid Fever, of late years has been reduced to comparatively narrow limits. Budd[1] proved that it was essentially contagious in its nature, and although the specific bacillus discovered by Klebs[2], and Eberth[3], and confirmed by Coats[4], Watson Cheyne[5], and others, may not be constant, the disease is universally held to be a member of the Zymotic Group. At the same time the question whether the disease can originate only from direct contagion is not quite settled, and the following series of cases would appear to favour its occasional spontaneous origin; or that it may be "dirt-produced," or "Pythogenic," as Murchison[6] believed.

Case I. In the autumn of 1883 Mrs F——, aged 30, became highly fevered, had headache and vomiting, and an eruption of rose-coloured spots

spots, but no diarrhoea. This was noted at the time as an obscure case of Typhoid.

Case II. Three weeks after the onset of Mrs F——'s illness, her husband, aged 35, had a well marked, and severe attack of Typhoid, accompanied by profuse Melaena.

Case III. During the convalescence of Case II his sister who had acted as nurse to the above patients was seized with well marked Typhoid Fever.

Case IV. Another sister living 2 miles off in the country and who had helped to wash some clothes, to be mentioned subsequently, at her brothers house, about 10 days before, developed the same disease.

Cases V and VI. Two children of a neighbour, living next door aged 4 and 6 years respectively and who had been in the habit of going into Mrs F——'s house, also had typical attacks of Typhoid Fever, their illness being contemporaneous with that of Case II.

Case VII.

Case VII. A young girl, living in the room above F——'s house, also suffered in the same way.

All the patients made a good recovery.

Cases IV and VII were not attended by me, but I have good authority for stating that the illness they had was certainly Typhoid.

There were at the time no other Typhoid Fever cases in the neighbourhood, there was no history of milk infection, and no bad drains, as there were no drains of any sort in the house; and if there had not been the following history to account for the outbreak, it would not have differed from many others of unexplained origin. However for four months the F——'s house which was of one room only, had been shut up, the family living during that time at a farm about a mile away, and they had only returned to their own house eight days before Case I sickened.

For about ten months, i.e., six months before

before they left their own house and shut it up, their child aged 16 months, had been troubled with Chronic Diarrhoea evidently due to improper feeding, and the discharges were very offensive. Mrs J— when shutting up her house had left innumerable napkins, and clothes soiled with these discharges, lying about under the bed, and in a press, and these were neither removed, nor washed until the day, which was during the illness of Case II, that the two sisters (Cases III and IV) washed them. Until this was done there was a heavy disagreeable smell in the house which I had remarked and enquired about several times, and the cause of which I was not told, until it had been removed by the washing.

The question naturally arises; was it Typhoid Fever the child had in the first instance? The evidence seems to be against this; as for example the Chronic nature of the childs illness, and the fact

fact that no one took ill during the six months before they closed their house, and no one was affected at the farm, they stayed at, the child being in the same condition all the time. It was only after their return to the house, where the discharges had been left to decompose that the outbreak took place, and I think this points to some virulence in the dirty clothes which they did not possess four months previously. All the cases seem to have taken it from the same cause, Cases II, V, VI, and VII, being later, probably from being less in the house than Case I; and of the two sisters certainly Case IV was affected by washing the clothes in question, as that was the only occasion, on which she came to the house. There was moreover no further spread of the disease.

There are many other instances on record the explanation of which is difficult, unless the occasional "de novo" origin

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(1) British Medical Journal Nov 6th 1880. Page 740.

(2) British Medical Journal Jan 1875. Quoted from "A System of Practical Medicine by American Authors" London 1885.

of Typhoid Fever is admitted, the outbreak in Norfolk Island[1] being one of many examples.

Although, in accordance with the teaching of the present day, I would incline to believe in the "continuous propogation theory" of Budd, yet these instances tend to cast doubts on the absolute truth of this doctrine. According to Burdon Sanderson[2] "the influence of environment on organisms such as bacteria is so great, that it seems as if it were paramount." and Pasteurs methods of attenuating the virus of Chicken cholera, and of Hydrophobia are also in favour of this view. It seems a fair question therefore to ask; whether, given the necessary conditions of developement and concentration is it not possible for the ordinary bacteria of decomposition, to become the exciting cause of Typhoid Fever? or, whether attenuated forms of the Specific bacilli of Typhoid Fever

(1) A Treatise on the Theory and Practice of Medicine.
by John Syer Bristowe M.D London 1880 Page 201.

(2) British Medical Journal Jan 26. 1884 Page 174. Vol I
and, Micro-organisms and Disease. E. Klein M.D F.R.S. Page 73.

(3) British Medical Journal Sep 5. 1885 Page 442. Vol. II

(4) British Medical Journal May 15. 1886 Page 924. Vol I.

(5) British Medical Journal Jan 29. 1887 Page 203 Vol. I.

fever may not be present in the atmosphere at least in certain districts, at certain times, which meeting with a suitable nidus such as these filthy clothes ~~may~~ become powerful enough to cause the disease? It is probable also that in these cases the virus gained admission to the system by the lungs, and not at the surface of the alimentary mucous membrane which Dr. Bristowe[1] considers to be probably the only mode of entrance.

Klebs and Eberth[2] also were the first to describe the presence of Micrococci in Pneumonia, and in confirmation of the view that Pneumonia is a specific infective disease, there have been many Epidemics recorded, among others by Lloyd Roberts,[3] Bruce[4] and Foulis[5], and to these may be added the following instance of apparent contagion which came under my notice.

The family in which this outbreak of pneumonia

pneumonia occurred consisted of the Father and a grandson.
Mother, ~~and four sons~~ & The sons all worked in Ironstone Pits, and the father was a broken down Miner. They lived in a Miners Row in a house consisting of two rooms.

Case I. Peter M—— aged 21, on the 9th Nov. 1885 had rigors, acute pain in his right side and pyrexia. On the 11th he was expectorating rusty sputum, and had all the signs of right basal pneumonia. His temperature that day was 103°. The disease ran a favourable course and he was convalescent on the 20th Nov.

Case II. Thomas M—— aged 23, on the 15th Nov. five days after the onset of his brother Peters' illness, began to have rigors, and developed left basal pneumonia with rusty expectoration and pain. His attack however was not so severe as the preceding and he was convalescent on the 28th Nov.

Case III. William M—— aged 17, on the 4th Dec. nineteen

nineteen days after Case II, took rigors, vomiting, pain in right side, followed by rusty spit, and all the physical signs of right basal pneumonia. He had slight diarrhoea, but no typhoid symptoms. He made a slow recovery, but on Jan^{y} 10th 1886, his right lung was normal.

Case IV. Robert M—— aged 14, on Dec^{r} 14th had shiverings, pain in his head, and right side, and complained of feeling sore all over. On Dec^{r} 19th his pulse was 120 respirations 36, and temperature 103°6. Both bases were slightly dull and full of râles, and he had bloody expectoration. On the 21st his pulse was 88, respirations 28, and temperature 102°. Expectoration was rusty and left base very dull. From this condition he made a good recovery and was well on the 28th Dec^{r}.

Case V. M—— aged 60, the father of the preceding on Jan^{y} 2nd 1886 took an attack of Acute Bronchitis. He had long been troubled with Chronic Bronchitis and

and Emphysema in consequence of which he had been unable to work for several years. He very soon began to suffer greatly from dyspnoea, his lips becoming blue, and his legs oedematous. His heart became very weak and slightly irregular, and his urine was albumenous. On Jan.y 10th both bases were dull behind from hypostatic congestion, his sputum was frothy, and the dyspnoea was very great. He got gradually worse and died on the 2nd Feb.y. 1886.

The only persons living in the house who were not attacked were the Mother, and grandson.

The previous history of the four sons was also very peculiar. They had all suffered from one or two attacks of Pneumonia before, and one of them Peter was said to have had three similar attacks previous to the one in question. Also since the occurrence of the illness reported I have again attended Peter and William for slight but perfectly typical

typical attacks of pneumonia.

During the time they have been having these illnesses, which has extended over many years, they have occupied the same house, and it was a question whether there was some cause in the house itself, or whether there was some peculiar idiosyncrasy in the family. The house was the same as those on each side of it. None of the patients had been exposed to any chill, and the question of these having been cases of Typhoid Fever may be excluded as they were typical cases of Pneumonia, with the exception of Case V which was more bronchitic than pneumonic in character. I think they were probably septic in nature, and they seem to prove that some forms of pneumonia are contagious.

The following cases exemplify the predisposition or idiosyncrasy to suffer seriously from Scarlet Fever which

which affects certain families. When they occurred there were a great many cases of Scarlet Fever in the immediate neighbourhood, but all of them with the exception of these three were of a mild type. They also point to a very excitable condition of the nervous system.

Case I. Rebecca L—— aged 3 years. In the afternoon of the 27th Nov: 1886 she became hot and feverish. She had been apparently quite well previous to this. At 10.30 p. m. she took a violent convulsion, and became pale and pulseless. There was no rash. She died at 11.30 p.m the duration of her illness being less than 12 hours. The diagnosis made was Scarlet Fever, from the fact of an epidemic being in the neighbourhood.

Case II. Adam L—— aged 5 years, a brother of the above, two days afterwards viz:- on the 29th Nov: became highly fevered. His throat became congested and red, and the lymphatic glands at the angles of the jaw

jaw became swollen and tender. There was no rash on the skin, but there was a red punctuate rash on the fauces, and soft palate. Next morning Nov. 30th there was no rash, but the skin was hot and dry and the child had a strawberry tongue and was very drowsy. In the evening he was delirious, flushed and excited. His tongue was dry and cracked, his pulse quick and feeble, and he died next morning Dec. 1st about 8 a.m. having been ill rather more than two days.

Case III. Janet L—— aged 13 months, a sister of the above on the morning of Dec. 1st the day her brother died, was noticed to have a slight rash on her neck. She had no sore throat and was not fevered and was taking the breast as usual. In the evening there was a typical scarlet rash all over her body. On Dec. 2nd the rash was still out and the child appeared to be doing well like the other mild cases in the place. That night at 11 p.m. she took

(1). Glasgow Medical Journal. March 1888.

took three violent convulsions, which followed each other very rapidly and died, the duration of her illness being about 2 days.

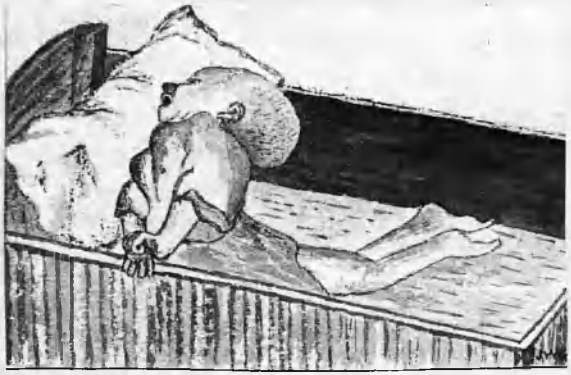
These three children constituted the whole family. The father and Mother both had sore throats at the time, and some difficulty in swallowing, but developed no rash and soon recovered.

As the district I was practising in at Cumnock was not far from Galston, where Dr Frew[1] had so many cases of Cerebro-Spinal Fever, it is of interest to note that Tubercular Meningitis among young children was very common, although I did not diagnose any cases of Cerebro-spinal fever.

One case of which I shall give the history was altogether anomalous, and I have not been able to find any case reported that was like it. If such a thing were possible it might be called "Chronic simple cerebro-spinal fever". It certainly may with

with truth be called Chronic Cerebro-Spinal Meningitis.

Case. The patient was a female infant aged 2 months. The illness began on the 4th Jan 1857. The onset was gradual commencing with slight bronchitis, pyrexia, vomiting, constipation fretfulness, and fits of screaming. Shortly after this slight convulsive attacks came on, the eyes being turned down, and the hands clenched. The head was rolled about and burrowed into the pillow, and a diagnosis of Tubercular Meningitis was made.
In February the head gradually became retracted and fulness of the anterior fontanelle became apparent. From this date the head enlarged and its retraction and the rigidity of the extensor muscles of the neck became more marked. This rigidity extended downwards, involving the muscles of the back, so that in March the condition was that of marked opisthotonus. This condition was persistent even during sleep. On the 22nd April the following



Sketch made from a photograph taken on April 22nd showing the position of the child lying in her cradle. If the child was taking the Mothers right breast, with its head directed to the left shoulder of the Mother, the heels of the child also pointed in the same direction.

following note was made :- Age of child 6 months. No change in its condition since March. The child lies very quiet, occasionally giving a short monotonous cry, opening its mouth very widely, the angles of the mouth being retracted (risus sardonicus). The decubitus is lateral, the head being thrown far back the occiput almost touching the upper dorsal vertebrae. The muscles of the neck, back, and the extensors of the thighs are all rigid. The spinal column is arched backwards, the thighs drawn back, and the knees slightly flexed, continuing the opisthotonic curve. The feet are extended and the toes flexed. The arms are abducted and brought forward, the forearms semiflexed, the hands extended at the wrists, the thumbs flexed across the palms, and the fingers flexed over the thumbs. The pupils are moderately dilated. There is no strabismus or ptosis. Pulse and respiration are apparently normal. The

condition

condition of Opisthotonos is never relaxed either when asleep or when at the breast.

Note on May 5th – The child's head has been getting much larger, the measurements being as follows:- Circumference round occiput and brow, 18½ inches. From one auditory meatus to the other over the vertex, 11½ inches. From the occipital protuberance to the root of the nose, 12 inches. The bones of the cranium are widely separated. The fontanelles and sutures are very tense and pulsate. There have been no convulsions since the rigidity came on in February. It has been a condition of tonic spasm all the time.

18th May. Condition unchanged except that a day or two ago, the spasm was relaxed for a few minutes and the child lay straight out in its cradle, but after this it came on again as before. The child died on the 19th May, the duration of its illness being nearly five months. No post mortem examination was made.

"Family

(1) "A Manual of Diseases of the Nervous System."
by W. R. Gowers M.D. F.R.S. Vol II Page 315-
London 1888.

(2) Gowers 'Op. cit.,' ~~Page 315~~. Vol I. Page 192.

"Family history". The parents are healthy and have had six of a family, all healthy, except one child who died aged 11 months of Tubercular Meningitis. There was no history of Syphilis.

Gowers[1] speaking of chronic infantile meningitis says "After death the signs of chronic meningitis are always found chiefly in the posterior fossa of the base; generally lymph glues together the Medulla and cerebellum, and then there is chronic hydrocephalus, the openings from the fourth ventricle being closed by the lymph. There may also be slight inflammation in the membranes of the Spinal cord, and this as I have seen without special symptoms of the Spinal meningitis." In the case recorded above the symptoms of Spinal meningitis were well marked, agreeing with Gowers'[2] statement in reference to Spinal meningitis, "that the muscular Spasm ..... may be so ~~general~~ general and severe as to cause opisthotonos resembling that of tetanus."

The

The following case of what I considered to be acute meningitis presents a marked contrast to the foregoing in point of duration.

Case. William M— aged 34. Miner. I was sent for on the 9th May 1887 at 7 p.m. and found him in bed, with his shirt and trousers on, having recently lain down. He was lying as if asleep; his face was pale, and his corrugator supercilii contracted. When spoken to he answered perfectly sensibly, saying that he had great pain in his forehead, and that he wished to be left alone. He had great intolerance of light. His pupils were contracted, but there was nothing abnormal about them or the eyeballs. His pulse was 64 and irregular. His breathing was normal and his temperature 99°. His heart was rather irregular in action but I could find nothing abnormal in the lungs or abdomen. The history was that on Friday the 6th May, he was at work and on Saturday morning

(1) Gowers 'Op. cit.,' Vol II Page 86.

morning (the 7th) he awakened with a headache. This was better on the 8th, but became worse on the 9th the day I was called in. He had vomited some yellow bilious matter a few hours before I saw him, but there was no persistent vomiting. I gave a guarded prognosis, and left instructions that they were to send for me if he became any worse. I was sent for shortly before 11 p.m., the message being that the man was taking fits. I found him taking violent convulsions, which were "hysteroid"(?) in nature. He was waving his arms and legs about with a quick rotatory motion, like a windmill. His fingers were outspread, and his jaw clenched. This continued for about a couple of minutes, when he took a deep breath, which was followed by a lull of about half a minutes duration, after which the waving of the arms and legs would begin again. His face was flushed and he had very marked left external strabismus. His pupils

(1). Gowers 'Op. cit.', Vol II Page 308.

pupils were both widely dilated and insensible to light. His pulse was about 120. These convulsions began a little after 10 p.m., shortly before which hour he had been speaking quite sensibly. They continued with these momentary intermissions until 12·30 a.m. (10th May) when they ceased and his breathing became irregular (Cheyne-Stokes) and stertorous, and he died before 2 a.m. seven hours after I had first seen him.

His habits had been rather intemperate, but he had not taken much alcohol for a week before his illness began. He had no ear disease as far as I could discover. I was unable to obtain a post-mortem examination.

According to Gowers[1], in acute meningitis "death may occur within 48 hours after the onset of the symptoms." which tends to confirm the diagnosis given above.

As an instance of a convulsive attack brought

brought on by loss of blood, and alcohol, in a man who was not an epileptic, this case is of interest

Case. F. H. aged 36 a bricklayer had been drinking from 4 p.m until 11 p.m., when he staggered home bleeding profusely from a wound of his middle temporal artery, caused by a fall on his way home. I found him sitting with his head bent forward between his knees, the wound bleeding freely. He had lost a large quantity of blood. When dressing the wound he became pallid, snorted two or three times and fell forward. He was laid down on the floor, and immediately a violent convulsion came on. His mouth was tightly shut, and his body became rigid, and he had involuntary evacuation of faeces. There were convulsive movements of his eyes, followed by marked external Strabismus, there being eversion of the right eyeball, and rolling about of the left. The right pupil was larger than the left, both being about half dilated, and both were

Sensitive

sensitive to light. Respiration stopped for a moment, and his pulse was feeble and slow. A deep inspiration followed and he then lay in a state of coma. In about ten minutes he could be roused, and he answered questions rationally. Next morning he was fairly well and had slept during the night, and the day following he went to his work as usual.

In conclusion I wish to point out the great difficulty there is in obtaining post-mortem examinations in a country place. If they could be oftener obtained it would be a benefit to the practitioner, and also indirectly to the general public. For the present state of matters we are to a certain extent to to blame, as an examination in a private house means a great deal of trouble, and therefore is not pressed. If however P.M.'s were more common, the public would overcome their natural repugnance to them, & practitioners also from making them oftener, would have handier methods of working, and their performance in a private house would not be more difficult than in the post-mortem room of an Hospital.