caused by alimenta can be assumed with certainty only when the intestine suffers first—that is, when a so-called primary tuberculosis of the intestine is found. But such cases are extremely rare. Among many cases of tuberculosis examined after death I myself remember having seen primary tuberculosis of the intestine only twice. At the Charité Hospital in Berlin ten cases of primary tuberculosis of the intestine occurred in five years. Among 3,104 necropsies of tuberculous children Biedert observed only 16 cases of primary tuberculosis of the intestine. Hitherto nobody could decide with certainty in such a case whether the tuberculosis of the intestine was of human or of animal origin. Now we can diagnose them. All that is necessary is to cultivate in pure culture the tubercle bacilli found in tuberculous material, and to ascertain if they belong to bovine tuberculosis by inoculating cattle with them.

The question whether human and bovine tuberculosis are the same or different diseases has exercised the minds of scientific and clinical workers since 1901, when Koch made his famous declaration. Two Commissions were appointed to investigate this question. The work of the Imperial German Commission¹ was purely experimental, portions of tuberculous material being used from different parts of the body with special reference to the organs of children affected by tuberculosis. The report is unanimously in favour of Koch's contention that the bacilli from human and bovine sources have distinct morphological and bacteriological characters, which are sufficient for their easy differentiation.

This is of the highest importance, and confirms the observations made by me in the BRITISH MEDICAL JOURNAL of February, 1903. In addition, the German Commission has clearly proved that bovine tuberculosis is conveyed to children, and also particularly emphasizes the fact that tuberculosis is often primary through the intestines and mesenteric glands. From three cases of miliary tuberculosis in children they found the disease to have been introduced through the intestines, and in one case the infection was bovine from milk.

If we add to this the 7 cases of primary tuberculosis of the intestines or the mesenteric glands with those recognized as *Perlsucht* infection, we have 10 certain cases of primary infection of the digestive organs, of which 3 have to be considered as infection of *Perlsucht* or bovine tuberculosis by means of milk.

The result, then, of the work of the German Imperial Commission is to establish (1) that there is a distinct difference between the bacilli of human and bovine origin, and (2) that bovine tuberculosis is conveyed to children by means of infected milk, although they add not to any great extent. My own clinical and experimental work is in entire agreement with these results, excepting with regard to the amount of bovine tuberculosis conveyed to humans, which I consider is considerable. I believe that man is attacked by two distinct varieties of tubercle, one conveyed by infection from one person to another, the other by receiving into the body bovine bacilli from infected milk.

It is important to bear in mind that although primary intestinal tuberculosis is extremely rare, yet primary infection of the mesenteric glands is comparatively common; in children the tubercle bacilli pass readily through the walls of the small intestine and are conveyed by the lymphatics first to the nearest glands and then by direct extension throughout the abdomen, and in many cases through the diaphragm to the pleurae, lungs, and bronchial glands. This process of extension to the lungs I have met with in many cases, and numerous feeding experiments on animals have confirmed it.

During the last seven years I have had under my care 306 children suffering from what I have diagnosed as "*tabes mesenterica*," meaning by this tuberculosis of the mesenteric glands. A large proportion of these children recovered under careful treatment, whilst of the total number I had an opportunity of making a necropsy on 48. There can be no doubt that the abdominal glands and organs may be infected from the thorax downwards as well as from the intestine. In several cases it was obvious that the primary infection was through the lungs, and that the child by swallowing the sputum had infected the abdominal organs. On the other hand, the majority of my cases showed primary infection of the digestive tract with secondary lymphatic extension to the chest, and in some cases to the meninges. My cases of acute miliary tuberculosis all appeared to be of intestinal origin and bovine in origin.

The following are very brief notes of three cases of primary intestinal tuberculosis:

CASE I.

A child aged $2\frac{1}{2}$ was admitted to Mill Road Infirmary on June 13th, 1903, with the usual symptoms of intestinal tuberculosis. There was great emaciation, with enormous distension of the abdomen, with diarrhoea and offensive stools. The history of the child's feeding was carefully taken. It was fed on the breast till 14 months old, then on

cow's milk, which was usually bought at a milk shop or in the street. Enlarged mesenteric glands could be palpated through the thin abdominal wall, but the lungs showed no trace of disease. The usual treatment was adopted, careful feeding, and various nutrients, but he died on September 10th, 1903, having made little effort to improve.

At the necropsy there was great emaciation. The intestines showed extensive ulceration of the ileum and caecum with enormous enlargement of the mesenteric glands. Some of the lymphatic glands on the anterior surface of the vertebrae were much enlarged, but the viscera were not affected and the lungs appeared to be quite healthy. There were two recent perforations of the intestine near the caecum with local peritonitis.

CASE II.

A girl, aged 2, was admitted on June 29th, 1903, with all the symptoms of abdominal tuberculosis, constant diarrhoea, distension of the abdomen, and enlarged glands which could be easily palpated. The milk history was carefully taken. Child fed on the breast for ten months, then cow's milk which was never boiled. In spite of all treatment she died on August 22nd.

The necropsy revealed a condition almost exactly the same as Case I. There were fifteen to twenty typical tuberculous ulcers in the ileum with perforation of three of them and matting of the bowels. The mesenteric and abdominal glands were caseating and enlarged, whilst the lungs were quite healthy.

CASE III.

A child, aged $1\frac{1}{2}$, was admitted on March 13th, 1904, with cough, diarrhoea, and in a general hectic condition. She had a curious spasmodic crowing cough resembling whooping-cough and she was isolated on that account. She died in ten days.

At the necropsy extensive ulceration of the intestines, with old cheesy mesenteric glands, and a general lymphatic extension to all the abdominal viscera, then through the diaphragm to the pleurae, which were studded with grey tubercles, apparently quite recent. The bronchial glands were enlarged and compressing the trachea.

In conclusion, whilst I admit that primary intestinal tuberculosis is extremely rare in children, yet in my experience tuberculosis is very frequently conveyed to the digestive tract by infected milk, the bacilli readily passing through the intestinal wall into the nearest lymphatic glands, leaving no visible trace on the intestine.

This tuberculosis is not true human tuberculosis, but is bovine in origin, and may rapidly spread to all the viscera in the body, or may remain (as it most often does) localized in the mesenteric glands. Hence it is that, whilst tuberculous milk will not produce human tuberculosis, it nevertheless sets up bovine tuberculosis in the human body, more especially in children, and is even more virulent to children than the human variety. It is thus of the highest importance that tuberculosis amongst dairy cattle, especially those affected in the udder, should be recognized early and the cattle removed at once, and also that milk should be sterilized for all children under the age of 10 years.

REFERENCE.

¹ BRITISH MEDICAL JOURNAL, April 9th, 1904, p. 847.

SUPRARENALIN IN PULMONARY HAEMORRHAGE.

By DUNCAN MACDONALD, M.B., C.M.,
Oban, N.B.

IN view of the recent notices in the BRITISH MEDICAL JOURNAL I think the following case will prove interesting to your readers:

A.D., male, single, aged 28 years, had an acute attack of phthisis pulmonalis four years ago. There were distinct physical signs of the disease in the right apex and infracavicular region, with a temperature ranging from 99° F. morning to 103° F. evening. With open-air treatment and guaiacol carbonate he improved considerably, and was able to resume his work as a post-office clerk after about three months' treatment, being then transferred from a large city to a small seaside town post office.

The following year he had a sharp attack of pleurisy of the right side, for which he was tapped, and was able to resume work within three weeks from the date of the attack. With the exception of this slight interruption and his annual holiday he never lost a day's work from the time of his illness four years ago until January 16th, 1904. On that day on his way home from the office he called to see me, as he had observed a little blood in his sputum that morning. I then learned that about New Year's time, when the post office was exceptionally busy, he had done a good deal of extra work and heavy lifting for some weeks, had overheated himself, and had sweated and felt shivery in the intervals. His cough also became much worse; indeed, his cough had never left him in the morning since his first illness. I found well marked physical signs of cavity in the right infracavicular region, but he had no rise of temperature. I ordered him to bed and gave him a mixture containing iron, digitalis, sulphuric acid, and ergot. He progressed favourably till January 25th, when I allowed him to rise a little daily, but not to go out. On January 28th there was fresh bleeding, and the sputum contained more or less blood daily after that. I changed

his medicine on January 30th to extract *hydrastis canadensis* fluidum, tincture hamamelidis, and ext. glycyrrhizae, as the haemorrhage was a little severe, and gave him morphine $\frac{1}{4}$ gr. hypodermically at bedtime.

The bleeding became less again and things seemed to go on favourably until February 12th, when at 11 a.m. without any cough or warning he had a copious haemorrhage which nearly filled a bedpan. He was very collapsed after it. I gave him morphine $\frac{1}{4}$ gr. hypodermically then, and afterwards $\frac{1}{4}$ gr. night and morning. For two nights before this haemorrhage the temperature rose to 102° F. in the evening. On February 17th I resolved to try the effect of suprarenalin, looking to the results obtained by Graesser,¹ Bird,² and Hedley,³ and I tabulate the routine treatment.

Suprarenalin (Armour and Co.)

February 17th, 5 p.m.	3j (1 in 5,000).	
" 18th, 1 a.m.	do.	... Slight bleeding morning.
" " 9 "	do.	...
" " 1 p.m.	do.	...
" " 6 "	do.	...
" " 12 night	do.	...
" 19th, 6 a.m.	do.	... Slight bleeding 11 p.m.
" " 12 noon	do.	...
" " 6 p.m.	do.	...
" " 12 night	do.	...
" 20th, 6 a.m.	do.	...
" " 12 noon	24 minims (1 in 1,000).	...
" " 6 p.m.	do.	...
" " 12 night	do.	...
" 21st, 6 a.m.	24 drops (1 in 1,000).	... Slight bleeding.
" " 12 noon	do.	...
" " 2 p.m.	5 drops do.	...
" " 4 "	do.	...
" " 6 "	do.	...
" 22, 2.20 "	10 do.	... About teacupful of haemorrhage 1 p.m.
" " 4.20 "	do. do.	...
" " 6.20 "	do. do.	...
" 23rd, 4 a.m.	do. do.	... Fatal haemorrhage 4.40 a.m.

During this time the sputum was more or less coloured, but there was very little cough and the temperature remained normal. Morphine $\frac{1}{4}$ gr. was given hypodermically night and morning to allay the cough, with the exception of the mornings of February 20th and 22nd. The fatal haemorrhage came on suddenly without warning and ended the scene in a few minutes. It was very copious, about half a basinful coming up. The treatment of the case beyond this included perfect rest in bed in the recumbent position, cool liquid food, ice to suck, regulation of bowels by salines and enemata, and avoidance of all excitement and talking.

REMARKS.

Suprarenalin in this case evidently did no good so far as the ultimate issue of the case is concerned. Indeed, owing to its raising the blood pressure its use seemed rather contraindicated than otherwise; still, after reading the results obtained and looking to the hopelessness of the case I felt justified in giving it a trial. That suprarenalin increases the blood pressure was evident from the marked extra tension in the pulse after administration. Twice when given frequently in one day I noticed profuse perspiration following its use. It did not interfere with the patient's digestion in any way, and gave rise to no discomfort of any kind. He always expressed himself as "feeling nothing" from taking it. Any little effect it had seemed to be more marked when the morphine was given at the same time. As to whether its exhibition delayed the final haemorrhage terminating the case I cannot say.

In this case evidently a large branch of the pulmonary artery must have ruptured, probably as the result of aneurysmal dilatation into a phthisical cavity, and probably medication of any kind would have proved ineffectual, but in less severe cases I should be inclined to try the results of suprarenalin again. After the first slight haemorrhage in this case I gave a hypodermic of ergotine, but as it made the patient feel very sick and faint I did not repeat it.

REFERENCES.

- ¹ EPITOME of the BRITISH MEDICAL JOURNAL, December 14th, 1903, par. 75. ² BRITISH MEDICAL JOURNAL, January 23rd, 1904, p. 191. ³ BRITISH MEDICAL JOURNAL, February 13th, 1904, p. 365.

A CASE OF HAEMOPHILIA TREATED WITH ADRENALIN CHLORIDE.

By ERNEST FRANCIS, M.R.C.S.ENG., L.S.A.LOND.,
Chief Medical Officer, Assam-Bengal Railway.

On February 23rd, 1904, I was urgently summoned to see a patient who was said to have been bleeding from the mouth for about six hours. I found a young man, blanched and anxious-looking, his pulse too rapid to count, feeble and fluttering. A large quantity of coagulated blood was in a basin at the bedside and blood was being expectorated at frequent intervals.

The view of the mouth and pharynx being obscured by blood and blood-clot, a Gowan's gag and tongue depressor

was introduced and the pharynx and mouth cleaned out. It was then seen that there was deep purple ecchymosis of the right side of the hard palate, sharply limited by the middle line. The soft palate was engorged and oozing blood rapidly; the uvula was engorged, about $2\frac{1}{2}$ in. long, and bleeding freely. The uvula was seized by forceps and removed with scissors, the whole of the pharynx swabbed out with sponges saturated with adrenalin chloride solution (1 in 1,000) (Parke Davis), and a sponge saturated with the solution held to the stump of the uvula. On its removal, after several minutes all haemorrhage was found to have completely ceased. All the treated parts were completely blanched, with the exception of the ecchymosis on the right palate, which remained a dark purple and appeared extraordinarily sharply defined, presenting a striking contrast to the adjacent mucous membrane.

Adrenalin chloride solution πxv was given by the mouth. The urgent haemorrhage having been arrested, I had leisure to obtain the history of the case:

The patient was a male Hindu (Brahmin), aged 22. At the age of 12 years he received a blow on the right temple from a tennis ball. He bled from both nose and mouth for "several days afterwards."

At the age of 15 years he fell from a horizontal bar. He sustained no apparent injury, but on the following day began to bleed from the nose and mouth and to pass blood with both urine and faeces. This lasted five days. On the present occasion no blow or injury had apparently been sustained.

A puncture caused by a hypodermic injection was oozing freely. Adrenalin solution was applied to it, and no more oozing took place.

At 10 p.m. the patient vomited a large quantity of blood (probably swallowed earlier in the day). He passed a stool containing much black blood, and his urine was dark port-wine coloured. There had been no recurrence of bleeding from the mouth. There was a large, tender, and obscurely-fluctuating tumour in the right iliac fossa. The temperature was 98.4° F., and the pulse 134, regular, and much improved. The face was less anxious. He was ordered a mixture containing adrenalin solution πx and ergotinin citrate $2\frac{1}{2}$ gr. by the mouth every four hours.

February 24th, 7 a.m. Condition improved; no bleeding from mouth; appearance less anxious. Temperature 98.6° F.; pulse 118; urine black; faeces black. Adrenalin and ergotinine citrate mixture continued.

February 25th, 9 a.m. Much better. No external bleeding. Swelling in right iliac fossa less. Pulse 110, good; temperature 98.8° F.; urine clear, but slightly bloodstained; faeces black. Able to sit up in bed and talk. Adrenalin mixture discontinued, and full doses of iron and arsenic substituted.

February 26th, 9 a.m. Appearance very anxious; very restless; no external bleeding; urine port-wine coloured; has passed a copious stool consisting almost entirely of fresh blood; pulse 132, fluttering, and intermittent; breathing shallow and sighing; a hypodermic injection of ergotinine citrate $\frac{1}{10}$ gr. and strychnine $\frac{1}{10}$ gr. was at once given, and a rectal injection of 1 pint of saline solution at 100° F. The adrenalin mixture was again ordered by the mouth.

February 26th, 6 p.m. Face less anxious; breath fetid; pulse 135, non-intermittent; temperature 100.2°; evacuation of the saline rectal injection stained black; urine clear, no visible blood; no external bleeding. Another saline rectal injection given and mixture continued.

February 27th. The patient became very restless, and his temperature rose to 103° F. on the night of the 26th. I was called to him at 2 a.m., and found him extremely restless, semi-conscious; temperature 104° F.; respiration very hurried and shallow; pulse 150, failing. At 2.15 a.m. the patient became completely unconscious, the pulse imperceptible, and the respiration more rapid and shallow. Respiration ceased at 2.20 but the heart continued to beat irregularly for nearly a minute afterwards.

REMARKS.

The patient's family being wealthy and high caste Hindu no family history was obtainable, nor for the same reasons was a necropsy possible. Had I at the time been aware of the patient's previous history I should not have removed any portion of the enormously enlarged uvula. It is of interest, however, that the bleeding from the stump was at once controlled, and that there was no recurrence of any haemorrhage from the pharynx.