small areas of sac wall that, without impeding in the least the blood current, their dissolution rather than their accretion quickly follows. By the application of a strong galvanic current through coils of wire so disposed that all areas of the sac are reached it follows without exception, as has been noted in all recorded cases, that consolidation by virtue of clot formation is promptly and invariably produced. The solidification is rapid, and is generally manifest before the end of the electrical session, through changes apparent to the eye and hand, in the pulsation and in the degree of consistence of the sac wall. These changes become more decided in the course of a few days, until after a time in the most favourable cases a hard nodule, with a communicated pulsation only, replaces the previous expansible tumour. This was the history of four of the ten cases now recorded—that of Kerr, that of Rosensten, the second case of my own, and the case of Hershey, and partially so in the case of Barwell, of Roosevelt, and in the first of my own, all of which latter cases were totally beyond the slightest hope of cure at the time of treatment, as was also the case of Abbe.³

A striking demonstration of the power of this method to promote clot formation is shown in my second case, the specimen of which I now show you. In this, as stated, 10 feet of fine gold wire had been passed, and a current of 85 milliamperes employed for one and a quarter hour. Four weeks after this procedure a needle of quite large calibre was thrust into the sac to a depth of 2 inches in several situations, in which at the time of operation blood spurted at its introduction when apparently only beneath the skin. The needle in the second attempt was found to firmly engage the clot, though thrust perpendicularly up to its hilt in the sac. It could not be circumducted save with effort, and escape of even a drop of blood at any depth did not occur; nor was the needle or wire (which last had been attempted to be reinserted through the needle), when withdrawn, blood tinged. This beautifully demonstrated the apparent complete solidification of the aneurysm.⁹

One other effect possessed by the method in producing clot formation should not be lost sight of. It is that through portions of the coils of wire which must in small sections lie in direct contact with the sac wall, under the current's influence an electrolytic effect is produced on the endothelial lining of the sac, such as Macewen has demonstrated is highly favourable to the subsequent deposition of leucocytes derived from the vessel wall and by segregation from the blood stream, resulting in the formation of organisable white thrombi, from which vascularised fibrous tissue readily springs. From these wall thrombi, which in a fair number, if not in all cases treated by this method, are likely to result, organisation may rapidly extend to the red thrombi formed directly about the centrally disposed wire, and this solidification and organisation of the sac promptly occurs.

REFERENCES.

¹ *Therapeutic Gazette*, September, 1896. ² *American Journal of the Medical Sciences*, October, 1892. ³ *American Journal of the Medical Sciences*, August, 1896. ⁴ *Med.-Chir. Trans.*, xlvii. ⁵ It is within the meshes of the solidified contracting coagulum that the wire must lie. Should the wire be excessive in amount or of undue firmness, or so highly drawn that its fracture is likely, not only contractions of the organised thrombus be interfered with, but the rupture of the sac may be induced in process of the former. These imperfections in method I pointed out in detail in my first paper on the subject. ⁶ As to this point, see the last part of my first paper, in which some experiments with soft iron wire are detailed. ⁷ The quantity of wire that was originally used, both when wire alone was employed and wire reinforced by galvanism, was far in excess of that necessary to reach all portions of the sac cavity, and could not but be inimical to cure. The cases in which a moderate amount of wire was used, as in Loreta's (6 feet) and Morse's (4 to 5 feet), resulted in cure. Loreta's case, an abdominal aneurysm the size of a fetal head at term, contracted to the dimensions of a walnut with obliteration of sac cavity. Death resulted from rupture of the aorta below the sac. In Morse's case ("abdominal aneurysm the size of two fists") all indications of aneurysm had disappeared within two months, a hard nodule replacing the pulsating tumour. In the cases in which the combined treatment was employed, the amount and character of wire, duration of application of electric current, and polarity employed are as follows:—Corradi (large aneurysm, ascending part of the aortic arch): 1½ ft. No. 30 annealed wire, material not mentioned; anode active; sixteen elements; twenty-five minutes. Barwell (large aneurysm, ascending and transverse part of the aortic arch): 10 ft. finest steel wire; anode active; 10 milliamperes seventy minutes. Roosevelt ("aggravated, thoracic, aortic aneurysm threatening death"): 22½ ft. fine steel piano wire; polarity not stated; 25 milliamperes thirty minutes. Abbe ("a rapidly-advancing thoracic aneurysm at the root of the neck"): Cavity 4 by 5 inches; 100 ft. of catgut first introduced; nine days subsequently

150 ft. fine wire, material not stated; anode first active pole, 50 milliamperes thirty minutes; cathode active pole second thirty minutes. Kerr (fusiform thoracic aneurysm): 6 ft. drawn silver wire; anode active, fifty minutes. Kerr (aortic intrapericardial sacculated aneurysm): 10 ft. drawn silver wire; anode active, thirty minutes. Rosenstern (aneurysm, ascending part of the aortic arch): 2½ ft. moderately thick softened silver wire; anode active, thirty minutes. D. D. Stewart (enormous thoracic-abdominal aortic aneurysm): 2½ ft. No. 23 silver wire; anode active, 70 milliamperes sixty minutes. D. D. Stewart (innominate aneurysm): 10 ft. fine gold wire, No. 30; anode active, 80 milliamperes seventy-five minutes. Hershey (innominate aneurysm): Gold wire, No. 28; attempt to pass 10 ft., but, through kinking of wire, 2½ ft. only introduced; 40 to 70 milliamperes for sixty-five minutes; anode active. * Details of these cases will be found in my first and second papers. ⁹ An account of the exploration of the solidified sac with the needle is given in the second paper (*American Journal of the Medical Sciences*, August, 1896).

CAN BERI-BERI BE CAUSED BY FOOD SUPPLIES FROM COUNTRIES WHERE

BERI-BERI IS ENDEMIC?

By NEIL MACLEOD, M.D. EDIN.,
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THIS is a question of no small importance in the prevention and spread of outbreaks of the disease like those occurring recently in the Richmond Lunatic Asylum, near Dublin, on board ships, in various parts of Australia, and elsewhere. One cannot help being struck by the unanimity with which every writer on beri-beri considers that overcrowding and its accompaniments play an important part in the causation of the disease. My own experience has tended mostly in the same direction; nearly every year our Shanghai municipal gaol, which is apt to be overcrowded at times, furnishes its victims to beri-beri. Ships' crews, more especially in former times the Japanese navy, barracks, gaols, schools, asylums are noteworthy seats of the disease. The following outbreak on board ship excludes overcrowding as a factor, and from the peculiar limitation and unusual selection of its victims points suspicion so strongly to a particular part of the food supply which is known to come from beri-beri infested countries as almost to amount to a natural experimental demonstration.

The sailing ship *Ancona*, 2,800 tons, sailed from New York on August 19th, 1896. In the end of December the captain, aged about 50, noticed that both his feet and legs were numb, and on January 21st, 1897, that they were swollen. He did not feel particularly ill, but was depressed and felt his legs weak. The first and second officers, aged 26 and 27, noticed a week later that their legs, which had been numb for some time, were swollen. A few days later the third mate, aged 21, son of the captain, made a similar observation. They were then amongst the Solomon Islands, where they had been driven out of their course by a hurricane. The first and second officers became rapidly weaker in the legs, and a fortnight later gave up work. The captain and his son had considerable difficulty in keeping on their feet, but stuck to work, as none of the crew could be depended on for navigating the ship amongst cannibal islands where light and adverse winds detained them for 45 days. The first mate's thighs became swollen, and 35 days after the disease was first noticed he, and 10 days later the second mate, died suddenly and unexpectedly, the former shortly after having his hair cut, the latter while eating, both displaying for several days before death great restlessness, palpitation, throbbing of vessels about head and neck, difficulty of breathing, and both being clearheaded to the end. The swelling of the captain and his son's legs lasted about three weeks, and on their arrival in Shanghai on April 17th, 1897, there was still some degree of numbness of the legs and the captain's knee-jerks were absent. The son had had numbness of the tips of some of his fingers in both hands, and also of the ball of the right thumb.

The ship was built of steel in 1893, and previous to the outbreak had been only in the Clyde, Australia, Portland, Oregon, and New York, between which place and Shanghai she had touched at no other port, and received no supplies except yams, bananas, and coconuts at the Solomon Islands on one occasion, from natives who came alongside after the disease had broken out. The third mate made almost daily inquiries amongst the crew of 23 others for numbness or swelling of the legs or any other complaint, expecting the

disease to manifest itself in the forward part of the ship, but could discover no sign of sickness. The ship's cargo was kerosene. The four men attacked had been in the ship at the places mentioned, and were apparently healthy and strong on leaving New York. They messed alone in the saloon, but it is possible that the steward and cook, both Chinamen, ate to some extent of the cabin food. The rest of the crew messed forward, and consumed the same food as those aft, with the following exceptions: haddies, clams, oysters, lobsters, and salmon all in tins; ham (not eaten by the captain), bacon, sago, tapioca, arrowroot, and fruit preserved in bottles. The four officers took alcohol in no form, and the captain alone did not smoke. The saloon was a roomy apartment, and each officer had a large cabin to himself. The ship had a flush deck, and all accommodation was on the same deck. The first and second officers after they were unable for duty lived in the pilot house on deck till they died, the captain and his son not changing their quarters during the voyage. The water supply was the same for all on board, but that of the cabin was passed through a filter with a brass tap. When the captain first became sick, thinking he had indigestion and before the swelling appeared, he restricted himself entirely to maize meal, stale bread, molasses, condensed milk, and hot water. He also put his son on this diet when his legs were noticed to swell, and both took no other food until they reached Shanghai, while the first and second officers continued to eat of the usual cabin supply till they died. The absence of anything like wrist drop and colic (both which I specially inquired for), the characteristic onset, the mode of termination, and the absence of any sign of the blue gum line in the survivors, put lead poisoning out of court. No signs of irritant poisoning were manifested at any time. I am not aware of any ailment or poison that could produce a history like that presented except beri-beri. Dr. Milles, of Shanghai, heard the history independently and without any communication from me, and arrived at the same diagnosis after having seen the survivors. I may add that I have known the disease for twenty years.

Overcrowding could not be said to exist in these cases. Only officers were attacked—well housed and well fed, with greater variety of food than the crew forward. The disease was confined to and attacked the whole cabin mess. Supplies were taken on board at New York. Suspicion is at once directed to the cabin food supply. Either it was contaminated before shipment or on board. In either case it must have been that part of the cabin supply confined to cabin consumption. Contamination after shipment is almost inconceivable. The cook was not ill and cooked for all on board. The steward was associated specially with the cabin food but not so intimately as the cook, and he was not ill. Neither ship nor cargo had been in a country where beri-beri is prevalent. Thus contamination before shipment is to be looked for, and of the supplies alone consumed in the cabin sago, tapioca, and arrowroot come from a beri-beri infested country. Rice was consumed forward as well as aft. It is noteworthy that the captain and his son, who both recovered, ceased early in the outbreak to partake of the general cabin supply, and confined themselves entirely to articles which were also consumed by the crew (a limitation coincidental, not intentional, as the captain assured me), while the first and second officers, who continued to eat from the usual cabin supply, both died. They had never heard of beri-beri before reaching Shanghai.

Outbreaks of beri-beri are not uncommon on board ships that have visited countries where it is endemic, where one is apt to regard each case that occurs as having contracted the disease before sailing. These outbreaks have been almost, if not entirely, confined to Chinese, Japanese, and other Orientals, whose food stores are invariably drawn from beri-beri countries, and therefore always of suspicious origin. It is not simply a question of limitation to the fore-castle or overcrowded part of the ship, but the Oriental part of a ship's crew or passengers. In the Richmond Asylum in both epidemics the disease attacked nurses as well as overcrowded patients, both being no doubt fed from some supply in common. This Ancona outbreak is allied to the Richmond ones, inasmuch as none of the sufferers had been to beri-beri countries. Whether overcrowding exists or not in such cases, there is another condition present which is liable

to be overlooked. Food supplies are apt to be stored in large rather than in small quantities, from financial and other considerations, so that if the supply is from a contaminated source the poison may, so to speak, be given in continuous doses until the stored material is exhausted. This may very easily happen on board ship, where stores have to be provided against a long voyage. On the other hand, a contaminated supply scattered amongst retail dealers, and distributed by them amongst their customers in small quantities, is thus so quickly and widely disseminated that probably few customers can be supplied continuously with the poison—a condition very different from its concentration among the inmates of an asylum or a ship's crew. Disease caused in this way, especially one commencing so insidiously as beri-beri does, may easily escape notice, few doses of the poison being taken by the same individuals. Dr. Milles, of Shanghai, informs me that on the occasion of an epidemic of beri-beri in the Shanghai municipal gaol he examined at the same time a larger number of Chinese hospital out-patients, and was much surprised to find a striking proportion of them with numbness of the legs, with or without swelling or patellar reflex, some of them merely complaining of not being well.

In an editorial article in the *BRITISH MEDICAL JOURNAL*, vol. ii, 1895, the writer regards beri-beri as endemic in Australia on the following grounds:

1. Dr. Molloy (*a*), of the Melbourne Hospital, in a paper entitled *Is Beri-beri Endemic in Australia?* mentions that "in March, 1888, 9 Chinese were brought from a particular shop to the Alfred Hospital, all of them suffering from symptoms of peripheral neuritis and oedema;" and he further states that "subsequently a series of similar cases cropped up in Melbourne."

2. Dr. Graham (*b*) describes a form of peripheral neuritis epidemic amongst the Chinese in Sydney; and Dr. R. T. Paton (*c*), who has seen beri-beri in its recognised haunts in the East, has concluded that these cases of peripheral neuritis are really of this nature.

3. An outbreak of beri-beri among some aboriginal Australians (*d*) at Wyndham, E. Kimberley. (This, I may add, was amongst 60 aborigines in gaol, who, it is stated in the original paper, were allowed half a pound of rice each daily.)

The article continues: "It is evident, therefore, that an endemic form of peripheral neuritis is extensively distributed in Australia, seeing that we have accounts of it from three widely-separated points; and now Dr. Corlette's account, based on an experience of 60 cases, does away with any doubt that may have existed as to its true nature and significance. ....Hitherto only Chinese and other Orientals have been attacked, and from this circumstance it might have been conjectured that the disease was imported; but on inquiry it is found that many of those attacked had been resident in the colonies for years. Moreover, it was ascertained that the disease tended to occur in little epidemics, several cases coming from one house—a fact distinctly pointing to the existence of local endemic foci. ....Although hitherto, so far as is known, confined to Chinese, there can be little doubt. ....," etc.

Here the italicised (by me) extracts seem to me to direct suspicion to the food supply certainly obtained from beri-beri infested countries, and thus possibly infected with the poison, rather than to an endemic source. Here again the question of storing a food supply presents itself, for Chinese families prefer Chinese rice and lay in a store of it. The gaol mentioned consumed 30 lbs. daily amongst 60 prisoners, doubtless also from a stored quantity.

This disease is almost certainly toxic, and in the first instance probably microbic, but whether the toxin or the microbe is administered to its victims we are not yet in a position to decide. Diphtheria toxin produces a degeneration of nerves, whether its organism or its toxin be introduced into an animal. Alcohol by continuous administration in some cases is known to produce a peripheral neuritis which to me is almost impossible to distinguish from certain cases of beri-beri. Alcohol is also microbic in its source.

How would a beri-beri microbe or toxin in food such as rice, sago, etc., be affected by the cooking process? Dishes made of tapioca, sago, etc., are not necessarily boiled, though rice usually is, so that even a microbe might pass through the

cooking process unharmed, and certainly a toxin could do so even if boiled.

In the light of these and other similar occurrences of the disease, the effect of a change of the Japanese navy dietary in practically banishing it from that service, and similar experiences in Shanghai gaol outbreaks, the cases of the *Ancona* here recorded seem to me to have almost the force of a demonstration that food supply is the vehicle carrying the disease poison into countries where it is not endemic. When it occurs in epidemic form, at least in such countries, and in a gaol, asylum, or like institution, or at sea, the first step towards stopping it is to cut off those articles of food which have come from countries where the disease is known to occur—a simple matter surely.

Beri-beri is so frequently insidious in its onset that it has probably escaped detection in European countries hitherto, except when epidemic, or it has been mistaken for other forms of peripheral neuritis, for without doubt peripheral neuritis is a marked feature, and often confined to the lower extremities in beri-beri.

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(a) *Transactions of the Third Session of the Intercolonial Medical Congress of Australia*, 1892. (b) *Australasian Medical Gazette*, No. 146, November 15th, 1893. (c) *Ibid.*, No. 158, November, 1894. (d) *BRITISH MEDICAL JOURNAL*, October 27th, 1894.

CASES OF ANEURYSM OF THE AORTA COMMUNICATING WITH THE PULMONARY ARTERY.

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ALTHOUGH rupture into the pulmonary artery is not a very uncommon sequel to aneurysm of the aorta—6 per cent. Fagge,¹ 8 per cent. Sibson²—owing to the rapid onset of serious symptoms quickly terminating in death, which seems to be the usual occurrence in this lesion, the number of cases in which the physical signs diagnostic of the condition have been noted is very small. A typical case of this description having occurred in a woman under the care of Dr. Eustace Smith at the City of London Hospital for Diseases of the Chest, and the patient having survived long enough for the characteristic signs and symptoms of this peculiar complication of aortic aneurysm to be noted, I thought the case well worthy of record.

I am indebted to Dr. Eustace Smith for permission to publish this case, to Dr. Colbeck for giving me the opportunity of seeing the patient before her admission, and to Dr. Perkins for the *post mortem* notes.

On February 19th, 1897, M. A., a married woman aged 26, came to the out-patient department complaining of pain across the chest and between the shoulders, with a slight cough and scanty aerated expectoration. She had at this time little or no dyspnoea or other urgent symptoms of chest trouble. A loud continuous murmur was heard in the second left intercostal space close to the sternum, with no audible pulmonary second sound. Her lungs were clear. A week later she was in much the same condition except that she was rather more short of breath, and a few rhonchi were heard over both lungs.

She was admitted as an in-patient on March 5th, when she gave the following history: Her family history was good. She had had a cough as long as she could remember, both winter and summer, which had gradually become worse during the past three months; she had not, however, actually taken to her bed until two days after her second visit to the hospital as an out-patient, five days before her admission; since then the dyspnoea had rapidly become more urgent. There was no history of scarlet fever or chorea. She had had "rheumatic gout" in the right ankle and left shoulder and fingers about eight months earlier, with pains in the joints frequently since; she complained of pain in the right shoulder at the time of her admission. She had been married four years, and had had one child three years ago, who died of bronchitis at 3 months of age. She had had no miscarriages. She had "ulcers of the legs" after her confinement;

these ulcers did not heal until nearly a year afterwards. There was no further history pointing to syphilis. About three months ago, when going upstairs, she was suddenly taken with severe pain of a stabbing character across the chest and back between the scapulae. It was from this time that she dated her illness. A week before admission she suffered from a persistent cough, day and night, with profuse, frothy expectoration, night sweats, and dyspnoea.

The patient was a thin, pale woman, with a slight hectic flush, but no cyanosis, jaundice, or clubbing of the fingers. She had orthopnoea and a short hacking cough with profuse blood-stained aerated expectoration. The respirations were 44, the pulse small, quick, and irregular, could not be counted; the heart-rate was 132. The heart's impulse was felt in the fifth interspace 3 inches from the middle of the sternum, and $\frac{1}{2}$ inch outside the left nipple line.

A brief examination showed that the right chest contained a considerable amount of fluid, and 33 ounces of clear serous fluid were withdrawn by aspiration.

The following morning (March 6th) the patient was much less distressed, so that a more complete examination was possible. The chest was slightly rounded and expanded badly, respirations being mainly abdominal. There was marked dullness on the left side below the ninth rib, and below the angle of the scapula on the right side. The vocal fremitus and resonance and breath sounds were diminished, though not lost, at both bases, the latter being distinctly of bronchial type. A coarse friction sound was audible over the upper line of dullness on the right side. The heart's impulse was in the fifth space just inside the left nipple line. A prolonged systolic thrill was felt over the second, third, and fourth left costal cartilages, but not over the apex of the heart. The cardiac dullness was limited above by the upper border of the third rib, on the right side by the right edge of the sternum, and on the left by the nipple line.

On auscultation in the second left interspace, close to the sternum, over an area not larger than the chest-piece of a stethoscope a loud continuous murmur, resembling in quality the hæmic *bruit* heard at the same spot in anæmia, was audible. This murmur was accentuated at the systole, and continued throughout the cardiac cycle, gradually becoming less marked until the next systole. No second sound could be heard.

In the interspace below, and also over the sternum and in the second right interspace two murmurs, systolic and diastolic in time, were audible, the latter being well conducted down towards the ensiform cartilage, whilst the former was conducted into the neck, where the second sound was faintly heard. The second sound was also distinctly audible, both at the apex and outside the nipple, and was apparently quite free from murmur, though it was sometimes reduplicated. The pulse (108) was fairly regular, the upstroke sharp, with rapid fall, entirely collapsing between the beats. The radial pulse was visible at the wrist, and there was capillary pulsation. No enlargement of liver or spleen was detected. The urine, moderate in quantity, contained a trace of albumen; specific gravity 1022. In front of both tibiae and in front of the left thigh old scars, with serpiginous outline, but not pigmented, were seen, those on the thigh especially pointed to syphilis as their cause.

The following evening, the dyspnoea again becoming urgent, venesection was performed, and the right pleural cavity was again aspirated, 32 ounces of blood-stained serum being withdrawn. During the subsequent week the patient seemed to improve a little, except that, in spite of treatment, she was much troubled with excessive flatulence day and night, irrespective of food. The sputum was still large in quantity, muco-serous, aerated, and blood-stained, sometimes bright in colour, at other times typically "rusty."

She again began to suffer from dyspnoea, and complained of a heavy, dull pain over the upper part of the sternum. Though the breath sounds were fairly well heard at both lung bases, they were of bronchial character, so it was again decided to explore for fluid at the right base, where the dullness was more marked, but though the needle was inserted twice nothing beyond a little blood was withdrawn.

The flatulence now became even more distressing; every few breaths she had large eructations of gas from the stomach, which nothing seemed to relieve. She died at 3.30 A.M. on