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.

REFERENCES.

(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.

By C. LAMPLOUGH, M.R.C.S., L.R.C.P.,

Resident Medical Officer to the City of London Hospital for Diseases of the Chest, Victoria Park, N.E.

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

March 18th, twenty-seven days after the first examination, and about four months after the onset of the dyspnoea. The temperature remained normal until the day before death, when it ran up to 103.2° F., but returned to normal again before the end.

Necropsy.—Twelve hours after death, rigor mortis present; no oedema. There were 6 ounces of clear fluid in the pericardium, but no pericarditis. Between the pulmonary artery and the aorta anteriorly the vessel was obviously too prominent and thickened. The heart, with four inches of the aorta, weighed 15 ozs. The left ventricle was dilated, but not hypertrophied, whilst the right was much thickened. The mitral and aortic valves were healthy. Immediately above the central cusp of the pulmonary valve was a small ulcer the size of a pea, to which the edge of the cusp was firmly bound down for about one-third of its extent. Starting about half an inch above the valve, and reaching to about half an inch below the bifurcation of the pulmonary artery, the wall of that vessel was bulged inwards over a space about the size of a pullet's egg; two small openings, with smooth thinned rounded edges, were present in the lower part of the bulging, while in the upper part were a large superficial ulcer, about half an inch in diameter, and several small atheromatous spots. The opening led into an aneurysmal sac containing a good deal of blood clot, and placed apparently between the aorta and pulmonary artery. In the wall of the aorta, about three quarters of an inch above the aortic valve, was a circular, smooth-edged opening, one-third of an inch in diameter, at the base of a small depression leading into the above-mentioned sac. Scattered through the thoracic aorta were a number of small patches of early atheroma, and just below the site of the obliterated ductus arteriosus was a circular patch, the size of a florin, where the wall was bulged, weakened, and wrinkled, but without atheroma or breach of surface. The right pleura contained 25 ounces, and the left 15 ounces of clear fluid. Both lungs were oedematous, and the right especially showed a red induration. The stomach was greatly dilated, its lower edge reaching to the level of a line joining the two anterior superior iliac spines, and the organ occupying the whole of the left and middle regions of the abdomen above. The liver, which weighed 46 ounces, was "nutmeg." The other organs were fairly healthy.

An excellent paper was read before the Medico-Chirurgical Society by Mr. John Thurnam, nearly sixty years ago, when the symptoms and physical signs produced by aneurysms of the aorta communicating with the surrounding venous trunks were fully and minutely described. Very few cases which have been even suspected during life, and in which the pulmonary artery was the communicating vessel, have been published since. I have been able to find only 15 cases in which a careful examination was possible before the death of the patient. The following are brief abstracts of the published descriptions of these cases:

CASE I.—Mr. J. Thurnam (*Med.-Chir. Trans.*, 1840, xxiii, 323). **Previous History.**—Male, aged 33, baker. Had severe rheumatism thirteen years ago.

Symptoms following Rupture.—Sudden sense of "cracking" in chest during violent exertion, with faintness and dyspnoea, followed by anasarca and sanguinolent and viscid sputa. Pulse extremely jerking. A very superficial and continuous sawing sound, with purring tremor supplanting normal sounds of heart, very intense in second left inter-space 2 inches from sternum.

Probable Length of Life after Rupture.—Eleven weeks. **Necropsy.**—True aneurysm of right aortic sinus, projecting into and communicating by two small rounded apertures with the right ventricle; more or less insufficiency of pulmonic and aortic valves; hypertrophy and dilatation of heart; hydropericardium; hydrothorax of right side; pulmonary, bronchial, and hepatic venous congestion.

CASE II.—Dr. Munro (*Edin. Med. and Surg. Journ.*, 1836, xlv, 322). **Previous History.**—Male, aged 24, porter, accustomed to lift heavy weights. Had acute rheumatism ten years previously.

Symptoms following Rupture.—Dyspnoea, palpitation, and oedema. Countenance tumid and livid. Pulse 112, large, hard, and "thrilling." Cardiac dulness enlarged. A loud, blowing, continuous (Dr. Hope) murmur, best heard over midsternum, also heard all over front of chest and in the back.

Probable Length of Life after Rupture.—Ten months, with final rupture just before death.

Necropsy.—Several pounds of serum in each pleura. Heart dilated and hypertrophied. Aortic valves thickened, but no actual disease of cardiac valves. Aorta atheromatous. Ascending aorta was dilated into a large irregular sac, which adhered to pulmonary artery and communicated with it by two openings; the larger would admit the point of the little finger, the smaller a crow-quill; edges were rounded, regular, and cartilaginous. Nearer the arch was a third opening with ragged edges.

CASE III.—Dr. Smith (*Dublin Med. Journ.*, 1841, xviii, 164).

Previous History.—Male, aged 22, subject to epileptiform attacks. **Symptoms following Rupture.**—Dyspnoea, faintness, and palpitation, with precordial pain. The face was pale and bloated, and the lips blue. Pulse intermittent. Intense *fremissement* over precordia and systolic murmur. He died in an epileptiform attack.

Probable Length of Life after Rupture.—Four months.

Necropsy.—Left ventricle dilated but not hypertrophied. Right ventricle somewhat thick. Aneurysm of commencement of aorta communicating with the pulmonary artery near its origin by a small opening with thickened and rounded edges.

CASE IV.—Dr. Turnbull (*Lancet*, 1845, ii, 91).

Previous History.—Male, aged 41. A seaman. He fell down a hatchway three months before death.

Symptoms following Rupture.—Dyspnoea and a cough rapidly increasing for three weeks before death. Pulse 92, strong, full, and jerking. Thrill or purring tremor felt over base of heart to left of sternum. A loud harsh, continuous murmur with intensity during the ventricular systole. Over aortic area there was a dry double murmur. Both sounds audible at apex, but obscured by aneurysmal *bruit*. No lividity or oedema.

Probable Length of Life after Rupture.—About three weeks.

Necropsy.—Left ventricle hypertrophied. Cardiac valves normal. A multilobular sacculated aneurysm of the ascending aorta communicating by one of its pouches with the pulmonary artery, and another with the right ventricle, one opening being just above, the other just below the pulmonic valves.

CASE V.—Dr. J. W. Ogle (*Trans. Path. Soc.*, 1852-53, iv, 114).

Previous History.—Female, aged 27. Had occasionally suffered from palpitation and dyspnoea for some years.

Symptoms following Rupture.—Hæmoptysis and dyspnoea, with oedema of the lower extremities. Face pale and lips blue, but no marked cyanosis. Heart irregular, pulse 120 and very jerky. "Rusty" sputum mixed with bright blood. Systolic tremor felt in third left intercostal space, and a loud systolic murmur plainly audible over this situation also heard all over precordia. Further to the left of sternum the second sound was absent, but it was clear and distinct over the aortic area.

Probable Length of Life after Rupture.—About six weeks.

Necropsy.—Cavities of heart were dilated. Valves except pulmonic, were healthy. An aneurysm of the aorta just above valve flaps communicating by an oval opening with the pulmonary artery. Portions of the semilunar flaps of the pulmonary valve were adherent to the aneurysmal sac.

CASE VI.—Dr. Hughes Bennett (*Prin. and Pract. of Med.*, 3rd Ed., 1859, p. 595).

Previous History.—Male, aged 33, a teacher. Had long suffered from hæmorrhoids. Four months before death felt as if something in his chest had ruptured.

Symptoms following Rupture.—Dyspnoea and cough with blood-stained sputum. Feet oedematous. Heart's impulse strong and irregular. No thrill felt. Over manubrium there was a continuous blowing murmur occupying period of both sounds; also audible below the clavicles, softer and more distant to right of sternum. Over third left costal cartilage the first sound was long and blowing, the second abrupt and rasping. Pulse 90, irregular, of natural strength; became rapid and weak before death, and the skin was jaundiced.

Probable Length of Life after Rupture.—About four months.

Necropsy.—Skin and conjunctivæ tinged yellow. Cardiac valves were competent. Left ventricle slightly dilated and hypertrophied. An aneurysm of the ascending aorta about the size of a walnut and communicating with the pulmonary artery by an oval, smooth-edged opening, four lines in length, which was situated one inch above the sigmoid valves.

CASE VII.—M. Pierreson (*Bull. de la Soc. Anat.*, 2me Serie, 1860, p. 98).

Previous History.—Male, aged 61, a labourer, formerly a soldier.

Symptoms following Rupture.—Increasing dyspnoea and oedema of lower extremities. When seen had orthopnoea, lividity of lips and face; pulsating tremor in second left intercostal space near the sternum; *souffle* with the first sound and purring tremor; and later had distension of external jugulars.

Probable Length of Life after Rupture.—About one year.

Necropsy.—Recent pericarditis, slight hypertrophy of left ventricle; valves healthy. A biloculate aneurysm of ascending aorta one inch above valves, communicating by a small opening with the pulmonary artery.

CASE VIII.—Sir Willoughby F. Wade (*Med.-Chir. Trans.*, 1861, xlv, 211).

Previous History.—Male, aged 35, a railway porter. Had sudden attack of dyspnoea and precordial pain whilst making a violent effort to avoid an engine.

Symptoms following Rupture.—Cough, dyspnoea and emaciation, with pain across the chest. Two loud murmurs, audible over fourth left costal cartilage, that with the second sound being of a hissing character, and so prolonged as to continue till commencement of the next ventricular systole. At this spot there was a considerable purring tremor accompanying the second murmur. At the apex there was a systolic murmur, with a distinct second sound unaccompanied by any *bruit*. There was marked pulsation in the carotids, and no cyanosis.

Probable Length of Life after Rupture.—The opening into the pulmonary artery had probably existed about six months, whilst that into the right ventricle possibly occurred a fortnight before death, when the patient was suddenly seized with severe pain over the precordia, followed by severe dyspnoea.

Necropsy.—An aneurysm, the size of a hen's egg, springing from the origin of the aorta, compressing the right ventricle and communicating with it by an opening with thin and ragged margins. It also communicated with the pulmonary artery by a vertical slit with regular and smooth edges.

CASE IX.—Dr. Peacock (*Trans. Path. Soc.*, 1868, xix, 111).

Previous History.—Male, aged 26, who had had rheumatic fever four years before death.

Symptoms following Rupture.—Pain in abdomen with dyspnoea and cough, followed by oedema. Cheeks flushed, lips livid, pulse sharp and of somewhat tegurgitant character. A systolic murmur, best heard at

fourth costal cartilage, and followed by a ringing second sound, which was followed by a faint murmur. The second sound was audible at the apex, and unaccompanied by a murmur.

Probable Length of Life after Rupture.—About three months.

Necropsy.—Heart hypertrophied, and weighed 28 ozs. Aortic valves were thickened and incompetent. Immediately above the aortic valve was an aneurysmal sac, the size of a hen's egg, which protruded into and obstructed the lumen of the pulmonary artery, and communicated with that vessel by an oval opening with thin and smooth edges. One pulmonary valve flap was completely destroyed, and another partially so.

CASE X.—Sir William Roberts (BRITISH MEDICAL JOURNAL, 1868, i, 421).

Previous History.—Male, aged 28, an organist, somewhat intemperate.

Symptoms following Rupture.—Cough and dyspnoea followed by oedema and ascites. Face pale and thin; lips somewhat livid; sputum, profuse liquid albuminous matter mixed with froth and specks of blood. Pulse 112, markedly jerking, and visible in all the superficial arteries; loud, harsh, and rasping double murmur, or rather a single murmur consisting of two parts, covering both sounds completely, best heard over a point midway between manubrium and left nipple, diminishing in intensity in all directions from this point, audible but faint at the apex but not heard beyond, well conducted into the neck; murmur accompanied by an intense vibratile thrill.

Probable Length of Life after Rupture.—About two months.

Necropsy.—Cardiac cavities enormously dilated and the ventricles hypertrophied; mitral and tricuspid valves were fairly patulous; aortic and pulmonary valves were competent; a wide-mouthed aneurysm just above aortic valves bulging into and communicating with the pulmonary artery just above its valves by three openings, one the size of a pea the others much smaller; the edges of these openings were thin and diaphanous, perfectly smooth, and free from atheromatous patches.

CASE XI.—Dr. Murchison (Trans. Path. Soc., 1868, xix, 190).

Present History.—Male, aged 48, a brushmaker. No previous illness.

Symptoms following Rupture.—Fell off a plank in a sawpit, and afterwards suffered from palpitation and dyspnoea, followed by oedema with ascites. Six months afterwards face was pale. Pulse 112, feeble but regular. A prolonged bellows murmur well heard at apex and also at mid-sternum and over the back of the chest, but not audible in the vessels of the neck. The urine contained one-tenth volume of albumen, but no tube casts.

Probable Length of Life after Rupture.—About eight months.

Necropsy.—Dilatation of all the cardiac cavities, with hypertrophy of right heart. Mitral and pulmonary orifices were moderately patulous. The aortic and tricuspid valves were competent. A fusiform aneurysm of the ascending aorta communicated with the pulmonary artery $\frac{1}{4}$ inch above its valves by a circular hole about four lines in diameter, with rounded and regular margin, and with a fringe of minute vegetation on its aortic surface. Kidneys slightly granular.

CASE XII.—Dr. Cayley (Trans. Path. Soc., 1870, xxi, 122).

Previous History.—Male, aged 37, a glider; had lead colic five years previously.

Symptoms following Rupture.—Sudden dyspnoea followed by oedema. When seen shortly before death patient was anæmic but livid. Pulse had a collapsing character. A double bellows murmur audible over second left interspace and down left of sternum, with a loud pericardial friction sound. Urine albuminous, and contained blood and granular casts.

Probable Length of Life after Rupture.—About three months.

Necropsy.—An aneurysm of the ascending aorta, the size of a walnut, communicating with the pulmonary artery just above the semilunar valves, by a circular orifice a quarter of an inch in diameter, with thin and smooth margins. The aortic valves were thickened but competent, and the other valves were normal. Left ventricle much hypertrophied and the right cavities dilated. Aorta atheromatous. Recent pericarditis and peritonitis. Kidneys in an advanced stage of degeneration.

CASE XIII.—Dr. Frederick Taylor (Trans. Path. Soc., 1874, xxv, 56).

Previous History.—Male, aged 30. Had been a soldier. No history of rheumatism, gout, or venereal disease.

Symptoms following Rupture.—Gradually increasing dyspnoea with oedema of the legs, and pain in the chest. Seen forty-eight hours before death, the face was thin and the skin yellowish, with oedema of lower trunk and legs. Marked thrill over precordia. At base a loud systolic and rather less loud diastolic murmur, the latter heard also half way down the sternum, the former towards the apex and in axilla, but not in the back. Pulse 108, not splashing in character.

Probable Length of Life after Rupture.—Probably about three weeks.

Necropsy.—Heart weighed 20 ounces. Both ventricles hypertrophied and the right was dilated. An aneurysm of the ascending aorta, the size of a walnut, communicating with the pulmonary artery by an opening about half an inch long, with smooth margins, except at the upper end, where there were a few fine vegetations. Aortic valves were healthy. The left half of the right posterior cusp of the pulmonary valve was bound down to the arterial wall.

CASE XIV.—Dr. Samuel West (Trans. Path. Soc., 1882-83, xxxiv, 64).

Previous History.—Male, aged 38, a groom.

Symptoms following Rupture.—Sudden pain in front of chest, with cough and dyspnoea since. A coarse thrill could be felt in the fourth left intercostal space. A loud double murmur could be heard over the same area and also at the apex and in the back. The murmur seemed to commence during the latter part of diastole and to extend through the whole of the systole.

Probable Length of Life after Rupture.—About 10 weeks.

Necropsy.—Both ventricles dilated, with fatty walls. A somewhat sacculated aneurysm of the aorta the size of an egg, communicated by a circular hole half an inch in diameter, with the pulmonary artery just above the septal cusp of the sigmoid valve; about a quarter of an inch of this cusp was adherent to the margin of the opening; aorta slightly atheromatous.

CASE XV.—Dr. Handford (Edin. Med. Journ., 1888, xxxiii, 695).

Previous History.—Male, aged 35. No history of rheumatism or syphilis.

Symptoms following Rupture.—Complained of precordial pain and orthopnoea. Pulse 120, irregular, small, feeble, and fluttering. Slight oedema of legs and abundant frothy sputum. Marked systolic thrill and harsh

systolic murmur in third left intercostal space. Over the aortic area the first sound was prolonged, and the second, accentuated, but no murmur, was audible. Later a tricuspid murmur developed.

Probable Length of Life after Rupture.—Not stated.

Necropsy.—Right heart much dilated. Tricuspid and mitral valves moderately patulous. Pulmonary and aortic valves were healthy. An aneurysm of the ascending aorta, arising from the left anterior wall by a large opening, and communicating with the pulmonary artery by an opening about the size of a threepenny piece, which was crossed by a narrow bridge. The sac contained only post-mortem clot. There were no signs of visceral syphilis and no general atheroma. (This specimen is now in the museum of the Royal College of Surgeons, No. 3,165A.)

CASE XVI.—(See ante).

In addition to this series I have been able to find records of sixteen additional cases, but in these the symptoms were not described, owing usually to the patients dying shortly after the rupture had occurred; these will only be referred to, therefore, when the results of the necropsies have a clinical interest.

By studying the salient features described in this series of sixteen cases, the following appear to be the most important points upon which to form a correct diagnosis of this lesion:

1. *The Sex.*—As aortic aneurysm is much more common in men, its rupture into the pulmonary artery is proportionately more frequent in the male sex. In only two cases (v, xvi) were the patients women. In one case (Dr. Hebb's) of the sixteen above mentioned the patient was a woman.

2. *Age.*—The youngest patient was aged 22, the oldest 61.

3. *The History of the Illness.*—The onset was sudden in nearly every case, and accompanied by pain across the chest and dyspnoea; cough and oedema usually followed, with profuse blood-stained expectoration. The sudden onset would assist in the diagnosis from valvular disease of the heart.

4. *The Appearance of the Patient.*—In seven cases (ii, iii, v, x, xi, xii, xvi) the countenance was pale and the lips livid, cyanosis was absent, and there was but little oedema, thus differing markedly from cases of aneurysm of the aorta communicating with the superior vena cava, in which there is much cyanosis of the face and oedema over the upper half of the trunk and the head and neck, and also from cases in which the communication is with the right auricle or right ventricle, when there is usually general oedema and cyanosis. Communication with the left auricle may be practically excluded from its extreme rarity, but in such a case the signs would be those of mitral disease.

5. *Physical Signs on Examination of the Heart.*—A systolic thrill, most marked to the left of the sternum, near the base of the heart, was felt in 10 cases (i, iii, iv, v, vii, viii, x, xiii, xiv, xvi), whilst in only 1 (vi) of the remaining 6 was it definitely stated that no thrill was present. A thrill caused by a valvular lesion at the base of the heart would frequently be difficult to separate from that caused by an aneurysm of the ascending aorta; but the former is rarely accompanied by a thrill, whilst when the latter communicates with the pulmonary artery it apparently gives rise to a "purring tremor" in over 60 per cent. of all cases. It is a striking fact that neither an expansile pulsation nor an area of dullness separable from that due to the heart was noted in any of these cases, for in no case was the aneurysm very large; in 3 it was compared to a hen's egg, and in 5 other cases it was described as being as large as a walnut, whilst in the remaining cases the size of the sac was not mentioned. The presence of a continuous murmur over the base of the heart is apparently almost pathognomonic of an aneurysm of the aorta communicating with one of the surrounding large venous trunks or with one of the cardiac cavities, any one of which lesions is called a "varicose aneurysm" by Dr. Thurnam. As no other condition could well produce a rhythmic continuous bruit; and as this peculiar characteristic was noted in no fewer than 8 (50 per cent.) of the cases recorded (i, ii, iv, vi, viii, x, xiii, xvi) it is at least a most important sign. The murmur is best heard to the left of the sternum, when the communication is with the pulmonary artery, and to the right of that bone when the opening is into the superior vena cava. A simple opening between the aorta and a venous trunk without the interposition of an aneurysm would probably not produce a continuous murmur. Indeed, a case of this kind occurred at this hospital a year ago. At a necropsy on a patient who had died with the usual signs of aortic disease, in addition to the aortic valvular disease, the pulmonary artery and aorta were found to be firmly adherent to one another, and a

round hole about one-third of an inch in diameter, having a smooth, slightly thickened margin, situated about an inch above the aortic valves, was found in the attached walls of the two vessels. There was in this case no sign of the communication, but the double murmur of aortic disease was plainly audible. This peculiar murmur is probably caused by the forcible systole of the left ventricle ejecting blood through the aneurysmal sac into the pulmonary artery, and so producing the systolic portion of the murmur, whilst the elastic recoil of the aorta exerts sufficient pressure to produce a continuous flow from that vessel through the aneurysmal sac (which apparently acts as a reservoir) into the pulmonary artery, and so gives rise to the second portion of the murmur. The *bruit* has, therefore, its maximum intensity during the ventricular systole, and gradually diminishes during the remainder of the cardiac cycle until the next systole. The absence of this murmur in half the cases might be explained by (a) disease of the walls of the aorta interfering with their elasticity; or (b) the presence of a large amount of blood clot in the aneurysmal sac; or (c) excessive regurgitation through the aortic orifice, in disease of its valves, rapidly lowering the blood pressure in the vessel beyond. The presence of the second sound without any accompanying murmur outside the apex beat is an important point in the exclusion of aortic regurgitation; at the same time its absence, either at the apex or in the carotids, would not confirm a diagnosis of aortic incompetence, for the valves at the aortic orifice might be quite normal and yet, owing to the regurgitation from the aorta into the pulmonary artery, the reaction of the blood upon the aortic valves would be insufficient to produce an audible second sound.

6. *The pulse* in no fewer than 9 cases (I, II, IV, V, VIII, IX, X, XII, XVI) was described as jerking or regurgitant, though in only 2 of these cases was there disease of the aortic valves sufficient to allow regurgitation. In Case XVI the pulse was typically collapsing and there was marked visible arterial and capillary pulsation, though *post mortem* the aortic valves were found to be quite competent. Although there was no incompetence of the aortic orifice, there was nevertheless regurgitation from the aorta into the pulmonary artery, which would account for the quick fall in the pulse. Unfortunately no pulse tracing was taken in this case, though this characteristic is well shown in a sphygmogram figured with the description of Sir William Roberts's case (x) in the *BRITISH MEDICAL JOURNAL* of 1868. In the *XX Century Practice of Modern Medical Science*, vol. iv, Whittaker says that in this lesion "the peculiar pulse of aortic insufficiency is, however, never seen." The above cases appear to show, however, that this characteristic is by no means infrequent without co-existing aortic regurgitation.

I can find no reference in any existing textbook to a symptom of thoracic aneurysm which was very marked in the case (xvi) which was primarily the subject of this paper—I refer to the excessive flatulence present for a week before death; and, though this was more probably due to the acute dilatation of the stomach which is sometimes seen in patients dying from cardiac failure, I find that this symptom was noticed by Dr. W. F. Atlee nearly thirty years ago, and described by him in an excellent paper on A Case of Thoracic Aneurysm, with some remarks upon Continued and Obstinate Eructations as a Symptom of the Disease,⁵ due, he states, to pressure on the vagus. In the present case, however, the trunk of the vagus was not found to be involved.

This lesion then usually occurs in an adult male; the onset is sudden and often accompanied by pain across the sternum, and is followed by dyspnoea and cough, with profuse expectoration. The patient is usually pale and rarely cyanosed. The pulse is jerky or regurgitant in character, even when no signs of aortic insufficiency are present. In over 50 per cent. of all cases a purring tremor or vibratile thrill, systolic in time, is felt over the pulmonary area, and a continuous murmur is audible over the same situation.

Prognosis.—Death usually occurs in a few weeks or months, in only one case was the patient found to have survived the rupture for a year.

REFERENCES.

¹ *Textbook of the Principles and Practice of Medicine*. ² *Diseases of the Aorta*. ³ (1) *Med. Chir. Trans.*, 1840, xxii, 349; (2) *Edin. Med. and Surg. Journ.*, 1840, liii, 99; (3) *Ibid.*, 101; (4) *Ibid.*, liv, 114; (5) *Krank. der Arter.*

Wien., 1857-7. *Beob. Taf.*, x, 51; (6) *BRITISH MEDICAL JOURNAL*, 1853, 683; (7) *Trans. Path. Soc.*, 1883-84, xxxv, 134; (8) *BRITISH MEDICAL JOURNAL*, 1880, 521; (9) to (13) Five specimens in St. Bartholomew's Museum, Nos. 1473, 1475, 1476, 1476A, 1477; (14) and (15) Two specimens in the Museum of the Royal College of Surgeons, Nos. 3164 and 3165; (16) One specimen in the Museum of the Faculty of Medicine of Paris. * *Pepper and Griffith, International Journal of Med. Sciences*, October, 1890. ⁵ *Amer. Journ. of Med. Science*, 1869, xviii.

THE TREATMENT OF TUBERCULOUS DISEASE OF THE HIP-JOINT IN ITS EARLY STAGES.

By HOWARD MARSH, F.R.C.S.,
Surgeon to St. Bartholomew's Hospital.

I HAVE no wish to intrude upon the attention of the readers of the *BRITISH MEDICAL JOURNAL*, or, for a moment, to challenge any views by which Mr. Tobin regulates his own practice in the treatment of hip disease in its early stages. But when a teacher of surgery occupying Mr. Tobin's position gives an account¹ of a novel proceeding, in reference to an important subject, some discussion of points that seem doubtful may, I hope, be allowed.

1. As to the "position of greatest ease." In what light are we to regard this posture, whether in the hip or in the other joints, when they are in the early stage of tuberculous disease? How is it to be explained, and in what manner, and in what degree, if at all, does it contribute to the advantage of the joint in which it is observed? Mr. Tobin, speaking only of the hip, says it is desirable to retain this position, and he adds further on in his paper, "If it is an expression of a desire for a rest in any particular position it is no small indication. Should we not believe in such an indication until it is proved to be false? Are not Nature's ways as a rule conservative?" In reply I will venture to say that in no joint except the shoulder,² and least of all in the hip, should the position of greatest ease be retained. On the contrary, it is the duty of the surgeon either at once, or as soon as may be, to exchange this for some other position which, however, will vary with the joint concerned.

To make good this contention it is necessary in the first place to study this position as it is observed in healthy joints. Take, first, the simplest examples—the wrist and the ankle. The wrist, during muscular activity of the forearm and hand, assumes many positions—flexion, extension, abduction, etc.; but when voluntary muscular action ceases the hand, unless it is supported by some external agency, drops by its own weight till it forms an angle of about 120 degrees with the forearm, so that all its component structures are in a position of easy relaxation. In the same way, under similar conditions and with a like result, the foot drops to an angle of about 120 degrees with the leg. In both the wrist and the ankle the agency which at a certain point counteracts the weight of the hand and foot respectively seems to be the tonic contraction of the extensors; for when these muscles are paralysed by damage to the musculo-spiral nerve in the upper, or as the result of anterior poliomyelitis in the lower limb, the drop of the hand and the foot is much more marked. As to the elbow. When voluntary muscular action ceases, and when the limb is left without external support, the forearm drops so as to form an obtuse angle with the upper arm. In the case of the knee under like conditions (that is, when we are sitting on a high seat so that the foot clears the ground, when voluntary muscular action ceases, and when the leg is unsupported), this segment of the limb drops by its own weight till it is suspended by the tonic contraction of the quadriceps extensor and by the ligaments of the joint. In the hip the position of greatest ease is assumed when we sit, and voluntary action of the muscles of the lower limb ceases. By sitting down we flex the joint, while abduction and eversion are due to the weight of the limb, opposed, however, at a certain point, by tonic muscular contraction.

Thus far it is seen that when voluntary muscular action ceases, the joints are left in a relaxed condition, and that the position which they assume is mainly determined by two factors (a) the weight of the distal segment of the limb, opposed when a certain point is reached by (b) the tonic con-