painful and the seat of numbness and "pins and needles;" a certain amount of paresis and anaesthesia existed, signs suggesting neuritis,¹ which might be attributed to the long-continued use of morphine hypodermically. Constipation, always troublesome, was now almost insuperable. There was continual vomiting of bile-coloured fluid. At no time was ascites or oedema of the feet observed. The patient died on August 16th.

Permission to perform a *post-mortem* examination was refused.

The absence of ascites is not uncommon in cancer of the liver. According to Frerichs² it was present only in about half the number of his cases—18 out of 31. He quotes 60 cases recorded by other authors, in 30 of which ascites was present, in 19 it was absent, and in 11 cases no mention was made of this symptom. Jaundice also is often absent, its presence, like that of ascites, depending upon the situation of the new growths in the liver. "When only a few branches of the hepatic ducts are destroyed, or when the compression is incomplete," the icterus is slight,³ as in this case. Murchison asserts⁴ that if the ducts are not compressed, almost the whole of the secreting surface of the liver may be destroyed without jaundice. The disappearance of pain during the course of the illness is usual.⁵

Cancer of the liver is secondary four times out of five,⁶ and in many cases the primary seat of the disease is not discovered during life, but in this case the unconfirmed surmise was that the hepatic cancer was secondary to the tumours, probably intestinal, in the left lumbar region. The general characters of the hepatic enlargement also were not those of primary cancer, of the liver,⁷ consequently this case was looked upon as one of secondary carcinoma of the liver. Murchison⁸ says that primary cancer of the liver is "exceedingly rare" before the age of 35 or 40, but that secondary cancer may occur "at any age."⁹ Budd¹⁰ likewise says that secondary cancer may occur "at any age," but that it is "rare" under 30. Frerichs¹¹ observed only two cases of "cancer" under 30, these secondary to (1) eyeball and (2) testis. Leichtenstern, quoted by Langenbuch,¹² gives a table compiled from several authors dealing with this point, from which it is seen that 7.8 per cent. occur between 20 and 30, 12.9 per cent. between 30 and 40, 53 per cent. between 40 and 60, 19.3 per cent. between 60 and 70, 6.9 per cent. over 70.

According to Clifford Allbutt's *System of Medicine*,¹³ it is all but unknown under 20. A case of primary cancer has been reported in a boy aged 12 years,¹⁴ but doubt has been thrown upon it.¹⁵ It is remarkable and unfortunate that the authors who have written upon this point do not discriminate, where it is possible, between sarcomatous and carcinomatous growths of the liver. One is not so surprised at the occurrence of sarcoma running an acute course in childhood or youth, but the frequency in young persons of carcinoma of the liver, primary or secondary, must be less marked than the foregoing tables would lead us to believe.

NOTES AND REFERENCES.

- ¹ Frerichs, *Clinical Treatise on Diseases of the Liver*, New Sydenham Society translation, 1861, p. 373, narrates a case of cancer of the liver, with "paralysis of the right arm," etc. ² *Loc. cit.*, p. 303. ³ Frerichs, *loc. cit.*, p. 303. ⁴ Murchison, *Diseases of Liver*, 1868, p. 190. ⁵ Frerichs, *loc. cit.*, p. 302. ⁶ Leichtenstern, quoted by Langenbuch, *Chirurgie der Leber und Gallenblase*, 2 Hefte; Stuttgart, 1897, p. 52. ⁷ Langenbuch, *loc. cit.*, p. 52, etc. ⁸ *Loc. cit.*, p. 192. ⁹ All cases reported in his book are over 30. ¹⁰ Budd's *Diseases of Liver*, 1852, p. 391. ¹¹ *Loc. cit.*, p. 299. ¹² *Loc. cit.*, p. 58. ¹³ Vol. iv, p. 237. ¹⁴ Pye-Smith, *Pathological Transactions*, vol. xxxi, p. 125. ¹⁵ Clifford Allbutt's *System of Medicine*, vol. iv, p. 206.

REMARKS ON ANEURYSM OF THE CORONARY ARTERIES OF THE HEART;

WITH NOTES OF TWO CASES.*

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ANEURYSMAL dilatations in the course of the coronary arteries of the heart are so uncommon that the experience which fell to my lot lately of seeing two examples in two successive *post-mortem* examinations in one afternoon must, I should think, be unique. These two examples I shall describe, and any remarks I have to make afterwards will spring chiefly from their consideration.

* Read before the Leeds and West Riding Medico-Chirurgical Society.

CASE I.

F. S., a young woman, aged 19, was admitted on July 18th, 1900, when I was on temporary duty during the absence of Dr. Chadwick.

History.—She gave a history of rheumatic pains in both knees twelve months previously, which soon disappeared under treatment. Symptoms of nephritis supervened about nine months afterwards. Mitral disease, with right and left-sided hypertrophy, was diagnosed; there was albuminuria and hæmaturia, but no casts in the urine. Shortly after admission symptoms of acute endocarditis became very obvious, and she died on August 31st.

Necropsy.—At the necropsy the pericardial sac was found to contain about 3 ounces of clear fluid. The heart, much enlarged, weighed 15 ounces. The mitral orifice admitted the passage of three fingers: its segments and chordæ tendineæ were much thickened. Superadded to this, which was manifestly an old lesion, was a recent vegetative endocarditis of the usual type affecting the auricular aspect of the segments near their free edges and extending up the posterior wall of the auricle. The aortic valve also showed the changes produced by slight vegetative endocarditis. A small aneurysm was found on that branch of the right coronary artery which passes down the posterior interventricular groove. It was situated two inches from the auriculo-ventricular furrow, was about the size of a small pea, and had tough fibrous walls. The opening into the sac, which had smooth margins and would readily admit the passage of a crow quill, was situated at a point where a branch came off, and in such a way that the wall of the sac was formed partly by the parent stem and partly by this branch. The continuation of the main artery was pervious, while the branch became impervious shortly beyond the sac, though no plug was found. Elsewhere the coronary arteries were quite free from disease, being soft, smooth, and elastic. The spleen and the kidneys did not present any evidences of infarction; the latter were good examples of the large white kidney.

The second case presented many features of marked similarity with that just described.

CASE II.

History.—A. M., a woman aged 45, had had rheumatism when 14 years old, and Dr. Churton, whose patient she was, summed up the clinical condition at the time of her death as "old mitral disease with heart-muscle failure."

Necropsy.—At the necropsy 3 ounces of clear fluid were found in the pericardial sac. There was great cardiac enlargement, dilatation rather than hypertrophy predominating. Some clot, manifestly of *ante-mortem* formation, was found in the right auricular appendix. The mitral valve presented signs of old disease in great thickening of its flaps and chordæ tendineæ, while extensive recent changes, due to vegetative endocarditis, were found along the free margins of the segments and on the chordæ, and also extending up the posterior wall of the auricle to cover an area the size of a crown piece. A fringe of recent vegetations was found close to the free margin of the aortic segments. An aneurysm was found on the same branch of the right coronary artery as in the former case, but 1 inch nearer the auriculo-ventricular furrow. Its external dimensions were about those of an orange pip, its walls were thick, and it was almost filled with firm laminated, moderately adherent clot. Both coronary arteries presented patches of atheroma and were slightly tortuous, but the branch with the aneurysm was free from atheroma. On the margo obtusus was a firm scar in the substance of the heart, into which I traced a vessel which became impervious. The aorta was healthy. The spleen showed signs of old infarction, and the kidneys, which were markedly granular, had depressed areas, which I attribute to infarction from embolism of ancient date. The lungs were indurated from back pressure, and there was *ante-mortem* clotting of the blood in many branches of the pulmonary artery.

COLLECTED CASES.

Most of the textbooks on general medicine and on diseases of the heart do not refer to the subject of aneurysm of the coronary arteries. Dr. Walshe, in the fourth edition of his work, makes brief reference to it, as also does Dr. Cutler in Pepper's *System*. Sir R. Douglas Powell, in Reynolds's *System*, refers to examples as museum curiosities and mentions two specimens, while the same writer in Allbutt's *System* contents himself with saying that the secondary effects on the cardiac muscle of aneurysm of the coronary arteries are of less importance than those of thrombosis or embolism, a statement which is doubtless accurate.

I have therefore made a careful search through the literature of the subject, getting my references from many different sources, but endeavouring as far as possible, and in most cases with success, to get access to the original papers. This latter proceeding has led me to discard several of the examples mentioned in the primary references, which, in my opinion, should not strictly be included in the category of aneurysm of the coronary artery. I have thus thought it right to classify as doubtful cases three examples noted in Crisp's list, namely, his own case of dilatation of the orifices of the arteries, a condition which, as I have found it absolutely unassociated with any disease in the vascular wall, I do not regard as truly aneurysmal, but more allied to those dilatations of the pulmonary artery which are probably congenital, and Stevenson's and Ward's cases, which I regard as aneurysms of the sinus of Valsalva, involving the coronary artery. A specimen in St. Bartholomew's Hospital, formerly described as coronary aneurysm, has since been correctly de-

scribed by Thurnam as aneurysm of the sinus of Valsalva. Baly's case also is, on the authority of Duckworth, not put in the category of aneurysm, being more of the nature of fusiform dilatation, while Ogle's case (1864), which has a special interest of its own, I shall return to later.

TABLE I.—*Doubtful List.*

	Date.		Date.
Crisp	1848	Ward	1857
Barts	1849	Ogle	1864
Baly	1852	Stevenson	1871

In this way, including my own 2 examples, there remain of those I have got together 24 cases, the earliest of which was published by Bougon in 1812, and the latest by Jackson Clarke in 1896. Of 6 of these I can get but few particulars. Thus Dr. Ogle reports that the late Dr. Chadwick of Leeds said to him that he distinctly remembered having heard "of two cases of aneurysm of the coronary artery of the heart, one in the practice of one of his colleagues, and the other in or about the hospital," but he only spoke "from hearsay and memory." The remaining 4 cases are those of Finnell, Buchner, Shradly, and Wiegand.

TABLE III.—*Aneurysm of Coronary Arteries.*

	Date.		Date.
Bougon	1812	Chadwick (2 cases)...	1867
Weigand	1830	Ogle	1867
Peste	1843	Buchner	1867
Merat	1849	Bart's, H. (11 yrs.) ...	1871
Hedland	1849	Bart's, H. & Gee (7 yrs.)	1871
Peacock	1849	Crisp	1871
Heuse	1856	St. Thos. H.	1879
Finnell	1856	Malet and Evans ...	1887
Bristowe	1856	Clarke	1896
Wood	1860	Griffith	1900
Markoe	1860	Griffith	1900
Shradly	1860		

Before commenting on the remaining 18 cases, I ought to say that I have, in the course of my search, come upon 15 examples of rupture of the coronary artery without any mention of aneurysm, and in some cases with a distinct statement that there was no aneurysm. Possibly some of these may have nevertheless been due to small aneurysms, the rupture of which might obscure their existence, and lead to their being overlooked.

TABLE II.—*Rupture of Coronary Artery.*

	Date.		Date.
Kramer	1732	Youl	1872
Fischer	1740	Clark	1876*
Feigneaux	1859	Lilly	1882
Harlan	1860	Battenham	1887
Clark	1860	Humphry, L.	1898
Osborne	1862		

* Five cases.

Coming now to the 18 cases, I find that death from rupture into the pericardial sac occurred in exactly one half. In Bougon's case no less than 40 oz. of blood was found in the cavity. Dr. Ogle states, on the authority of Mr. Pick, that the capacity of the normal pericardial sac with the heart unremoved is 18 to 20 oz., and doubtless in such cases the death is due not so much to the loss of blood as to the pressure on the heart.

NUMBER AND SITUATION OF ANEURYSMS.

In 11 instances the aneurysm was solitary, occurring 4 times on the right and 4 times on the left artery, while in 3 instances

its situation is not noted. In the 7 instances in which the aneurysms were multiple, they varied in number from 3 up to 12. In only 2 instances is it noted that the aneurysm was in the auricular wall; in all the others it appears to have been on the main stem of the artery, or on the ventricular part of the heart, and usually on the latter. Some writers who mention the site state that the aneurysms were at or close to the points of origin of branches, a fact of some importance in relation to causation. In size they have varied from that of a chestnut to that of a small pea. In 14 instances the affection occurred in the male sex, and in 4 in the female. Of the 14 instances in which the age is noted only 5 were above the age of 26, and the ages varied from 5 to 77.

CAUSATION.

It is now a well recognised fact that embolism plays a very important part in the causation of aneurysm, especially in the case of those occurring on the smaller vessels of the body.

It is commonly held that Tufnell was the first to point out this connection, but by referring to the original papers I find that Dr. Percy Kidd was quite right when he said that Dr. Senhouse Kirkes had forestalled everyone in suggesting embolism as a cause of aneurysm. This he did in 1852, in a remarkable paper on embolism, which, falling as it did in point of time in the midst of Virchow's great work, entitles him to share with that observer the credit of putting our knowledge of embolism on a sound anatomical basis.

In making this suggestion Kirkes himself owes some of his inspiration to Dr. Burrows, for in discussing the changes occurring in embolic clots, he says:

Or, as suggested to me by Dr. Burrows, it may sometimes happen that the coats of the artery may, by the pressure behind, be sufficiently dilated around the clot to allow of the transit of some blood along the canal.

To Tufnell, who published his observations in 1853, and who refers to Kirkes's paper, is due the credit of being the first definitely to prove this connection in the case of a man with vegetative aortic disease, who developed a popliteal aneurysm while under his care.

I believe that the next observation was that of Ogle, who, in 1857, in commenting on a case of aneurysm of the superior mesenteric artery associated with recent fibrinous granulations on the valves of the heart, suggested the probability of the formation of aneurysms as a result of embolism. Again, in 1866, while still in ignorance of Tufnell's published views, Ogle contributed an able paper to the *Medical Times and Gazette* on this association. He mentions an experiment he performed, designed to elucidate the connection, which is interesting in its result. By passing a tube down the carotid artery of an ass, he injected portions of fibrin into the left ventricle of the heart. On examination of the cadaver 21 days afterwards an aneurysm was found on one of the branches of the superior mesenteric artery. So far so good, but, when the sac was opened two or three worms of the genus *strongylus* were found inside, a common cause, I believe, of aneurysm in the horse and ass.¹

Cases bearing on the subject were then contributed by Wilks, Pollock, Holmes, Church, Bryant, and others. Then came a paper by Ponfick in 1873, to whose conclusions I shall presently refer. In 1877 Goodhart published his lucid paper in the *Transactions* of the Pathological Society. This was followed ten years afterwards by Langton and Bowlby's article in the *Medico-Chirurgical Transactions*, and now, as I have said, the doctrine is generally accepted.

The exact way in which embolism gives rise to aneurysm has been discussed by various writers, and three of these views which call for comment are:

1. *The Retro-embolic view*, that is, the view that the artery will dilate behind the plug from an increase of pressure consequent on the obstruction. Against this it is urged that aneurysm behind the point of ligature on an artery is rare, and, when occurring, is usually due to inflammatory softening of the vessel, and one cannot help seeing that, if an increase of pressure did occur to any extent behind a ligature, any attempt to bring about the cure of an aneurysm by either Brador's or Wardrop's operation would not be likely to be attended with success. Although this view is referred to by Goodhart as Holmes's view, I find that it was entertained by Ogle as part of the explanation, that it was fully enunciated by Kirkes, and that Tufnell, while adopting it in his paper, was fully alive to the objection that dilatation above a

surgical ligature is not common, while both this observer and Holmes clearly show by their writings that they regard a weakened state of the arterial wall as essential to the retro-embolic causation of aneurysm.

2. *Mechanical Injury*.—Ponfick's view that the artery is mechanically injured at the point of impaction by calcareous spiculi may be the case in some rare instances, but it is clear that very few cases are capable of bearing this interpretation.

3. *Ulcerative Endocarditis*.—Goodhart's view finds most general acceptance. He points out that most embolic aneurysms are associated "not with a simple endocarditis, but with an ulcerating form of the disease, a very severe form, generally producing fever and septic conditions"; and he points out that the clot from such a part will "lead to acute softening of the arterial wall by inoculating it with its own inflammatory nature." This opinion of Goodhart's, published as it was in 1877, at a time that the part played by micro-organisms in disease was not so well understood as now, strikes me as being singularly astute.

It is true that the term "ulcerating endocarditis" has been found to be too narrow, as many cases, highly septic in nature, are more vegetative or fungating than ulcerating, and as Welch says:

There is every transition from ordinary warty endocarditis to the most malignant forms, and as the same species of micro-organisms may be found in the relatively benign as in the malignant cases, no single type of endocarditis is exclusively associated with these aneurysms:

while Moxon points out that in one and the same case of endocarditis some of the emboli may be followed by suppurative changes and some by the changes of ordinary infarction.

In harmony with this view the vessel dilates at the point of impaction and infection and not above it, and this removes any difficulty in accepting the embolic causation of aneurysm that might arise from the fact that as a rule the plug is not found in these cases. Ponfick, indeed, is said by Langton and Bowlby to be the only writer who has ever actually found the plug which had caused the aneurysm.² On the other hand, plugging of arteries by detached vegetations from the valves of the heart has been demonstrated again and again in cases where no aneurysm followed.

This is exactly what one would expect. A non-septic embolon plugs the artery without causing any softening of its wall, and without therefore producing any dilatation, or, in the case of septic emboli, death may occur before it has had time to do so, and on *post-mortem* examination the *corpus delicti* is found, while if a septic embolon causes inflammatory softening of the walls, it will, by causing an aneurysm, lead to a condition favourable to its own disintegration and removal, and it is doubtless this fact that has led many writers entirely to omit all reference to embolism as a cause of aneurysm.

It is interesting to note that prior to Goodhart's paper, Shaw, Callender, and Ogle had all associated embolism with a dilatation of the artery at the point of impaction.

It being generally accepted, therefore, that aneurysm, especially of small vessels, is frequently due to embolism, let us see how this affects the view we take of aneurysm of the coronary arteries. In other words, is embolism of these vessels common?

In this connection Sir R. Douglas Powell writes:

The situation of the vessels at the commencement of the aorta, the wide angle at which they leave the vessel, and the bulk and impetuosity of the blood current at this portion, are all conditions unfavourable to the passage of clot into these small side branches.

Now, before considering whether these speculations harmonise with the facts of the case, I would point out, first, that as regards the angle the coronary arteries come off, the renals also come off from the aorta at right angles, and, without doubt, embolism of these vessels is of common occurrence, and secondly, that as regards "the bulk and impetuosity of the blood current," one must not forget that between the ventricular contractions there must be comparatively little movement of the blood, in an onward direction, in the first part of the aorta. We no longer have an impetuous stream, with its well-known tendency to keep heavier particles in its central axis, but a somewhat stagnant pool, with a flow, determined by the elastic recoil of the artery, partly towards the periphery, and partly towards the coronary orifices, the aorta getting relief in both directions. Are we not also taught by physiologists that towards the end of the systole, eddies and

side currents develop around the free margin of the semi-lunars, eddies which may carry potential emboli towards the orifices of the coronary arteries?

Welch, while agreeing that thrombosis is much more common than embolism, differs strongly from Marie, who holds that not more than 1 or 2 cases of embolism of the coronary arteries reported are free from criticism. He calls attention to the remarkable fact that "the heart ranks next to the kidneys as the most frequent seat of abscesses following intravascular injection of the pyogenic staphylococci in rabbits," while metastatic abscesses in the heart are not uncommon in pyæmia.

Not only therefore is there no valid *a priori* reason against the probability of embolism of the coronary arteries, but many writers have recorded undoubted examples, namely, Virchow, Chiari, Hebb, Cutler, Welch, Hektoen, Kidd, Rolleston, and Ogle. I have read the original accounts in the case of the last 7 writers, and they are without doubt clear examples of embolism.

It is a very remarkable coincidence that the very next necropsy I made after the two, the results of which I have related, afforded me an opportunity of seeing recent embolism of both coronary arteries.

The patient was one of Dr. Barrs's, a girl of 12, who died with symptoms of infective endocarditis, with right hemiplegia. The necropsy revealed very extensive vegetative endocarditis of the usual type, with plugging of the auriculo-ventricular branch of the left coronary, 1 inch from the bifurcation of the main stem, and at the point of origin of a considerable branch, while the posterior interventricular branch of the right coronary presented an embolic plug at the point of origin of a twig. There was also plugging of the left middle cerebral artery, evidences of infarction of the spleen, and an aneurysm, doubtless embolic in origin, on the splenic artery.

Embolism of the coronary arteries, therefore, is not uncommon, though it is not mentioned by such careful writers as Dreschfeld or Osler in their articles on infective endocarditis. If the coronary arteries are not examined in all cases as a matter of routine, embolism should certainly be looked for in all cases of infective endocarditis. Cohnheim mentions it as a cause of death, in cases where no other adequate cause could be made out, and I note that Anstie, in publishing "a case of ulcerative endocarditis in which sudden death occurred in a manner difficult of explanation," but in which the pulse got quick, irregular, and thready, does not appear to have examined the coronaries.

APPLICATION OF EMBOLIC THEORY TO CORONARY ANEURYSM.

The first writer who definitely attributes coronary aneurysm to embolism is Ogle in his paper in the *St. George's Hospital Reports* for 1867, and the case he published in 1864 of recent plugging of a cerebellar artery and of a coronary, in a lad who died of acute infective endocarditis, and in which the coronary artery was much dilated at the part plugged, would seem to illustrate the very commencement of an aneurysm. This case, however, I have not included among my 18 undoubted examples:

Of these 18 there was distinct vegetative endocarditis in 4, namely, in those recorded by Bougon and Ogle, and in the 2 I have described this evening, and I think we can, with confidence, attribute the aneurysms to embolism in each instance, though it must be noted that, in Bougon's case, the patient was an old man, and the coronary arteries were studded with osseous plates.

In Peacock's case the aortic valves were diseased, but the coronary arteries were bad, and the cause is not here so manifest.

In Jackson Clarke's case, a girl aged 10, with about a dozen aneurysmal swellings, there was mitral disease, but no recent vegetations, and although the coronaries and their branches are noted as having their inner coats much thickened, I hold with Clarke that embolism is the most likely cause, as there were two extravasations of blood in the axilla and gluteal region, presumably due to embolic changes there.

In Heuse's and Crisp's cases the arteries are also mentioned as sound, and, though the latter patient was 63 years of age, the history of a copious hæmatemesis favours embolism of a gastric artery, and renders the embolic causation of the coronary aneurysm probable. I do not attach much importance to Crisp's expression of opinion that the case was traumatic, for he clearly had not the embolic causation of

aneurysm in his mind, as he quotes cases manifestly due to embolism without mentioning the points in favour of this.

In Peste's case, a man of 77, the arteries were diseased extensively, and the presence of a cyst in the corpus striatum would harmonise either with embolism or hæmorrhage.

Bristowe's case must have been embolic I think. The patient was only 22, the aneurysms were mainly at points of divisions of the arteries, which elsewhere were quite sound, and there was pulmonary apoplexy, while the cortex of the kidney is described as being "thickly studded with buff-coloured patches and with dark red-coloured masses which were shown pretty conclusively by microscopic examination to be due to hæmorrhage occurring at different times in different situations." This was written in 1856, and I think most of us would now attribute the appearances to embolism, and not as Dr. Bristowe did at that time to the "existence of partial arterial disease."

It is difficult to give any cause other than embolism for Gee's case, in which the patient was 7 years old, and died with scarlatina, dropsy, pneumonia, and meningitis, for Malet and Evans's case in which the child was only 5 years old, though in this instance the valves were noted as normal, or for the remaining Bartholomew's Hospital case, where the boy was only 11 years old.

In the cases of Markoe, Wood, Hedland, and Merat, I can find no facts on which to form an opinion, but in the St. Thomas's Hospital case there was pulmonary hæmorrhage and hæmorrhage into the kidneys, conditions which I think suggest embolism more forcibly than that suggested by Douglas Powell, namely, that the patient, who was only 22 years old, was the subject of general arterial disease.

I have intentionally refrained from any attempt to discuss the clinical phenomena of coronary aneurysm. The symptoms are, with the exception of those caused by rupture, chiefly those of the associated condition. The affection might be guessed at, but hardly, I think, diagnosed, and the two chief points I desire to emphasise [are, the rarity of its occurrence, and the probability of its sharing the peculiarity of aneurysms on small vessels elsewhere, in being embolic in origin.

NOTES.

¹ Coats states that 90 per cent. of horses and asses are the subject of verminous aneurysms. The worms (sclerostoma armatum, or equinum) are found in the large intestine, and from this they work into the vessels of the viscera, largely into the gastric and mesenteric. ² But Pollock, quoted by Holmes in his *System*, is also reported to have found the clot.

BIBLIOGRAPHY.

Anstie, *Path. Trans.*, xix, 52. Baly, *Path. Trans.*, 1852, vol. iii, 264. Bougon, *Bibliothèque Médicale*, 1812, 37. Bristowe, *Path. Trans.*, vol. vii, 98, 1856. Bryant, *Path. Trans.*, xxviii. Buchner, *Aneurysma der Arteria Coronaria Cordis Sinistra*, Amsterdam, 1867. Callender, *Path. Trans.*, vol. ix, 93. Chiari, *Prog. med. Woch.*, 1897, Nos. 6 and 7. Church, *St. Barth. Hosp. Reports*, vol. vi, 1870, 99. Clarke, J. Jackson, *Path. Trans.*, 1896, xlvii, 24. Cohnheim, *Textbook on Pathology*. Crisp, *Path. Trans.*, 1871, xxii, 106-111, with table. Cutler, *Pepper's System*, 1885, vol. iii, 830. Dreschfeld, *Articles on Endocarditis in Allbutt's System*, vols. i and v. Duckworth, Quoted by Ogle as to Baly's case. Fennell, *N. Y. J. Med.*, 1856, N.S., xvi, 82. Gee, *St. Barth. Hosp. Reports*, 1871, vol. vii, 148. Goodhart, *Path. Trans.*, vol. xxviii, 1877. Hebb, *Path. Trans.*, 1885, xxxvi, 140. Hedland, *Otto's Compendium of Patholog. Anatomy*. Heuse, *Presse Méd.*, 1856. Holmes, *Path. Trans.*, 1860, vol. xii, 61; *System of Surgery*, 1883, vol. i, 363, vol. iii, 29 and 30. Hektoen, *Medical News*, 1892, lxi, 210. Kidd, *BRITISH MEDICAL JOURNAL*, 1886, vol. ii, 1033; *Path. Trans.*, xxxvii. Kirkes, *Med.-Chir. Trans.*, 1852, xxxv, 281. Langton and Bowlby, *Med.-Chir. Trans.*, 1887, lxx, 117. Malet and Evans, *Lancet*, 1887, vol. ii, 67. Markoe, quoted by Ogle. Merat, *Dict. des Sciences Méd.*, v, 484. Moxon, *Path. Trans.*, xix. Ogle, J. W., *Path. Trans.*, 1857, vol. viii, 168; *Path. Trans.*, 1864, vol. xv; *Med. Times and Gazette*, 1866, vol. i, 196; *St. George's Hospital Reports*, 1867, vol. ii, 285; *Med. Press and Circular*, 1867. Peacock, *Path. Trans.*, vol. i, 227; *Monthly Journal of Med. Science*, 1849, vol. ix, 592. Peste, *Arch. Gén. de Méd.*, 1843. Pollock, *BRITISH MEDICAL JOURNAL*, 1886, vol. ii, 1033; in Holmes's *System*, vol. iii, 29. Ponick, *Virchow's Archiv*, vol. lvi. Powell, Reynolds's *System*, vol. v, 132; Allbutt's *System*, vol. v, 900. Rolleston, *BRITISH MEDICAL JOURNAL*, 1896, vol. ii, 1566. St. Thomas's case (Powell in Reynolds's *System*, vol. v, 132). Shady, *N. Y. Med. Press*, 1860, N. S., iii, 225; *N. Y. J. Med.*, 1860, 3 series, viii, 384-386. Shaw, *Path. Trans.*, vol. vi, 34. Stevenson, quoted by Crisp. Tuinell, *Dublin Quarterly Journal of Medical Sciences*, 1853, May. Ward, *Path. Trans.*, 1857, vol. viii. Walshe, *Diseases of the Heart*, 4th ed., 1873. Welch, Allbutt's *System*, vol. v, 208-209. Wiegand, *Med. Convers. Bl.*, Hildburgh, 1830, 217-222. Wilks, *Path. Trans.*, 1859, vol. xi, 44. Wood, quoted by Ogle. Osler, *Goulstonian Lectures*, 1885.

THE German Emperor has conferred the honour of hereditary nobility on Professor Emil Behring, Director of the Hygienic Institute of the University of Marburg, whose name is so well known in connection with serumtherapeutics. The Professor is henceforth entitled to put the *particule nobiliaire*, "von," before his distinguished name.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

TWO CASES OF VARICELLA.

CHICKEN-POX is generally regarded as so trivial an ailment that perhaps the accompanying charts of two recent cases may be of interest.

Chart 1.

Chart 2.

The cases are those of two brothers, aged 13½ and 12½ years respectively. They were taken ill on the same day, and exhi-