and he still regarded himself as before everything else a doctor." Hereafter questions of philosophy, finance, education, trade, theology, &c., occupied his busy life, but through it all, and to the very end, there are references in his letters and journals to show that his first love was not forgotten. In the memorable and oft-quoted letter to Dr. Thomas Molyneux in praise of Sydenham's method, Jan. 20th, 1692-93, he speaks of himself as one who wishes well to the practice of physic "though he meddles not with it," yet in the same year we find him prescribing for a friend's wife. In 1694 he is corresponding with Dr. Hans Sloane on medical matters. In January, 1697-98, King William consulted him, believing that they suffered with similar diseases. In 1701 he writes again to Sloane about a patient with an obstinate fever, and in the following year he gives very wise advice to his old friend Lomborch, who was ill. Evidently Locke's clear, strong judgment was valued by his friends in all relations of life, and as Somers and other politicians turned to him for instruction in questions of trade and finance so his friends and others insisted upon utilising his medical knowledge. Sydenham, Boyle, Thomas, Mapletoft, and Molyneux had been his intimate associates. In Montpellier, in Paris, and in Holland he had been a welcome guest in medical circles, and in London we have met him in consultation with the most eminent practitioners of the day upon a most important case and handing in a written opinion as their colleague and equal.

For each one of us there is still a "touch divine" in the life and writings of John Locke. A singularly attractive personality, with a sweet reasonableness of temper and a charming freedom from flaws and defects of character, he is an author whom, liking at the first acquaintance, we soon love as a friend. Perhaps the greatest, certainly, as Professor Fowler says, the most characteristic, English philosopher, we may claim Dr. Locke as a bright ornament of our profession, not so much for what he did in it, as for the methods which he inculcated and the influence which he exercised upon the English Hippocrates. He has a higher claim as a really great benefactor of humanity, one of the few who, as was so finely said of Isocrates, "reflected the human spirit always on the nobler side." One of Locke's earliest writings was a translation for Lady Shaftesbury of Pierre Nicole's Essays, in one of which, on the "Way of Preserving Peace with Men," Locke seems to have found a rule of life which I commend to you: "Live the best life you can, but live it so as not to give needless offence to others; do all you can to avoid the vices, follies, and weaknesses of your neighbours, but take no needless offence at their divergences from your ideal" (Fox-Bourne).

The Hunterian Lecture

ON

THE CLINICAL STUDY OF HEART DISEASE.

Delivered before the Hunterian Society on Oct. 10th, 1900,

By NORMAN MOORE, M.D. CANTAB.,
F.R.C.P. LOND.,

ASSISTANT PHYSICIAN TO, AND LECTURER ON MEDICINE AT,
ST. BARTHOLOMEW'S HOSPITAL.

GENTLEMEN,—The great man whom your society commemorates had made many observations on the vascular system in men and in animals, some of which are preserved in his interesting "Treatise on the Blood, Inflammation, and Gunshot Wounds," written in 1793. He describes a patient who from boyhood had suffered from shortness of breath, palpitation, attacks of venous engorgement, and irregular action of the heart, and who died from dropsy. Post mortem John Hunter found the aortic valves to be thick, hard, and contracted, and he remarks: "This diseased structure of the valves accounts for every one of what may be called his original symptoms, and was such as to render them [i.e., the valves] of very little use; the blood therefore must have fallen back into the cavity of the ventricle again

at every systole of the artery." He discusses whether this state of the valves was a natural formation or a disease, and points out the wide effect on the circulation of such a condition. "It may be difficult," says Hunter, "to account for the increased size of the heart, whether it was a mechanical effect, as the blood would be thrown back into it at every systole of the aorta and diastole of the heart, or whether it arose from a particular affection of that viscus. The first idea is the more natural, but it is not necessary that the cause should be of this kind, for we see every day enlarged hearts where the symptoms have been somewhat similar, and yet no visible mechanical cause existed, and indeed it is a common effect where there is an impeded circulation." The account shows us how far a case of heart disease was understood by Hunter, and his views may be taken as the most advanced of his time. He had perceived how valvular disease led to palpitation, shortness of breath, venous engorgement, and dropsy, and he suspected, but was not sure of, the causal relation of aortic valvular disease to hypertrophy of the left ventricle. He was clearly not familiar with affection of the aortic valves as a very common disease; he does not seem to have considered whether the mitral orifice admitted regurgitation, as it very likely did in such a case and in some of the other examples of hypertrophy which he mentions; he knew nothing of the association of renal disease with hypertrophy of the left ventricle; and of course he was unacquainted with the method of ascertaining valvular disease by auscultation of the heart.

Hunter gave all the time at his command to the original observation of nature and did not pay much attention to the books of the past or of his own time. He had probably never read the account of an enlarged heart in which extensive disease of the aortic valves was found post-mortem which Dr. James Douglas had given in the "Philosophical Transactions" for 1716. Douglas had heard during the patient's life a loud sound proceeding from the cardiac area, the systolic murmur of aortic stenosis with which we are all perfectly familiar. Had Hunter read this observation and considered it in relation to his own observation on disease of the aortic valves it does not seem improbable that he might have anticipated part of Laennec's subsequent discovery and have initiated the auscultation of the heart and elucidated the meaning of valvular murmurs. Douglas has not, I think, received sufficient honour for his remarkable observation. He was a man of great ability, great industry, acute observation, and extensive learning. He died in 1742 and left behind him numerous medical, pathological, and anatomical observations¹ of a high order, some botanical works of interest² and an admirable bibliography and summary of all anatomical writers from Hippocrates to Harvey.³ He merits the honourable commemoration which he receives whenever the fold of Douglas is mentioned. The human body, a nobler structure than any temple made with hands, is a kind of Westminster Abbey for the first centuries of anatomists. There Willis, Fallopius, Malpighi, Tulpius, Lower, Wharton, and Glisson all have their monuments, more suitable to their work and more to their own taste than any "storied urn or animated bust." Douglas well deserves his place in that temple of fame. With regard to diseases of the heart we feel that we know a great deal more than he did, much more even than John Hunter did. Vast have been the additions to knowledge in that subject since Hunter's century. Dr. Mead was the most famous physician of London in Hunter's childhood, and in his medical writings all he has to say about heart disease fills only two small pages. We have at our disposal works on heart disease the titles alone of which fill 92 large pages with double columns of the catalogue of the Surgeon-General's Library at Washington, that admirable aid to study which we owe to the organising skill and indefatigable industry of Dr. Billings. Notwithstanding this vast accumulation of knowledge in the hands of the medical profession of to-day, difficulties still exist in relation to heart disease and errors are committed which we should all wish to avoid. Much as is known many cases are hard to make out and difficult to treat.

I propose to discuss a few of the questions which from

¹ Philosophical Transactions, Nos. 345 to 350, vols. xxv., xxvii., xxix., xxxii. Myographiæ Comparatæ Specimen, 1707. Description of the Peritoneum, 1730.

² Liliū Sarniense, 1725. Arbor Yemensis fructum Cofe ferens, 1727.

³ Bibliographiæ Anatomicæ Specimen, 1715.

time to time arise in the consideration of disease of the heart. A few rules with regard to the physical examination will save many mistakes. The first is, in writing notes of a case to make certain precisely what is heard or seen; to go on listening till certainty is arrived at with regard to the quality and distribution of every sound heard; and never to mix this observation with hypothesis as to the cause of what we observe. A note on a case of valvular disease should state the time, the quality, the place of maximum intensity and the distribution of the murmur. When these are precisely determined the conclusion may be added. Such expressions as "a mitral regurgitant murmur," or "an aortic obstructive murmur," should never be used in the original note on a case. The observations as to time and sound may be precise and perfect for each observer, but further circumstances may have been overlooked which may vitiate the conclusion he draws. A second independent observation may add more facts and so point to another conclusion. The mixture, at the beginning, of what is observed and what is inferred often prevents the correction of an error. It is wise when seeing a case of valvular disease for the second time not to look at the original note but to record an absolutely fresh observation and then to compare the two. A large experience will convince anyone that there are possibilities of error in relation to valvular disease which can only be avoided by great care in recording what we hear, by numerous observations, and by comparison of several observations. The habit of determining exactly what we hear should be cultivated and all doubtful or doubting expressions avoided so far as the evidence of the senses is concerned. Another practice which saves many mistakes is the careful observation of the pulse and comparison of its results with those of the auscultation. In examining a case of valvular disease it is not too much to feel the pulse with close attention throughout three complete and separate minutes, noting the number of the beat in the minute in which any inequality or other peculiarity is observed. This method, if pursued with regard to irregular action, affords an exact measure of the increase or diminution of the irregularity from day to day or from week to week.

Murmurs are, of course, sometimes very faint, barely audible. The inhalation of a capsule of nitrite of amyl will often be immediately followed by a marked increase in the sound of a murmur and is a much safer method of procedure than any violent muscular exertion.

One of the first points in the examination of a heart is to determine whether hypertrophy is present or not. The position of the apex beat and the character of the impulse are the most important guides. Too much care cannot be taken; the best observers have sometimes failed to distinguish between hypertrophy and pericardial effusion. I once witnessed a melancholy example of this difficulty which impressed me deeply. A physician of great experience after a careful examination decided that a man sitting up in bed with embarrassed breathing and irregular action of the heart had a large pericardial effusion. A surgeon was asked to puncture the pericardium with a trocar. He did so, no fluid came out, and the patient died immediately. I examined him post mortem. There had been no pericardial effusion, there was great hypertrophy, the trocar had penetrated the heart wall, and a fatal hæmorrhage into the pericardium was the result. Both the physician and the surgeon have long been dead. Both were men of the most careful habits of procedure. The surgical method was that of its time; the physician was a man of well-deserved reputation for skill in auscultation and percussion. His error demonstrated the occasional difficulty of being certain about the presence of hypertrophy of the heart.

Hypertrophy once determined must be explained. We must rigidly clear our minds from any idea that hypertrophy is a normal variation in the heart due to muscularity of the body. The idea is still prevalent that big men have a condition of heart resembling hypertrophy. Professor Haughton evidently held this view. In his ingenious writings on animal mechanism he suggests that Master Magrath, the celebrated greyhound, had a left ventricle of unusual size because of his great muscular development. I believe that there are good grounds for the opinion that this greyhound and others have had large and truly hypertrophied left ventricles because they were the subjects of chronic interstitial nephritis. Hypertrophy of the heart has always a morbid explanation. If it is an hypertrophy of the left side it means that valvular disease, chronic interstitial nephritis, or adherent pericardium is present. Aneurysm by itself is never a cause of hypertrophy.

When hypertrophy of the left ventricle is determined the next step is to decide which of these three causes has produced it. The largest heart, the *cor bovinum* of old writers, weighing from 24 to 36 ounces, is always due either to disease of the aortic valves or to chronic interstitial nephritis. The largest heart I have myself seen due to mitral regurgitation alone weighed 22 ounces. In few cases of hypertrophy due to mitral regurgitation does the weight of the heart reach 20 ounces; from 15 to 17 ounces is a common weight.

It would be useful if we could correlate each particular position of displaced apex with a definite degree of hypertrophy as indicated by increased weight. My own observations in the post-mortem room of cases examined during life do not entitle me to go further than the statement that when the apex beat is in the nipple line and sixth interspace the heart may be of double its natural weight—that is, it may in a man weigh 20 ounces. This observation is at least of use that it may impress upon us the considerable degree of any hypertrophy in which the apex beat is in the sixth space and nipple line.

The presence of dilatation as distinct from, or as associated with, hypertrophy may be inferred from symptoms but is rarely capable of accurate demonstration from physical signs alone. I know from my own observation that dilatation may be rapid, for I have seen a case of extreme dilatation of the right ventricle in a child who died after six weeks of whooping-cough at the beginning of which the heart was undoubtedly normal. In most cases of long-standing chlorosis, since the heart's action is feeble and anasarca of the ankles is common, I believe that some dilatation of the heart exists and I incline to the belief that the cardiac murmur in these cases is often, if not always, associated with a slight degree of mitral regurgitation due to the condition of the heart-wall and the muscoli papillares of the valve.

To return to cases of undoubted valvular disease. The cardiac impulse is sometimes accompanied by a thrill and we try to determine whether this is due to mitral stenosis, to extreme rigidity of the aortic valves, to pericardial causes, or to an aortic aneurysm. The thrill produced by mitral stenosis is never felt except at the apex, but the thrill of rigid aortic valves may be felt at the apex as well as at the base and is sometimes to be felt at the apex and not at the base. The character of the pulse will in such cases protect the observer from error. The physical explanation may perhaps depend upon the affection of the aortic cusp nearest to the ascending flap of the mitral valve, but I have not proved this from post-mortem experience. With this may be mentioned the fact that the systolic murmur of aortic obstruction, and the diastolic murmur of aortic regurgitation, and the double murmur caused when both defects are present, are all occasionally distinct at the apex and more rarely are inaudible at the base and audible at the apex only. The murmur of mitral stenosis is never audible at the right base. The murmur of aortic stenosis probably attains the loudest sound of all murmurs since it is the only murmur which I have ever heard distinctly at a distance of six inches and without contact with the patient's chest. The late Dr. Andrew maintained that the systolic murmur of mitral regurgitation was always to be heard at the angle of the left scapula and that it might thus be distinguished from the murmur of anæmia, but Dr. A. E. Garrod has shown that the murmur of anæmia is sometimes audible at the angle of the scapula. My own observation is that any systolic murmur which is distinct at the apex may be heard at the angle of the left scapula, but that if you take all the systolic murmurs heard at the apex and at the angle of the scapula a large majority of them will prove to be cases of mitral regurgitation.

The diastolic or presystolic murmur of mitral stenosis is never heard at the back. The careful auscultation of the back is important especially in relation to the separation of a group of murmurs in the same heart. If a patient has mitral regurgitation and aortic stenosis the systolic murmurs produced at the mitral and aortic orifices may thus be distinguished in many cases but not in all. On the back the mitral murmur will be loudest at the angle of the left scapula, the aortic murmur will often be well marked in the right supra-spinous fossa. If this systolic murmur be traced down towards the angle of the left scapula it will be found to grow fainter and then when the angle of the scapula is reached to be again loud. The murmur of aortic stenosis is generally audible in the right supra-spinous fossa and the diastolic murmur of aortic regurgitation is sometimes, but much less frequently, audible there too. It is sometimes

impossible to be quite sure that both mitral stenosis and aortic regurgitation are present. A distinct thrill confined to the apex will in many cases prove that the presystolic or diastolic sound heard at the apex is due to mitral stenosis, while an accompanying diastolic murmur heard at the right base is never due to mitral stenosis and is proof of the presence of aortic regurgitation. The same method of distinction will apply if there be both affections, stenosis and regurgitation, at each valve. Some weight may be attached to the observation that the murmur seems to be produced close to the applied end of the stethoscope. This will sometimes make one quite sure that a given sound belongs to the aortic valves. In such complicated cases an apex thrill may be safely assumed to be due to mitral stenosis if there be a distinct diastolic murmur and no thrill at the right base. If numerous observations are made the cases which cannot be fully worked out are very few.

Apart from congenital conditions diseases of the valves of the right heart are of course rare. I have only seen post mortem one case of tricuspid stenosis in which stenosis of the mitral valve was not present. Tricuspid regurgitation is always accompanied by well-marked backward flow in the veins of the neck, and no fancied localisation of a systolic murmur will justify this diagnosis if this movement in the veins of the neck be not present. I have seen three cases of stenosis of the pulmonary valves without any congenital malformation or other valvular disease. The characteristic features of such cases are that the systolic murmur is loudest at the left base, and that while on listening some uncertainty may be felt as to whether the sound may come from the aortic or from the pulmonary orifice the fact that the pulse is normal and has none of the characters of disease of the aortic valves or of the inequality common in mitral disease is the decisive observation. If this be repeated several times with the same result the diagnosis of pulmonary stenosis, rare as the disease is, may be made with confidence.

It is sometimes very difficult where disease of the mitral valve has begun in early childhood to distinguish it from the commonest congenital malformation, pulmonary obstruction with a small opening at the upper part of the septum of the ventricles. A careful and repeated examination of the pulse is often the best method of solution. Children with mitral disease usually develop better, though their growth is often retarded, than those with the congenital malformation.

One of the most difficult points to determine in relation to the physical examination of the heart is whether or no there is an adherent pericardium. In my opinion this diagnosis can be made with absolute certainty in only one case, and that is where mitral stenosis without other valvular lesion is present. This form of valvular disease is never on its own account associated with hypertrophy of the left ventricle. If, therefore, with mitral stenosis the left ventricle is hypertrophied and if there are no signs of chronic interstitial nephritis then the hypertrophy is certain to be due to an adherent pericardium. This is a rare association of changes and in every other condition the diagnosis of adherent pericardium is beset with difficulties.

Of all forms of valvular disease in the young mitral regurgitation is the commonest and it is also the one in which the possibilities of prognosis are best. The degree of improvement is often extraordinary. The patients in all cases of valvular disease which has begun in childhood and has continued into manhood run great risk of dying from pericarditis in addition to that of heart failure with dropsy, but if abundant rest and great care as to avoidance of probable causes of acute rheumatism and of over physical exertion be taken I have known such a patient grow well enough to enter the army and to do much active service in several climates.

In general relation to valvular disease in the young I think that we ought to believe that every case of rheumatic fever is a case of endocarditis and that we ought to take a child's temperature four times daily if possible for some weeks after such an attack and treat the slightest rise of temperature as if it were a fresh attack. By such care much serious permanent valvular damage would be avoided.

In the degenerative forms of valvular disease extreme care is well repaid (care as to nutrition as well as with regard to over-exertion), and the long duration of life of such patients with aortic disease often demonstrates this.

When angina pectoris is present, even if on a particular examination a murmur be not discovered disease of the aortic valves is almost always present. Now and then a

severe cardiac pain felt towards the apex occurs in mitral disease. The attack lasts longer than true angina pectoris. It may occur without any exertion. I once thought that it might be pericardial, but I am now satisfied that this is not so, and that it is probably a pain originating in the wall of the left ventricle.

Apart from attacks of angina pectoris mitral stenosis is of all forms of valvular disease the most distressing to the patient. The entire absence of such distress will sometimes make it clear that aortic regurgitation and not mitral stenosis was the cause of a thrill and diastolic murmur. In mitral stenosis engorgement of the liver takes place early and often causes much pain. Bleeding from the arm before a painful degree of hepatic engorgement has taken place often makes the action of the heart less embarrassed and greatly relieves the patient and after considerable engorgement of the liver has set in some relief may be obtained by leeches applied over the region of the liver.

The questions which I have discussed in this lecture are probably all familiar to everyone present, a fact which illustrates the vast increase of knowledge of heart disease since the time of John Hunter. He and his illustrious relative Dr. Matthew Baillie seem to perceive indistinctly and afar off the subject of valvular disease which we are now able to discuss in elaborate detail. They seem to have only a dim perception of what we see quite clearly, so true is the remark of Biot that nothing is so easy as the discovery of yesterday, nothing more difficult than the discovery of to-morrow.

THREE CASES OF GASTROTOMY FOR HÆMATEMESIS.¹

By C. W. MANSELL MOULLIN, M.D. OXON.,
F.R.C.S. ENG.,

SURGEON TO, AND LECTURER ON SURGERY AT, THE LONDON
HOSPITAL, ETC.

CASE 1.—This was the case of a woman, 42 years of age, who was admitted into the London Hospital under the care of my colleague, Dr. A. E. Sansom, on Oct. 27th, 1899, suffering from recurrent hæmatemesis. The first attack was six years before, and it was followed by other attacks at intervals of about six months, until 18 months before her admission into the hospital. Then there was a longer interval; but early in September, 1899, the hæmatemesis occurred again, and since that date there were two attacks, one on Oct. 23rd and another on Oct. 27th. The vomiting came on, as a rule, two hours after food, and it was followed by severe pain, which at one time used only to last for an hour or so, but latterly had been almost continuous. The epigastrium was tender on palpation, but not superficially. The patient complained when standing of a feeling as of a heavy weight in the stomach and of a dragging pain below the angle of the left scapula. Swallowing was followed by a burning sensation at the pit of the stomach, as if the stomach was raw. The patient was the mother of 11 children, six of whom were living. In spite of all the blood she had lost she was not markedly anæmic, and there was nothing worthy of note in her history or general condition except that during the previous 11 weeks she had lost 15 pounds in weight. In hospital the patient at first improved very considerably, the vomiting and the pain ceased, and she was much more comfortable, but as soon as the diet was increased the vomiting recommenced and the stools became black. As there was no evidence of permanent improvement Dr. Sansom very kindly transferred the case to my care for operation. After the ordinary preparation the abdomen was opened in the middle line above the umbilicus and the anterior surface of the stomach was examined. There were no adhesions nor was there any cicatrix. An opening was then made in the great omentum and the posterior surface of the stomach was explored with a similar result. The stomach was then drawn into the wound and was packed round with sponges. A vertical incision was made in the anterior surface about midway between the two ends and the edges of the stomach wound were held apart by means of four pairs of pressure forceps. Through

¹ A paper read before the Clinical Society of London on Oct. 12th 1900.