Observations on some points in dextral valvular disease of the heart / by Alexander Morison.

DEXTRAL VALVULAR DISEASE OF THE HEART.

BY

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OBSERVATIONS

ON SOME POINTS IN

DEXTRAL VALVULAR DISEASE OF THE HEART.

Being a Graduation Thesis, for which a Gold Medal was awarded by the Senate of the University of Edinburgh on the 1st *of August* 1878.

BY

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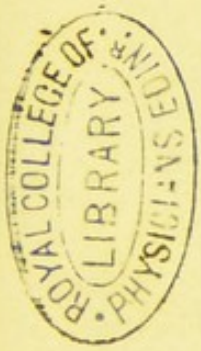

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Dedicated

TO THE MEMBERS OF THE

(NORTH LONDON) ÆSCULAPIAN CLUB,

BY THEIR FELLOW-WORKER,

THE AUTHOR.

DEXTRAL VALVULAR DISEASE OF THE HEART.

Introductory.

The recent legal restrictions imposed on the practice of vivisection are doubtless to be deprecated, on account of their limiting the number of scientific experiments, by reducing to a mere handful the number of experimenters, and also from their robbing clinical research of such corroborative or confuting physical evidence as one might otherwise seek for. It can, however, scarcely be denied, that thus restricting the area of this branch of biological investigation, and leaving it in the hands of a few physicists, is calculated to give a fresh impulse to clinical research, the proper domain of the physician, as we now understand this term; and, at the same time, to direct energies, which might otherwise be devoted to the prosecution of experimental studies, to the more minute and careful observation of clinical facts. From thus directing greater attention to clinical work, the restrictions we have adverted to will necessarily increase the number of clinical workers, generally, and by more equally matching the great body of the profession with those who have hitherto occupied the place of pioneers in the science and practice of medicine, are calculated to urge into the honourable race for professional distinction many who might otherwise, feeling themselves hopelessly overmatched, avoid the contest.

The arduous and multifarious duties of a general practitioner, from the time necessarily consumed by them, and the confusing impetus given to the mind in many different directions, interfere very much with the steady prosecution of any particular line of study. General practice is, however, not only a wide and varied, but also a rich field for scientific work, which might be utilized by a combination of general practitioners, intimate with one another, who, from their education and professional enthusiasm, would consent to act as colleagues in the prosecution of scientific inquiries, and to submerge for the common good the envious rivalry of traders in physic. I feel persuaded that such a combination among personal friends would not in any way interfere with pecuniary interests, would raise the professional status of the general practitioner, and be a powerful agent in promoting the progress of medicine and surgery, as the combined practices of several would afford as much material

as many a hospital, while the division of labour would utilize time to the utmost.

If, in the following pages, I should appear to have gone too minutely into the details of the cases recorded, I must plead a conviction that such minuteness is as desirable in the physician as in the physicist or physiologist, while for any deficiencies, such as the non-employment of the sphygmograph, which might have rendered valuable aid in the inquiry, I would urge the limited amount of time at the command of "the busy practitioner," as he at present exists. In the pathological, physiological, and therapeutic portions of the essay alike, I may be pardoned for thinking that prominence has been given to some topics not hitherto specially discussed (so far as I know), such as the comparison instituted between the circulation of a quadruped and a biped, the use of digitalis in organic valvular disease of the *venous* heart, etc.

Finally, the illustrations, with the exception of the sketch of the heart of E. W. G. M., which I made shortly after the necropsy, are, I need scarcely say, wholly or half diagrammatic.

Part I.

"There is no class of diseases which submits so difficultly to the trammels of nosological arrangement as the chronic affections of the heart. Their characters are so ill defined, so difficult is it to distinguish the idiopathic affections of this organ from those cases in which its functions are sympathetically disturbed, so impossible to anticipate with certainty, by the symptoms, the presence of structural disease there—in fine, so intimately are the functional disorders of the heart connected with those of the brain, that an attempt to arrange systematically this class of diseases may be considered as almost hopeless."[1]

So wrote Dr George Gregory of chronic affections of the heart in general, fifty-three years ago. Happily for suffering humanity, the lugubrious prospect entertained by him has not been justified by the scientific progress of the last half century, and failure to diagnose the vast majority of cases of diseased heart, which an accomplished physician at one time regarded as inevitable, would probably, at the present day, refer a tyro to his studies till he had learned to overcome a difficulty which is scarcely acknowledged to exist.

While the comparative frequency of valvular disease of the left chambers of the heart, and its careful investigation by physical processes, has resulted in a knowledge of the diagnosis of such diseases which is well-nigh perfect, the rarity of similar disease affecting the valves of the right side of the heart has naturally retarded our progress in the diagnosis and treatment of the latter—so much so, indeed, that one of the sites

[1] *Elements of the Theory and Practice of Physic*, by George Gregory, M.D., 1825, p. 445.

(pulmonary area) in which its diagnosis is possible, has gained the *soubriquet* of the "region of romance." The possible sacrifice or shortening of an occasional life, from imperfect knowledge of this kind of disease, is treated by some in their writings with contemptuous indifference, when the rarity of such cases is considered a valid preliminary to ignoring their discussion; others, who treat the subject with more consideration, confess a want of clinical experience, satisfactorily verified by post-mortem examination; and by none, so far as I know, has the matter been thoroughly investigated with a view to discover whether the physiological and clinical considerations differ sufficiently from analogous conditions in the arterial half of the organ, to necessitate a difference in the treatment of disease specially affecting the valves of the right heart.

The title of this essay indicates its scope. While avoiding any attempt at an exhaustive treatise, which is precluded by our comparatively limited knowledge, I have endeavoured to point out some "considerations," not usually discussed, which appear to have an influence upon the scientific treatment of such disease. The circumstance which has prompted my investigation of this somewhat uncultivated field of inquiry, has been my study of a remarkable instance of disease affecting the pulmonary and tricuspid valves, and should I be enabled, in the opinion of my compeers, to cast any light upon the therapeutics of these rare maladies, and thus mayhap contribute to the relief of one sufferer in a thousand, I shall (to borrow a stereotyped phrase from many a preface) consider myself amply rewarded.

The case I have referred to was communicated to the Pathological Society of London on the 7th of December 1875, and my paper, together with the report of the Morbid Growths Committee of that Society, confirming the accuracy of my description of the specimen exhibited, and agreeing with me in the general conclusions of my paper, are published in vol. xxvii. of the Transactions of the Society. So much of that paper as bears upon the argument of this thesis I shall transcribe from the above publication. I shall then consider some physiological and pathological conditions calculated to elucidate valvular disease of the right heart generally; and, finally, discuss the therapeutic conclusions derivable from the context.

OBSTRUCTIVE AND REGURGITANT DISEASE OF THE PULMONARY VALVES; OBSTRUCTION OF THE RIGHT AURICULO-VENTRICULAR ORIFICE; DISEASE OF THE TRICUSPID VALVES ALLOWING REGURGITATION, ETC., ETC.

Mr E. W. G. M., aged 20 years and 9 months, a bank-clerk, single, and of temperate habits, came under my care on 20th May 1875, complaining of breathlessness both when active and when in bed, with occasional attacks of severe pain over the heart, passing up to the right shoulder and down the right arm, being

particularly felt at the tip of the right little finger. He had also frequently experienced pain of a most acute and continuous character about the lower end of the sternum, which lasted on different occasions from half an hour to four or five hours, and was not influenced by food or drink. In bed he was most free from uneasiness when lying on the right side, with the head bent forward, and should he when asleep have lain on his left side, the heart pain awakened him. When lying on the back the head was usually turned to one side or the other. He thought that, on the whole, he was less embarrassed when sitting up than when lying down. He had also experienced a feeling of irritation about the nostrils, with a choking sensation, and during the last two or three months of life had occasionally slight attacks of epistaxis. For a year his feet were sometimes swollen across the instep towards night, but no trace of such remained next morning. He suffered much from general weakness, and occasionally had what he called fainting fits. When five or six years old the patient suffered from scarlatina, which was said not to have been severe, and to have left no perceptibly evil results. As a child he had frequent attacks of croup (rheumatic?) but there is no definite information regarding any other disease of childhood. When eight years of age he was examined by a medical man for admission to an educational charity, and was found to have heart disease, though such a condition was quite unsuspected before examination. At the age of twelve he ran a race, fainted, and became "cold all over." Animation was with difficulty restored, but next morning he did not appear much the worse of the accident. Except on this occasion he betrayed no sign of delicacy, and, but that his relatives were aware of his having heart disease, he was considered healthy till the age of fourteen, when he left home and entered into business. Though frequently overworked and exposed to cold, he continued in fair health for four years, having, however, on two or three occasions had severe "pain in the side," for which a doctor attended him. When eighteen years of age a medical man gave him permission to play cricket, and to this exercise he and his relatives ascribed his gradual decline in health.

Increasing dyspnœa and loss of strength forced him to abandon work about a year before death, since which his previous symptoms have progressively become more serious. He had never suffered from acute rheumatism. The patient's father died insane, and his mother of "consumption." Two paternal uncles and a paternal aunt died of heart disease between the ages of thirty-two and sixty. A paternal first cousin, who, with four or five brothers and sisters, has had rheumatic fever, suffers from heart disease. One maternal uncle died of heart disease, having had rheumatic fever, and another has had pneumonia. He has a brother and sister who have not had any serious illness.

The patient himself was fairly grown, but emaciated; with gray-blue eyes, light brown hair, a pale, somewhat livid countenance,

and anxious expression, and when first seen, a patch of purpura over the left malar prominence. The pulse, when lying, beat 78–84 in the minute, was regular, small, but distinct; both pulses were equal. There was well-marked pulsation in the supra-sternal fossa, and on pressure behind the notch a distinct systolic impulse was communicated to the fingers. This visible pulsation, though chiefly systolic, had an undulatory character, as if partly diastolic. There was distinct but not excessive regurgitation into both external jugulars. The carotid pulse was normal, though, from the emaciation of the patient, somewhat more apparent than usual. The præcordial region to the left of the sternum was uniformly bulged forward, and the left sub-clavicular region was sunken, apparently owing, in a measure, to a somewhat greater prominence of the left than of the right clavicle. The apex beat was diffused and its impulse weakened. There was visible systolic pulsation from the second left intercostal space to below the nipple, most distinct in the second and third left spaces, and again, in a limited area, immediately above the nipple, but at the latter point the heart's impulse was not more distinctly felt than elsewhere. There was well-marked pulsation in the epigastrium.

On laying the hand over the præcordial region, and especially over the second and third left spaces, a harsh systolic and diastolic "frémissement cataire" was felt. The heart's dulness was increased transversely, and ran with great distinctness into the liver dulness. No increase of dulness from above downwards was detected. On auscultation to the inner side, and below the left nipple, close to the left edge of the sternum, there was heard a distinct systolic bellows murmur, lost on tracing it outwards towards the left chest. On continuing to auscultate along the left edge of the sternum a double murmur also became audible, increasing in distinctness as it was traced upwards, until, over the cartilage of the third left rib, and in the second left intercostal space, a harsh grating systolic and diastolic bruit had its position of maximum intensity. This murmur was also heard, but less distinctly, at a corresponding point to the right of the sternum; but, over the aortic area, the aortic second might, on careful observation, be occasionally heard, apparently healthy, though much masked by the coarsely grating murmur to the left. The murmur was not propagated into the large systemic arteries, but towards the left in the course of the pulmonary artery. His respirations when lying numbered 40–42 in a minute, and there was very violent action of the nostrils,—a phenomenon which it appears came on gradually, but had been much more apparent during the three months previous to examination, and seemed to be quite as evident when the patient was in bed as when he was upright. The subclavian regions expanded imperfectly, and he was apparently unable, from the rapidity with which the respiratory acts followed one another, to take a deep and full inspiration. Percussion resonance generally was not markedly dull, but was high-pitched. Vocal

resonance was nearly normal, though rather increased than diminished. He had a short, hard, and frequent cough, with an expectoration principally mucous, but sometimes muco-purulent. The respiratory murmur generally was very harsh, but without any moist sounds. He had never had hæmoptysis. The tongue was clean, but the papillæ at its edge near the tip were enlarged and congested. His appetite was bad, and he suffered from flatulent eructations, but not vomiting. His bowels as a rule acted normally, but latterly more frequently with semi-fluid evacuations of a somewhat dark colour. The liver dulness in the mammary line measured about six inches. The spleen was not satisfactorily mapped out. He said he urinated freely, and had never had any symptoms specially referable to this system, but I regret that I did not at first more accurately determine the character of his urine.

The skin presented nothing abnormal, except the small patch of extravasation, before alluded to, over the left malar prominence. The special senses were healthy; he never suffered from headache, and the intellect was clear.

The treatment adopted was:—Rest in the recumbent position, nourishing diet, with restriction of fluid, syrup of the phosphate of iron, and 5–7½ minims of the tincture of digitalis in a mixture every three to four hours. Apparently by this means his heart's action became slower and less excited, the supra-sternal pulsation for a time almost entirely disappeared, and when it returned did so gradually. The jugular pulse also appeared less marked, but his general distress was not alleviated.

In the beginning of June he had a thickly sown eruption of purpura over the forearms, wrists, legs, and ankles, which gradually faded. Such was his condition till about the middle of June, when another and more copious eruption of purpura made its appearance in the same positions, and to a less extent on the trunk, together with a feeling of weakness and pain in the lumbar region, and, though the quantity of his urine remained unchanged and was sufficient, it had a dark and smoky appearance. On June 16th it is noted to have had the following characters:—"Sp. gr. 1005, acid, smoky, albuminous to about one-quarter of the quantity of urine in the test tube; abundant tube casts, principally granular, with adherent renal epithelium; many of the casts are dark-coloured, others more transparent; a few fragments of blood-casts; red blood-cells; renal epithelium, and crystals of uric acid." He also complained of headache, became drowsy, and had some diarrhœa. Mustard counter-irritation over the loins, with an occasional draught of sulphate of magnesia, was followed by a disappearance of the lumbar pain and greater intelligence, but his urine continued as before and began to diminish in quantity. The reddening caused by the mustard was seen to consist of a thickly sown sprinkling of minute points of extravasation, resembling a fine purpura, except that the points had an arterial hue.

June 19th.—Urine very scanty; slight diarrhœa; no vomiting;

purpura gradually fading. 10 P.M.—Called suddenly to see patient. Urine almost suppressed; temperature 101°·8; pulse 120; respirations 60; skin dry; lungs as before. After a purge, and poulticing the loins, the most urgent symptoms subsided, and the eruption continued to fade.

June 22d.—Purpura again, in larger patches than ever, especially over the right wrist and elbow, on which side he lies; urine still very scanty; drowsy; slight diarrhœa; pulse 104; respirations 42.

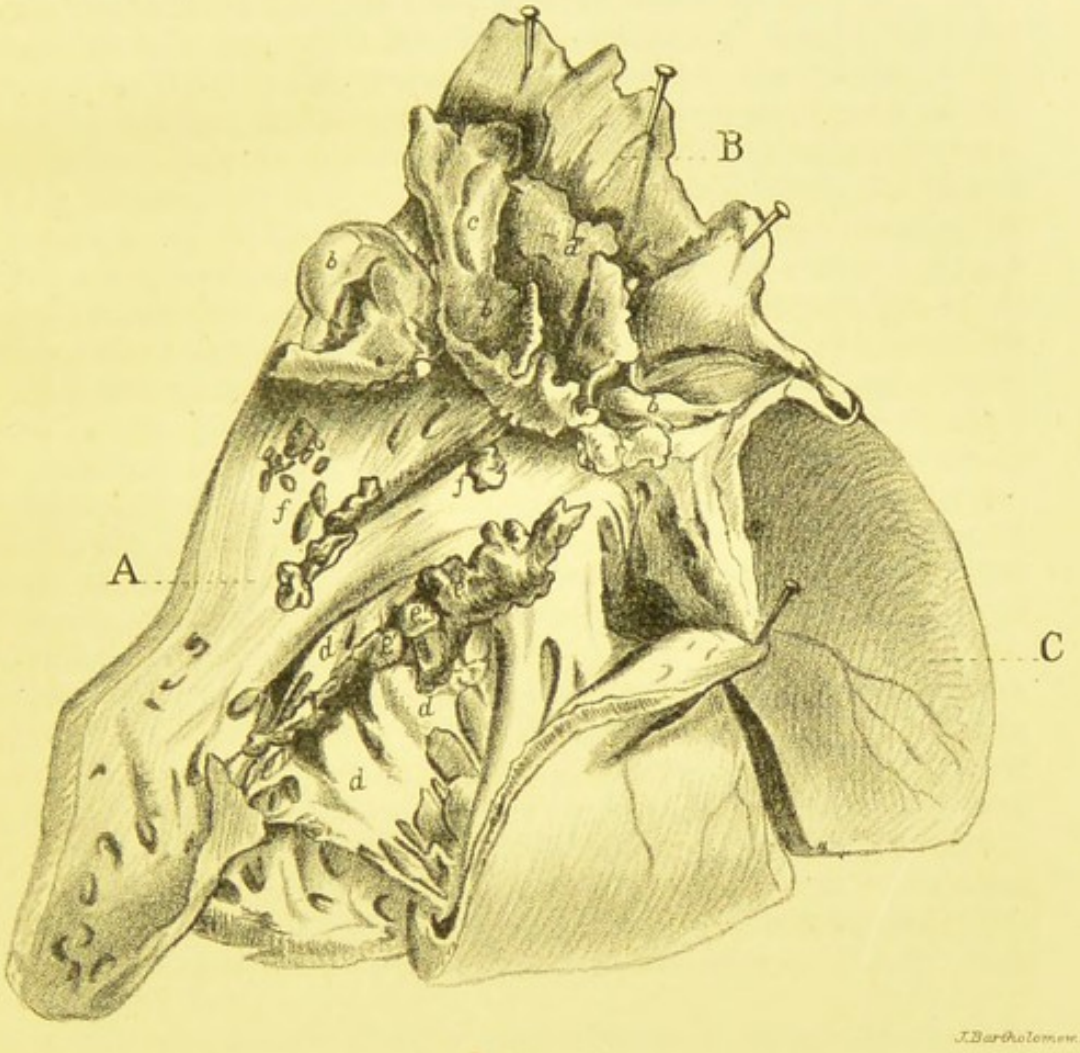
June 23d.—Urine, sp. gr. 1005.

June 24th.—About 7 A.M. began to swell about the right eye (he lies on his right side); at 11.50 A.M. the right eye was closed with œdema, the left becoming œdematous, and the forehead and scalp up to the vertex, especially towards the right side, was tense, painful, swollen, and pitted on pressure; no pitting over the sternum or tibiæ; pulse 102; respiration 42; urine as before. Was ordered a purge, and a diuretic mixture, containing digitalis, squills, spirits of nitrous ether, and liq. am. acetas, after which a larger quantity of urine was voided and the œdema disappeared. On June 27th he is noted to have had hæmoptysis to about one ounce, the blood being arterial and mixed with air, and the physical signs in the chest as before, with slight comparative dulness at the left base, but the extreme bases were throughout somewhat less resonant than elsewhere. The purpura was still persistent.

June 28th.—Heart's action regular, signs as before, pulse 108–114, regular, but very weak; respirations, 42; violent action of the alæ nasi; fæces fluid and dark; urine as before; drowsy. He died next day, having been conscious to within half an hour of death, when he is said to have become unconscious, and died without any evident increase in his distress of breathing, and without any sign of convulsion.

Necropsy, twenty-nine hours after death.—Body much emaciated. Purpura persistent on the arms, legs, and right side of the trunk. On opening the chest the lungs were seen retracted, their inner margins at the nearest point being fully an inch and a half apart, thus exposing the pericardium over the heart and large vessels. Pericardium smooth and without any trace of inflammation, recent or remote, its cavity containing about an ounce of straw-coloured fluid. The parietal layer, at a point opposite the heart's apex, finely mottled from an extravasation of blood; the visceral layer, also near the apex, having a similar extravasation. Heart, *in situ*, increased in size, especially transversely, the margin of the right ventricle, at a point opposite the axis of the conus arteriosus, being obtusely angular. The heart weighed, after being in spirit for some days, 9 ounces and 5 drachms. Right auricle dilated, with its closely-packed columnæ carniæ well marked. A warty vegetation springing up into its cavity, from the insertion of the anterior segment of the tricuspid valve at its inner extremity, and two others on its posterior wall, pass downwards and inwards through

the auriculo-ventricular orifice, their lower extremities going to form part of a warty mass on the internal segment of the tricuspid. The broadest ends of the growths are in the auricle, the largest nearest the lumen, the other two between it and the wall of the auricle, that next the wall being smallest and flat. THE DIRECTION AND SHAPE OF SUCH GROWTHS PROBABLY INDICATES THE DIRECTION OF THE PREVAILING CURRENT OF BLOOD, AND IN THIS INSTANCE ARE PROBABLY SIGNIFICANT OF TRICUSPID REGURGITATION. The posterior surface of the anterior segment of the tricuspid, *i.e.*, the surface looking into the auricle in systole, is covered, especially along its margin, with warty growths. These vegetations combined encroach considerably upon the auriculo-ventricular space. Right ventricle is dilated and hypertrophied, but the former condition preponderates. All the segments of the tricuspid are studded with warty excrescences, more especially the anterior and internal segments. The former has a thickened and irregular margin, and the latter is in fact replaced by these growths. The posterior segment lying against the right wall of the cavity is also thickened by a warty deposit. The musculi-papillares are somewhat hypertrophied. The pulmonary semi-lunar valves are replaced by a distorted mass of warty clumps. The segment most to the right when the artery is laid open retains its shape most perfectly, but its margin is enormously thickened, and below and on all sides of it there are excrescences. The central segment consists of an enormous mass of warty growths, a large pear-shaped process from which stretches into the pulmonary artery, and when unsupported, hangs at right angles to its stalk, and probably occupied this position in diastole, and may perhaps to some extent have acted as a valvular prevention to regurgitation. Behind this segment, and overlapping to some extent the segment to the right, there is a flattened mass of excrescences, attached to and passing into the pulmonary artery for more than an inch, occupying, in fact, that portion of the arterial wall which must have been struck during each systole by the mass of the central valve. The segment most to the left consists, likewise, of a misshapen warty growth, about an inch in length and breadth, having a faint resemblance to a sigmoid valve, and with an irregularly diamond-shaped perforation; and here, too, a crescent of small granulations on the wall of the artery coincides exactly with the upper margin of this altered segment. On the anterior wall of the conus arteriosus there are scattered excrescences of various size and shape. Towards the apex of the ventricle, and attached to a slender chorda tendinea, there is a vegetation. The circumference of the pulmonary artery at the line of the valves is $3\frac{5}{8}$ inches, and above the valves $3\frac{6}{8}$ inches. A large partially decolorized fibrinous clot passed from the site of the pulmonary valves up to and into the two principal branches of the artery, on dividing which the clot was seen to occupy the entire lumen of each vessel. The clot was most decolorized towards the ventricle. A large clot of a similar nature was drawn



Heart of E. W. G. M. About two-thirds the size of the original.—A, right ventricle; B, pulmonary artery; C, left ventricle; *d, d, d*, tricuspid valve; *b, b, b*, distorted pulmonary semi-lunar valves; *c*, pyriform vegetation; *d'*, flattened mass of vegetations in pulmonary artery; *e*, large vegetation where probe passed into left ventricle; *e', e'*, ends of vegetations passing into right auricle; *f, f*, scattered vegetations.

through the auriculo-ventricular orifice, and there were other smaller clots clinging to the chordæ tendineæ. The clots clung most tenaciously to the growths. The excrescences had a cartilaginous or gritty centre, with a soft circumference. Microscopically they appeared to consist of oval or rounded nuclei, in a granular, more or less opaque stroma, at places much impregnated by an earthy deposit. The portions examined did not present any appearance of a fibrous stroma. The sections cleared on adding dilute hydrochloric acid, and less so with acetic acid.

Left auricle normal. Mitral valves healthy, except that the shorter has two small warty deposits about the centre of its auricular aspect. At the margin of the other segment, and on the same aspect, there is a growth of the size and shape of a split pea.

Left ventricle normal. Circumference of the aorta at the line of the valves $2\frac{1}{2}$ inches; above the valves $2\frac{3}{8}$ inches. Semilunar valves competent and healthy. At the base of the centre segment there is a rounded cavity, with a minute warty excrescence at its orifice. On holding this part up to the light the septum is seen to be thin and translucent (a natural condition), and a large blunt probe passed into the above-mentioned small cavity tore through the partition, and presented itself below the internal segment of the tricuspid valve, the point emerging in a bed of granulations, the site of a large vegetation. At the origin of the aorta there is a trace of commencing atheroma. The endocardium in the right ventricle, especially in the neighbourhood of warty clumps, is thickened and opaque.

The coronary arteries, so far as they have been examined, are healthy. Muscular fibres taken from the right cavities of the heart had well-marked cross striæ. A few enlarged glands which lay between the pulmonary artery and aorta were dissected away, but I regret that the former vessel was not examined throughout.

The pleuræ were generally adherent over both lungs, presenting many old adhesions of varying length, those at the apex and base being shorter than those in the intermediate space. The cavities contained no fluid.

The left lung floated in water and crepitated uniformly, though not quite freely. At its extreme base it seemed to be in a state of chronic consolidation. The lung generally was of a firmer consistence than is natural, and contained circumscribed patches of induration. Below the pleura, at the base posteriorly, there were a few ecchymoses similar to those in the pericardium. The mucous membrane of the bronchi was reddened, and some tubes contained pus, others casts of inspissated secretion, but there was no evidence of breaking-down lung tissue anywhere. The right lung was in the same condition as the left, but rather firmer at the base, and contained in this portion a comparatively recent extravasation of blood about the size of a walnut. *Liver.*—The longest measurement of the right lobe was about 8 inches; of the left 5 inches. The organ was firm to the touch, with a smooth capsule, and on

section gross and microscopic, presented the appearances of "nutmeg liver."

The spleen was 7 inches long, 4 inches wide, firm to the touch, with a somewhat thickened capsule, a well-marked notch in its anterior border, and presented no distinct evidence of amyloid degeneration. The pancreas was firm, the connective tissue between its lobules increased, and its veins engorged. The left kidney was enlarged, its cortex increased, the capsule peeled off readily, and its surface was smooth, finely sprinkled with myriads of engorged Malpighian tufts, and had a few arborescent veins. The right kidney in its upper two-thirds was like the left; its lower third had a much more lobulated surface, and was separated from the remainder by a deep sulcus; the cortical and pyramidal portions of this were scanty, but the former was relatively increased. With the microscope many of the tubules were seen to be dilated, some bereft of epithelium, and others filled with blood or their own degenerated cells. The Malpighian tufts were intensely engorged. *Stomach.*—Mucous membrane engorged, with numerous ecchymoses, much thickened, and the follicles individually enlarged, which gave the whole a mammillated appearance. The small was of a darker colour than the large intestine, and the mucous membrane of both was passively engorged. The omentum contained a very small quantity of dark-coloured fat. The head was not examined.

General Remarks on the Case of E. W. G. M.

The physical signs in this case were indicative of pulmonary obstruction and regurgitation, with dilatation or distension of the pulmonary artery, and also of tricuspid regurgitation, and probably, too, of a minor degree of tricuspid obstruction. The unusual nature of the case rendered its correct diagnosis a matter of interest, and though I had some doubt as to the true significance of the systolic apex bruit, the pulmonary lesion was satisfactorily determined.

The situation of the murmurs, and their propagation to the left in the course of the pulmonary artery, coupled with their want of propagation into the systemic arteries, and the state of the radial and carotid pulses, together with the impaired and diffused apex beat, the site of visible pulsation, and the fact of the aortic second sound being occasionally heard, apparently healthy, through the murmur, served to exclude aortic disease. I was, however, inclined to believe that there was some mitral regurgitation, but the failure to follow the systolic apex bruit outwards to the left seemed to negative such a conclusion, which the necropsy ultimately showed to have been erroneous.

Well marked as were the morbid appearances, and self-evident the agents in the production of the various physical signs in the present instance, the true etiology of the case is open to much discussion. Was the disease congenital, traumatic, due to scarlet fever, to disease of the kidneys, or, finally, to a combination of two

or more of these possible factors, and influenced, it may be, by a rheumatic diathesis? Not to argue the point tediously, I may state shortly, that, from a careful study of the entire history of the case, and of the heart itself, it appears to me that the primary excitant to disease was scarlatina in a patient of rheumatic diathesis, and one of a family prone to heart disease; that this scarlatinal endocarditis had led to change in the valves, rendering them incompetent; that the altered segments themselves, from their disturbance of the blood current, and impact against various surfaces, tended to propagate disease; that the anæmic condition of the patient, possibly standing in some relation to his kidney disease, may have aggravated matters, and that finally rupture or displacement of one or more segments, perhaps a result of exercise at cricket, crowned the catastrophe, impairing still more the efficiency of an organ already labouring at great disadvantage, and precipitating the inevitable result.

This view of the causation of the pathological changes in the right chambers of the heart was endorsed by the Morbid Growths Committee of the London Pathological Society; but I am now inclined to attach more importance to that "rounded cavity" at the "undefended spot" in the ventricular septum, passed through which a large probe tore into the right ventricle. I consider it quite possible that this may have been a minute congenital communication, too minute to divert either side of the circulation, but quite sufficient to originate an irritative condition in its neighbourhood, or to promote such a condition otherwise originated. Latterly, the exuberance of vegetation in the right ventricle served to close completely any communication, if such had ever existed.

I shall conclude this portion of the subject by directing attention to a few points of interest in the present case.

When we consider the very serious obstruction at and regurgitation through the valves of the right heart, we cannot but be struck with the remarkable absence of dropsy into the subcutaneous tissue or serous cavities. It is feasible to suppose that when purpura appeared, the obstruction was so great as to cause that ecchymotic extravasation, and too great to allow of the gradual escape of the watery constituents of the blood, so as to produce anasarca; but this does not explain why, at a period prior to the occurrence of purpura, there was no or very trifling œdema. We can only conclude that the compensatory balance, established by hypertrophy of the right heart chambers, and the normal action of the left, to be discussed more fully afterwards, must have been very complete, and it is interesting to note that it was only during the last year of life that the patient occasionally noticed the œdema across the instep, and that it was during this year that a sensible increase of embarrassment in his condition was observed.

I would also indicate the unusual dyspnœa, little influenced by position, though somewhat increased by exertion, shown by the rapid respiration and violent action of the nostrils, as being almost

diagnostic of his lesions, and resembling, in some degree, the symptoms usually ascribed to partial embolism or thrombosis of the pulmonary artery, a resemblance as real as it was apparent, for the huge dendritic clumps and altered valves, filling up the mouth of the vessel, were no mean substitutes for a clot, and (though this only applies to a period shortly prior to death), the discoloured clot passing through the pulmonary orifice, along the artery and into its main, and, doubtless also, secondary bifurcations, WAS a true thrombosis, and this again may have increased the retraction of lung apparent on raising the sternum. The condition of the lungs seen on post-mortem examination was of interest, in so far as they contained hæmorrhagic infarcta, and otherwise evinced the presence of hyperæmia. As the obstruction and valvular insufficiency existed at a point in the circulation before it enters the lungs, one might *à priori* have expected a less, in place of an excessive quantity of blood in those organs. The contrary is probably due, IN SOME MEASURE, to the absence of adequate *vis-à-tergo* to propel the blood through the arterial and venous radicles into the pulmonary veins, the direct cause of the inadequacy being regurgitation through the pulmonary semilunar valves, the spring-board action of which, when competent, being lost. In a case such as the present, the circumscribed patches of induration may also, possibly, be owing to the detachment of small granulations, or portions of such, and consequent embolism of some smaller branches of the pulmonary artery, or to coagula transported hither, after having been switched into form, by the valvular see-saw, at the pulmonary orifice. It is also worthy of notice that the angina experienced by the patient was distinctly limited to the right side, aswe may conclude from the fixed agonizing pain at the lower end of the sternum, darting up to the right shoulder and down the right arm.

The tendency of the purpura and œdema of the face and scalp to gravitate towards the right half of the body was doubtless due to the position of the patient, and the latter (facial œdema, etc.) was probably of mixed renal and cardiac origin, as it disappeared when the flow of urine was increased; for here, as in many other circumstances, two factors, at least, were probably culpable—both kidneys and heart; the relief to the circulation afforded by an increased flow of urine, relieving likewise the obstructed venous flow into the heart, would materially lessen the tendency to œdema of the face and scalp; while, on the other hand, a more regular action of the heart might have been active at once in removing the œdema, and by relieving retrograde renal congestion, in favouring the secretion of urine.

OTHER CASES.—To assist further inquiry into the nature of dextral valve lesions, and to provide a larger number of data from which to draw conclusions, I have compiled the annexed table of cases of valvular disease of the right heart. The series does not pretend to be exhaustive of the literature of the subject, but will be found on examination to be thoroughly representative.

Table of Cases of Organic Disease of the Tricuspid and Pulmonary Valves.

No.	Reporter and Reference.	Name, Age, Sex, Occupation.	Previous History and Duration of the Disease.	Symptoms in Circulatory System.	Symptoms in Respiratory System.	Effect of Digitalis or other remedy.	Post-mortem Condition of the Heart and other Organs.	General Character of the Disease.
1	Bertin, R. J. Traité de mal du cœur, p. 197; 1824.	Williams Wiple, male, General in the Army.	Had suffered long from symptoms of diseased heart, but became worse in consequence of the hardships of the American Revolution.	Palpitation on the slightest exertion; "une grande anxiété:" continually cold extremities.	Not stated.	Not stated.	Dilatation of the right auricle; an ossified tricuspid valve closed the right auriculo-ventricular orifice, and was pierced at its free edge by two holes, connected by an intervening piece an inch long, and about as broad; at its base another hole bordered (about iss-ait) on the left ventricle, at the level of the mitral valve. The left cavities were normal (dans l'état naturel).	Tricuspid obstruction.
2	Balthazar Foster, M.D. Clinical Medicine, pp. 324–330.	T. S., 35 years, male, "Sticker."	He referred his illness to an attack of rheumatic fever, which had occurred six years previously, and had disabled him more or less ever since. Had experienced epistaxis, disturbed sleep, dyspnœa on exertion, palpitation and pain at the ensiform cartilage. Improved for three weeks in hospital, when he caught bronchitis from cold; and, though he recovered somewhat, rose too soon, and died next day, with great dyspnœa, angina at ensiform cartilage, and lividity of surface.	Face puffy and dusky, lips slightly blue, jugular veins distended, slight undulatory pulsation in the right vein, occurring prior to the heart's systole; slight œdema of feet, no albuminuria; transverse dulness of heart slightly increased, apex below sixth rib; epigastric pulsation very distinct, thrill most appreciable at ensiform cartilage; to right of sternum a presystolic bruit, most distinct at base of ensiform cartilage, and propagated very little in any direction from this spot. No transmission into the great vessels; no accentuation of pulmonary second sound; pulse trace dicrotic: for some hours before death radial pulses very weak, and the left was first to become imperceptible.	Lungs normal on percussion, and breath sounds healthy. For the rest see "Previous History."	No treatment mentioned, except free cupping with stimulants on the day before his death.	Right auricle greatly distended. Pericardium adherent to front of heart; left cavities contained little blood; left ventricle slightly hypertrophied; right ventricle full of blood, but neither dilated nor hypertrophied; right auricle distended and hypertrophied, in some places a quarter of an inch thick; tricuspid segments adherent; orifice admitted the first finger, as far as the first joint, edges of orifice thick and rough, with calcareous spots on the auricular surface; pulmonary mitral and aortic valves healthy; orifice of aorta and left auriculo-ventricular opening narrowed (not said to be from disease). Liver small, granular; spleen large, congested; kidneys congested, but otherwise healthy. Serum in peritoneum.	Tricuspid obstruction.

TABLE OF CASES OF ORGANIC DISEASE OF THE TRICUSPID AND PULMONARY VALVES—*continued.*

No.	Reporter and Reference.	Name, Age, Sex, Occupation.	Previous History and Duration of the Disease.	Symptoms in Circulatory System.	Symptoms in Respiratory System.	Effect of Digitalis or other remedy.	Post-mortem Condition of the Heart and other Organs.	General Character of the Disease.
3	M. B. Tod, M.D., etc. Dublin Quarterly Journal, 1st Feb. vol. v. New series.	Edward Ingram, æt. 21 years.	Two years and three months before admission, engaged in a riot, and was stabbed a little below the right mamma. Bled freely, and had pleurisy, melœna, and four weeks later hematemesis; the two latter conditions recurred repeatedly till within three months of death. During last month of life, rapid supervention of dropsy, beginning in the face and upper extremities. Died five days after admission, dyspnœa and dropsy being so great as to prevent examination.	Face and neck much swollen. Transverse dulness of heart increased. Visible and palpable pulsation at scrobiculus cordis. Systolic bellows murmur loud at apex and over sternum, and a second bruit, also systolic, but of a different tone, at the base, and in the course of the aorta. The second heart sound natural, but feeble; pulse small, thready, compressible. Urine scanty, but free from albumen.	Respirations, forty per minute, loud and puerile anteriorly; feeble and crepitant posteriorly. Position, semi-recumbent. Dyspnœa very great.	Diuretics and mild stimulants employed without benefit.	Anasarca, pleuritic effusion, and ascites. The lungs did not collapse, and were highly œdematous. "The heart presented considerable hypertrophy, with dilatation of the right ventricle and auricle; the left cavities were rather larger than the normal size." Valves of left side healthy, as also pulmonary sigmoid valves. Anterior and largest segment of the tricuspid valve severed from its musculi papillares from rupture of the corresponding cordæ tendineæ. Musc. pap. atrophied. "The aorta and its branches were unusually small, and their coats much attenuated, so that they resembled in structure the pulmonary artery, which on the other hand partook of the dilatation and hypertrophy of the right cavities. Mucous membrane of stomach pale and thin. Condition of other organs unimportant.	Tricuspid regurgitation from rupture of cordæ tendineæ.
4	Fox, Wilson, M.D., etc. Trans. Path. Soc., London. vol. xxi. p. 101.	C. H., male, æt. 50 years; Stableman.	Healthy, till he had "rheumatics" five years before seen. For seven or eight months somewhat short-breathed. Seven weeks before seen, a bad cold increased dyspnœa, and he had at the same time œdema of lower extremities. Died fifteen days after admission.	Face, lips, hands, almost cyanotic; jugular and subclavian veins intensely distended; external jugulars singularly large; pulse hard, weak, regular, jerking; heart's impulse weak, regular, diffused; transverse dulness increased, liver enlarged; urine albuminous.	Continuous orthopnœa, cough frequent; lungs hyper-resonant except at bases, where they were bronchophonic and crepitated.	Not stated.	Right auricle enormously distended; inferior cava, ditto; right ventricle firmly contracted; muscle very firm; walls average $\frac{7}{16}$ inch thick; tricuspid orifice 4$\frac{6}{8}$ inches. Cordæ tendineæ distinctly shorter than usual; edges of segments thickened and allowing regurgitation; pulmonary artery healthy; left ventricle greatly enlarged and thickened; pulmonary veins distended with p.m. clots; mitral valve healthy; circumference, 4$\frac{1}{2}$ inches; aortic valves competent; upper lobes of lungs emphysematous; elsewhere pneumonic with embolic patches; liver engorged. Kidneys contracted, and granular.	Tricuspid regurgitation.
5	Ormerod, Dr. Edin. Med. and Surgical Journal, vol. lxv. 1846.	W. C., æt. 28, male; Waterman.	Temperate; palpitation; dyspnœa on exertion for two years, but able to lie horizontally till the last. Died with dropsical symptoms in 1839.	No notable distension of superficial veins or jugular pulsation. Anasarca and ascites from time to time; systolic pulmonary bruit, which after a year became diastolic.	Refer to account of previous history.	Not stated.	Heart nearly twice its natural size; pulmonary valves agglutinated to a thick cartilaginous ring, leaving an orifice the size of a crow-quill; all the other valves healthy.	Pulmonary obstruction.
6	Begbie, J.W. Beale's Archives. No. 5; and Edin. Med. Journal, p. 560, 1859-60.	W. W., male, æt. 18; Light porter.	Always slightly short-breathed, especially after exertion; but could work actively. In hospital occasionally for breathlessness and palpitation; the latter seemed greatly functional. Fell while drunk, fractured his skull, and died in consequence.	Radial and other superficial pulses normal, 74; of good strength; no lividity of countenance; impulse not exaggerated, but readily appreciable; pulmonary systolic basic murmur ultimately diastolic.	Occasional attacks of breathlessness, always relieved by the care and comfort of hospital residence.	Henbane and iron; belladonna plaster; care and diet always removed dyspnœa and palpitation.	Pulmonary valves, consisting of four segments, one smaller than the rest; valve slightly incompetent; no hypertrophy or dilatation of the right ventricle; heart otherwise healthy. No account of other organs, whose condition was therefore probably unimportant.	

TABLE OF CASES OF ORGANIC DISEASE OF THE TRICUSPID AND PULMONARY VALVES—*continued.*

No.	Reporter and Reference.	Name, Age, Sex, Occupation.	Previous History and Duration of the Disease.	Symptoms in Circulatory System.	Symptoms in Respiratory System.	Effect of Digitalis or other remedy.	Post-mortem Condition of the Heart and other Organs.	General Character of the Disease.
7	Wilks, S., M.D., etc. Trans. Path. Soc., Lond. vol. xv. p. 74.	Joseph, S. æt. 31 years; Cabman.	Rheumatism nine years before seen; since then a cabman and intemperate; last two years occasional dyspnœa, but not sufficient to incapacitate for work, until four months before death, when pulmonary symptoms appeared.	Double bruit in the pulmonary area, but the disease was not diagnosed. Pulse gave no indication of aortic regurgitation.	"Extensive disorganization of both lungs, of which he died."	Not stated.	Two segments of the pulmonary valve had almost disappeared; remaining one perfect, and not in any way thickened. Aortic valves adherent for a small portion of their surface. "The heart was of the usual size and form; the right ventricle not at all enlarged." Lungs disorganized. State of other organs not specified.	Pulmonary regurgitation.
8	Paget, J., F.R.C.S., Trans. Med. Chir. Soc., vol. xxvii. p. 182; and Ormerod, Edin. Med. and Surg. Jour., vol. lxv. 1846.	Female, æt. 20.	Poor, pale, haggard prostitute. Died seven weeks after admission to St Bartholomew's Hospital. Subject to fainting fits.	Rapid, feeble pulse; violent palpitation, with increased impulse; loud basic bellows murmur transmitted to the left shoulder; albuminuria; some ascites and œdema of legs; before death signs of pleuro-pneumonia and effusion into chest. Shortly prior to decease purpurous spots made their appearance all over the body.	Slight cough, without expectoration; constantly lay on back, and did not appear to have any difficulty in breathing.	Not stated. Seemed to improve during first month of stay in hospital.	Right auricle somewhat dilated and hypertrophied; right ventricle much more so; tricuspid valve healthy; pulmonary valves, only two in number, thickened and opaque, with large vegetations attached; granulations and some ulcerations in the pulmonary artery; mitral and aortic valves healthy; left auricle and ventricle "were healthy." Adhesions and serum in the pleuræ; pulmonary artery and its ramifications filled with coagula, partly ante-mortem; upper lobes of lungs emphysematous. In lower lobes about twenty compact masses of pulmonary apoplexy. Liver pale, otherwise healthy; pint of serum in peritoneum; spleen large, firm, with extravasated patches; "kidneys healthy in texture, but pale."	Pulmonary obstruction and regurgitation.
9	Bertin, R. J. *op. cit.*, p. 199.	Male, æt. 25 years; Mason.	No particulars beyond what may be inferred from his having reached the age stated, and the nature of his employment.	All the symptoms of a great obstacle to the circulation. "Bruit de soufflet" over all the front of the chest, loudest near the sternum.	Symptoms of respiratory embarrassment.	Digitalis and bleeding were tried in vain.	The right chambers of the heart "enormously hypertrophied." A column (pilier) of the right ventricle was applied across the orifice of the pulmonary artery, which it tended to retract (qu'il concourait à rétrécir); the orifice of the vessel was considerably narrowed, from change in the sigmoid valves, which formed a sort of fibrous porter's knot (bourrelet), the aperture in which was about 2½ lines in diameter; the tricuspid valves were yellowish, thickened at their edges, adherent throughout, and partially ossified; at the commencement of the auricle, near the pulmonary artery, there was a hole 2 lines in diameter, which established a communication between that artery and the right ventricle ("ventricule droit," probably a mistake for *left* ventricle). The left chambers showed nothing peculiar (n'offraient rien d'extraordinaire).	Tricuspid and pulmonary obstruction.

TABLE OF CASES OF ORGANIC DISEASE OF THE TRICUSPID AND PULMONARY VALVES—*continued.*

No.	Reporter and reference.	Name, Age, Sex, Occupation.	Previous History and Duration of the Disease.	Symptoms in Circulatory System.	Symptoms in Respiratory System.	Effect of Digitalis or other remedy.	Post-mortem condition of the Heart and other Organs.	General Character of the Disease
10	Morison, Alexander, Trans. Path. Soc. Lond. vol. xxvii. p. 83, *et seq.*	E. W. G. M., etc., etc.	Refer to full particulars of the case in the body of the thesis.					Tricuspid regurgitation; some tricuspid obstruction; pulmonary obstruction; and regurgitation.
11	Gairdner, W. T., M.D., etc. Clin. Med., p. 603, edition 1862; and B. Foster, *op. cit.*, p. 329.	Patrick M., æt. 20 years, male; Labourer.	Firmly built; tolerably active; neither livid nor dropsical. Died in 1872.	Jugular veins on both sides dilated, enlarged and undulating, but without being much distended with their contents. Tricuspid presystolic bruit.	Not stated.	Not stated.	Alive when Clin. Med. published, but on death (B. Foster) the tricuspid obstruction was found to be due, not to valvular disease, but to a tumour attached to the auriculo-ventricular orifice, after the manner of a ball valve. "There was no appreciable hypertrophy either of the right or left ventricle, and almost no dilatation." (The last sentence is copied from a post-card, dated 12th Dec. 1877, which Professor Gairdner kindly addressed to me, in answer to a note asking for particulars, which have not, so far as I know, been published).	Tricuspid obstruction from floating tumour.
12	Kinglake, R. surgeon. Lond. Med. Journal, vol. x. pt. iv. 1789; also Kreyzig, "Die Krankheiten des Herzens," p. 430, 1815.	Catherine Kinch, female, æt. 21 years.	Delicate; constant pain in side for several years; uneasy motion of the heart for three years; cause assigned, a jolt in a carriage!! Two years under observation before death.	Enlargement of and viscid discharge from the left breast, without disease of its structure; later a similar discharge from the fauces; "uneasiness in heart and lungs, aggravated by transposition to a most formidable throbbing and sense of suffocating stricture, alternating with almost a state of inaction, and consequent syncope.'	No cough, except when due to discharge in fauces, and by no means characteristic of pulmonary affection.	Three hundred and twelve venesections during two years, averaging 4 oz. each time; less found to be useless. "To describe the benefit gained by each bleeding would be to exhibit the difference between the most afflicting pain and comparative ease."	Pericardium and pleura full of fluid; more than half the cavities of the right auricle and ventricle obstructed by polypous concretions, the largest as big as a walnut, and situated in the right auricle; half-an-inch beyond the pulmonary valves, a hard stony substance, weighing half a drachm, and diminishing the calibre of the vessel. Other organs apparently sound, with the exception of the stomach, which was thinned.	Tricuspid and probably pulmonary obstruction from polypous concretions and pulmonary endarteritis.

The cases tabulated present a complete series of valvular disease, pure and mixed, and likewise show the etiology of morbid valves in the right heart to be essentially the same as that of similar affections in the left heart, for in the former, as in the latter, we see endocardial inflammation, traumatic rupture, and polypous obstruction producing valvular disorder. It is also satisfactory to note, that the diagnosis of the disease from physical signs in some of the above instances was as precise and accurate as the discrimination of analogous conditions in the left heart by similar methods of exploration; thus rescuing the diagnostic phenomena in question from the "region of romance," and constituting them an integral portion of the armamentarium of the educated physician.

As regards the age of the patients, it will be seen that there were none in the first decade, when they came under observation, though three were probably congenitally abnormal, one in the second, seven in the third, two in the fourth, none in the fifth, and one in the sixth. The age of Bertin's patient, General Wiple, is not stated, but the fact of his having been a general officer may justify our placing him in the sixth decade.

Ten of the patients were males, and only two females. Nine followed active or exposed employments, while the occupation of two was sedentary or indoor, and finally, that of one is not stated; but as the exception is Dr Tod's case of ruptured tricuspid valve, the occupation of the patient was probably active.

I shall now criticise more particularly the condition of the walls of the cavities of the heart as regards hypertrophy, together with the theory of the production of such accessory development, and its significance in relation to the duration of the disease (*i.e.*, on the life of the patient), and its influence upon the state of the pulmonary and general circulation.

To facilitate such an inquiry I have appended the following analysis on page 20 of the cases already tabulated.

Hypertrophy with or without Dilatation of the Right Ventricle.

Dr Tod, in his account of Ingram's case in the *Dublin Quarterly Journal*, already quoted, regards hypertrophy of the right ventricle as the last of a series of compensating changes in cases in which there is regurgitation through the tricuspid orifice. The sequence of events, in Dr Tod's opinion, is dilatation with hypertrophy of the right auricle, retardation of the venous circulation, the latter being followed by dilatation and hypertrophy of the left ventricle and auricle—these again impeding the pulmonary circulation, which finally is the incentive to accessory development in the right ventricle. The analysis appended does not altogether support this theory. Dr Wilson Fox's case of tricuspid regurgitation certainly exhibited both right and left ventricular hypertrophy, but

Analytic Table of Hypertrophy, Etc.

No. of Case.	Name of Author.	Hypertrophy, with or without Dilatation of the Right Ventricle.	Hypertrophy, with or without Dilatation of Right Auricle.	Hypertrophy, with or without Dilatation of Left Ventricle.	Hypertrophy, with or without Dilatation of Left Auricle.	Dilatation alone of the Right Auricle.	Dilatation alone of the Left Auricle.	Duration of the Disease.	Degree of Embarrassment of the Pulmonary Circulation.	Degree of Embarrassment of the Systemic Circulation.	Character of the Disease.	Remarks.
1	Bertin.	?	?	0	0	1	0	"A long time."	Moderate.	Slight.	Tricusp. obs.	The numerals signify hypertrophy; numerals with a minus sign affixed, a doubtful degree of hypertrophy, which must nevertheless have been present; ciphers, the absence of hypertrophy; the mark of interrogation, cases in which the condition has not been reported. The terms "none," "slight," "moderate," "great," refer to the degree in which the secondary effects of circulatory obstruction have been noted, such as anasarca, ascites, albuminuria, pulmonary congestion, etc.
2	Foster.	0	1	1	0	0	0	Six years.	Moderate.	Moderate.	Do.	
3	Tod.	1	1	1	1	0	0	Two and a quarter years.	Great.	Moderate.	Tricusp. reg.	
4	Fox.	1	0	1	0	0	0	Five years.	Great.	Great.	Do.	
5	Ormerod.	1 –	1 –	1 –	1 –	?	?	Two years at least.	Moderate.	Occasionally great.	Pulm. obst.	
6	Begbie.	0	0	0	0	0	0	Congenital; 18 years.	Slight.	None.	Pulm. reg.	
7	Wilks.	0	0	0	0	0	0	Two years.	Moderate.	Moderate.	Do.	
8	Paget.	1	1	0	0	0	0	?	None.	Great.	Pulm. obst. & reg.	
9	Bertin.	1	1	0	0	0	0	Congenital; 25 years.	Great?	Great?	Tricusp. & Pulm. obst.	
10	Morison.	1	1	0	0	0	0	Twelve and ¾ years. Congenital?	Moderate, except in last 12 months, when great.	Slight; after digitalis great.	Tr. obs. & reg. Pl. obs. & reg.	
11	Kinglake.	?	?	?	?	?	?	Three years at least.	Great.	Moderate.	Tr. Pl. obst.	
12	Gairdner.	0	0	0	0	0	0	Ten years.	Slight.	Slight.	Tricusp. obs.	

the weight of the evidence afforded by this case on this point is diminished by the concomitant state of the kidney, which was one of granular contraction. It is difficult to determine how much of the left ventricular hypertrophy may have been due to cardiac, and how much to renal obstruction, as it is likewise difficult to ascertain whether the state of the kidney was the result of passive congestion (the "stauungs nephritis" of the Germans) pure and simple, or of this coupled with an independent renal affection.

Tricuspid obstruction, however, may be regarded as having much the same effect upon the circulation generally as tricuspid regurgitation. In both there is obstruction to the proper entrance of blood into the right heart. Dr Foster's case of tricuspid stenosis argues the correctness of Dr Tod's theory, for in it the right auricle and left ventricle were hypertrophied, while the condition of the right ventricle was normal, and, excluding the accidental bronchitis of which the patient died, the condition of his lungs was comparatively good, that is, they exhibited no evidences of any important obstruction in the pulmonary circulation. It will be remembered, however, that in this case there was some narrowing of the aortic and left auriculo-ventricular orifices, which may in some degree have favoured left ventricular hypertrophy.

On the other hand, if we exclude Dr Ormerod's and Mr Kinglake's cases, of which I have been unable to obtain sufficiently minute details, the remainder allow one to question the universal applicability of Dr Tod's theory. It is true that in Dr Gairdner's case the tricuspid valves themselves were intact, and in the other cases the lesion was either at the pulmonary orifice or at both the pulmonary and tricuspid orifices, and in Bertin's first case at the tricuspid orifice alone; but the obstruction to the general circulation in these cases was (with the exception of Dr Begbie's case) ultimately as great as in the cases recorded by Drs Foster, Tod, and Fox. In none of these cases, however, do we find any record of a hypertrophic condition of the left chambers of the heart, while in Dr Wilks's case there is a remarkable absence of accessory growth in the right and left chambers alike, although the obstruction at the pulmonary orifice and in the lungs must have been very considerable.

Taking the results of all the cases tabulated, irrespective of the nature of the obstructive or regurgitant lesion, we find that in five both the right ventricle and auricle were hypertrophied, with or without accompanying dilatation; in four, these chambers were almost or quite normal; and of three we cannot speak positively; while, on the other hand, the left ventricle was hypertrophied in only three instances, was normal in seven, and doubtful in two. The left auricle, again, is recorded as having been hypertrophied in one, normal in nine, and doubtful in two. This statement, curiously enough, applies to the right auricle as regards simple dilatation. That dilatation here was subsequent to previous hypertrophy is

probable, but from the record we cannot positively conclude that such was the case. Finally, as regards simple dilatation of the left auricle, we have a series of negations; in two cases alone was this doubtful, and these cases were likewise doubtful in every other particular.

We see, then, that the left cavities are protected in the cases tabulated in the ratio of 3:5 as regards the left ventricle, and 1:5 as regards the left auricle when compared with their corresponding chambers to the right; while the proportion of cases of disease of the right heart in which both right and left cavities have escaped hypertrophy is 4:7 as regards the ventricles, and 4:9 as regards the auricles. Doubtful cases are excluded in both estimates.

These facts, as I have said, allow us to question the universal applicability of Dr Tod's theory, for, if we admit the testimony of Dr Foster's case in support of that theory, we must accept the testimony of the other cases, whether regurgitant or obstructive, against it. Moreover, in Dr Wilson Fox's case there was, as we have seen, a renal complication, which may or may not have had a cardiac origin, but which probably assisted in the hypertrophy of the left chambers, and Dr Tod's case was one of sudden rupture in an otherwise healthy heart, and in such cases as we know the retrograde congestion is frequently more intense than in cases where valvular incompetency has been more gradually established, in consequence of disease.[1]

It is therefore probable, that we have in the right heart and in the venous section of the circulation a considerable force for the due propulsion of the blood through that portion of its circuit and pulmonary circulation, independently of the active propulsive force resident in the left side of the heart. This is likewise argued in those cases of valvular incompetency in the right heart, in which, notwithstanding the persistence of such for a lengthened period, there is no accessory development either of the right or left heart cavities, as, for example, in the cases recorded by Drs Begbie, Wilks, and Gairdner.

What it is my endeavour to demonstrate, then, is that although the left chambers may become hypertrophied in cases of valvular disease of the right heart, should the patients survive sufficiently long, there is an independent recuperative power in the right heart, the pathological evidence of which is compensatory hypertrophy of the right ventricle and auricle, independently of such change in the

[1] If the hypoplasial condition of the systemic arteries in Dr Tod's case was congenital, it may have been causal in the production of rupture of the cordæ tendineæ, by offering a serious impediment to the relief of the engorgement of the pulmonary and systemic venous circulation arising from the excitement and exertion incidental to participation in a riot. If, on the other hand, it was acquired, it was with greater probability the result of the diminished transmission of blood to the left heart, after injury to the tricuspid valve. The attenuation of the coats of the arteries in addition to their diminished calibre may perhaps argue in favour of the latter supposition, and this state of the arteries might then be regarded as a form of atrophy from insufficient use.

left heart, and that right ventricular hypertrophy may arise, without any impediment to pulmonary circulation due to dilated hypertrophy of the left chambers, as Dr Tod insisted.

One of these forces is undoubtedly the vertical upward pressure of the column of venous blood, which, according to a law of natural philosophy, is governed by the same laws as the vertical downward pressure (*Elementary Treatise on Physics, etc.*, by Ganot, translated by E. Atkinson, p. 70, 1868). This force, spoken of in some text-books of physiology as the force of "capillary pressure," together with the action of the left ventricle and resiliency of the elastic arteries on the one hand, and the force of the vacuum created by the respiratory movements on the other, are usually considered the chief factors in the maintenance of the circulation of the blood. But in addition to and in consequence of these, we have the force arising from the law which the blood like other fluids obeys, viz., the tendency of such substances to rise to their own level, or to the level of their source of supply, especially if the superincumbent atmospheric pressure be diminished or removed. It will be admitted that the vertical downward pressure of the column of arterial blood, independently of the systole of the left ventricle or elasticity of the aorta, comes into play at the origin of the descending aorta, the difference between the level of which and the entrance of the vena cava inferior into the right auricle is very considerable; so that the column of venous blood must have an impulse towards the pulmonary circulation independently of the force of respiration and action of the right chambers of the heart, etc., equal to the force of the vertical downward pressure of the arterial column, between the origin of the descending aorta and a point in that vessel on a level with the entrance of the inferior vena cava into the right auricle.

Yet another force, as I shall endeavour to prove elsewhere, is the force of an active diastole of the right ventricle exercising a measure of suction towards itself. Before discussing this point, however, it will be more convenient to examine the condition of the pulmonary circulation in cases of disease of the valves of the right heart, and the influence of such conditions on the anatomical state of the venous section of the circulation.

Pathological Condition of the Lungs.

In seven of the cases tabulated the condition of the lungs is not specified; in one (Dr Wilks's case) the cause of the patient's death is stated to have been extensive disorganization of the lungs, a phthisis pulmonalis, which is not stated to have had any distinct relation to the state of the heart. Our information is therefore only to be derived from four out of twelve cases, viz., those recorded by Tod, Fox, Paget, and myself. In Dr Tod's case the lungs were highly œdematous and did not collapse, when the thorax was opened, while the cases recorded by Dr Wilson Fox, Mr (now Sir James) Paget, and myself, agreed in exhibiting embolic patches

throughout the lower lobes. Dr Fox's, like Sir James Paget's, presented emphysema of the upper lobes; in Dr Fox's the lungs were, with the exception of the upper lobes, pneumonic, while in mine they were retracted, crepitated imperfectly, and showed chronic consolidation at their bases.

The pulmonary hyperæmia, in these cases, is explicable in different ways. In Dr Tod's and Dr Fox's cases, we may accept Dr Tod's theory, that the pulmonary congestion was a consequence of hypertrophy and dilatation of the chambers of the left heart, but in the cases related by Sir James Paget and myself, we must look for another explanation, as the left auricle and ventricle were normal in both these instances. The theory I have advanced in the general remarks on the case of E. W. G. M. appears to me to explain matters sufficiently. As I have already stated, *à priori* reasoning would lead us to expect an anæmic rather than hyperæmic condition of these organs, where we have an obstruction to the circulation at a point before the part congested, together with a normal condition of the cavities of the left heart. The hyperæmia or impeded pulmonary circulation is probably, therefore, due in such cases to the vis-a-tergo, consisting mainly of the action of the right ventricle being inadequate to the due propulsion of the blood through the pulmonary arterial into the pulmonary venous radicles. At any rate, the facts related justify the belief, that pulmonary hyperæmia occurs in cases of valvular disease of the right heart independently of any defect in the chambers of the left heart; and, if we admit such a possibility, we likewise admit the possibility of the occurrence of compensatory hypertrophy of the right ventricle as one of the initial rather than final events in some cases belonging to the class under discussion.

Where the pulmonary semilunar valves are perfect, a like pulmonary hyperæmia from imperfect systolic impulse would probably result, though perhaps more slowly, in cases of tricuspid obstruction or regurgitation from an imperfectly filled condition of the right ventricle, too little blood either entering that chamber (stenosis), or being projected from it (owing to regurgitation) towards the pulmonary radicles. In other words, we have a primary stasis limited to the venous section of the circulation, and a recuperative power manifested by the compensatory development of the right heart, independently of any participation in these changes by the left heart, whereby the system copes successfully, occasionally for a lengthened period, with the plethora of the venous circulation, sometimes maintaining the health of the vital organs in a high degree of perfection, and allowing patients from such valvular disorders to follow various occupations with comparative comfort.

Looking at the whole question, then, we shall probably find that here, as in most other matters, the truth lies in the "happy mean." In a certain proportion of cases, Dr Tod's theory of the sequence of events is probably correct, while in others the accessory development of the right ventricle is probably one of the first of

the chain of secondary changes. Our data are at present too scanty to allow of our distinguishing these cases exactly, but it is probable that the order indicated by Dr Tod will be found in cases where the venous engorgement is greatest, and where such engorgement from its very intensity is most apparent in the systemic venous circulation. The experiments with the rubber apparatus, to be related afterwards, seem to indicate that tricuspid lesions are more calculated to engorge the systemic venous circulation than any other form of valvular lesion taken singly. The left ventricle, therefore, under these circumstances would be most liable to hypertrophy in cases of disease of the right heart, when the tricuspid valve is defective, as Dr Tod pointed out. I have already, however, mentioned cases, and especially that recorded by myself, where there must have been serious tricuspid deficiency for a length of time, and where, nevertheless, the left ventricle had escaped hypertrophy. In my own and the other cases which had so escaped, and of which we have sufficient information (with the exception of Drs Begbie and Wilks's cases already commented upon), the right ventricle was hypertrophied, whether from a precedence of the pulmonary valvular lesion, or from pulmonary hyperæmia, it is difficult to say; yet it is very probable that such right ventricular hypertrophy was the principal agent in retarding the development in the other portions of the circulatory apparatus of the anatomical changes which result from retrograde stasis.

The sequence of events in these two classes may therefore be stated as follows, although, of course, either chain must only rarely be found complete in individual cases:—

Class I.	Class II.
1. Hypertrophy and dilatation of the right auricle.	1. Impeded pulmonary circulation with congestion of the lower lobes, with or without emphysema of the upper lobes.
2. Plethora of the systemic venous circulation, with disturbance of the functions of the parenchymatous organs.	2. Hypertrophy and dilatation of the right ventricle.
3. Impeded arterial circulation.	3. Hypertrophy and dilatation of the right auricle.
4. Hypertrophy and dilatation of the left ventricle.	4. Plethora of the systemic venous system with disturbance of the functions of the parenchymatous organs.
5. Hypertrophy and dilatation of the left auricle.	5. Impeded arterial circulation.
6. Congestion of the lower lobes of the lungs, with or without emphysema of the upper lobes.	6. Hypertrophy and dilatation of the left ventricle.
7. Hypertrophy and dilatation of the right ventricle.	7. Hypertrophy and dilatation of the left auricle.
8. Increase of pulmonary congestion.	8. Increase of pulmonary congestion.
9. Increase of systemic venous congestion.	9. Increase of systemic venous congestion.
10. Death.	10. Death.

We shall later investigate the influence which valvular disease of the right heart has upon the life of patients suffering from it, and shall then see in what measure the ultimate result is affected according as the secondary changes follow the order indicated in the one or the other of these two columns. In the meantime, as we have so far investigated the means employed by nature in coping with the problem of a difficult propulsion of the circulation arising from pathological causes, it will be instructive to examine the means she employs when an analogous difficulty is physiological.

Part II.

With this object, I purpose to institute a comparison between the circulatory apparatus and circulation of the sheep and that of man, and have composed the following table, which will show at a glance the comparative weight and dimensions of the heart in the two classes of animals. The measurements of sheep No. 1 were taken three days after death by "sticking," to allow the rigid systole of the heart to relax, that it might the more easily be overcome by gentle stretching; those of sheep No. 3 were taken seven hours after death, caused in a similar manner, and therefore during systole. As regards man, I have assumed 10 stones as the average weight of a healthy male. The weight of the blood effused by the latter is taken from the estimates formed by Weber and Lehman in the case of two criminals who were decapitated, and mentioned by Dalton in his treatise on *Human Physiology*, p. 215, ed. 1867. Were I to base my estimate of the amount of blood in the body of the sheep on the results of Weber and Lehman's investigations, the average would only amount to about 5½ pounds; but as the process of "sticking" a sheep and of decapitating a man are not identical, and as in the latter case greater facilities are afforded for the escape of blood from the cervical vessels, it is probably right to assume that the blood effused in "sticking" a sheep does not bear the same proportion to that which remains in its body, as does the blood which flows from the cervical vessels of a decapitated man to that left in *his* vessels. I have, therefore, broadly estimated the average of the whole quantity of the sheep's blood at from 6 to 8 pounds, but have no data to prove even the approximate correctness of this calculation.

The various estimates of the average weight of the male human heart will be found stated in Quain's *Anatomy*, vol. i., p. 321, seventh ed. 1867, where the proportionate weight of the male heart to the body is also given. If we assume that Dr John Reid considered 11 ounces to be the average weight of the male heart, and 1 to 169

Animal.	No.	Weight of animal (in case of sheep including wool).	Weight of blood effused.	Weight of heart.	Length of right ventricle.	Length of left ventricle.	Circumference of right ventricle.	Circumference of left ventricle.	Length of right auricle.	Length of left auricle.	Circumference of base of right auricle.	Circumference of base of left auricle.	Diameter of walls of right ventricle.	Diameter of walls of left ventricle.	Diameter of walls of right auricle.	Diameter of walls of left auricle.	Diameter of septum ventriculorum.	Diameter of lumen of right ventricle.	Diameter of lumen of left ventricle.	Circumference of tricuspid orifice.	Circumference of mitral orifice.	Circumference of pulmonary orifice.	Circumference of aortic orifice.	Condition of heart when measured.	Proportion of weight of blood to weight of body in sheep.	Proportion of weight of blood to weight of body in man.	Proportion of weight of heart to body in sheep.	Proportion of weight of heart to body in man.	Remarks on measurements of sheep.	Remarks on measurements of man.
Sheep.	1	7 st. 4 lbs. 4 oz. 8 oz.	4 lbs. 4 oz.	9 oz. 6 drs.	mm. 40 to 45	mm. 55	mm. 90	mm. 90	mm. 30 to 35	mm 20 to 35	mm. 100	mm. 90	mm. 5 to 7	mm. 15 to 20	mm. 1 to 4	mm. 1 to 7	mm. 15 to 18	mm. 60 by 5 to 11	mm. 25 by 25	mm. 100	mm. 90	mm. 60	mm. 60	A Systole.	6 or 8 lbs. is to 122 lbs. as 1 is to 15 or 20.	17½ lbs. is to 140 lbs. as 1 is to 8.	As 1 is to 196.	11 oz. is to 140 lbs. as 1 is to 203. 10 oz. is to 140 lbs. as 1 is to 224.	Other measurements of sheep No. 2 were all greater than those of No. 1, but were not minutely noted. Those of No. 4 not taken. Average weight of the four sheep, 8 st. 10 lbs. 10 oz.; of their effused blood, 4 lbs. 8 oz. 2 drs.; of their hearts, 10 oz. 1 dr. 15 grs.	Proportion of heart to body is, according to Keid, 1 : 169–173. " " " Glendinning, 1 : 158. " " " Tiedeman, 1 : 160.
	2	10 st. 5 lbs. 6 oz.	5 lbs. 6 oz.	13 oz. 2 drs.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...						
	3	9 st. 1 lb.	4 lbs. 4 oz.	8 oz. 5 drs.	80	65	90	75	30 to 35	25 to 35	90	80	5	9 to 20	1 to 3	...	2	30 by 4	15 by 15	90	80	70	...	Sys-tole.						
	4	9 st. 2 lbs. 3 oz.	4 lbs. 3 oz.	9 oz.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...						
Man (male).		10 st.	12·27 lbs.	9¾ oz. to 11 oz.	84·8	66·4	188·1	119·9	...	...	122·2	109·1	2 to 4	8· to 11·4	...	...	11·0	...	...	122·2	109·1	71·0 above valves.	69·6 above valves.	...						

or 173 the proportion which the weight of the heart bore to that of the body, we must assume that he considered the average weight of the body to be between 8 and 9 stones, for the proportion which 11 ounces bears to 140 pounds, or 10 stones, is as 1 : 203 ; and if we assume the average weight of the male heart to be 10 ounces, and the average weight of a healthy man 10 stones, we get the ratio of 1 : 224. To justify my assumption of 10 stones as the average weight of a healthy male, I may direct attention to the fact that Mr Gathorne Hardy, Secretary of State for War, stated in his speech during the discussion of the Army Estimates, on March 4, 1878, as reported in the *Daily Telegraph* of March 5, that 1452 men taken from 21 different corps, and whose average age was 20 years and 1 month, weighed on an average 10 stones 7 pounds.

The exact measurements of various parts of the human heart are those given by M. Bizot in his " Recherches sur le cœur et le système artériel chez l'homme," *Mémoires de la Société Médicale d'Observation de Paris*, 1836 ; transcribed by Hasse in his *Anatomical Description of the Diseases of the Organs of Circulation and Respiration*, p. 158, Sydenham Society's edition, and reduced by Dr Moore to English measure for Dr Stoke's work on *Diseases of the Heart and Aorta*, p. 257, note ; 1854. I have adhered throughout to the measurements of the male heart, and have reduced Bizot's calculations to the metrical scale, to be in conformity with the measurements given of the sheep's heart. On examination the table will show that the sheep, though an animal having a less average weight than man, possesses a heart of almost the same average weight as the latter ; and, moreover, that the main distinction between the hearts of the two animals is, that while the capacity of man's heart is greater than that of the sheep, the muscular power of the heart of the latter, as indicated by the diameter of the walls of the various cavities, is as undoubtedly greater than that of the former. That the capacity of the sheep's heart should be less than that of man's is what we might expect from the smaller quantity of blood circulating in that animal, but this circumstance increases the importance of the fact that the muscular power of the lower animal is still proportionately greater than that of man. It is the investigation of the causes of this muscular preponderance in the sheep's heart which will, in my opinion, assist us to solve the problem of the means best adapted for the due propulsion of an impeded circulation.

The principal vascular channels in the circulatory apparatus of the sheep may be considered horizontal, the chief exceptions being the vessels which supply the limbs, the cranium and its contents, the testicles, and the lungs.

The principal vascular channels in the circulatory apparatus of man may, on the other hand, be considered perpendicular. The first effect of this grand difference may be demonstrated by the fol-

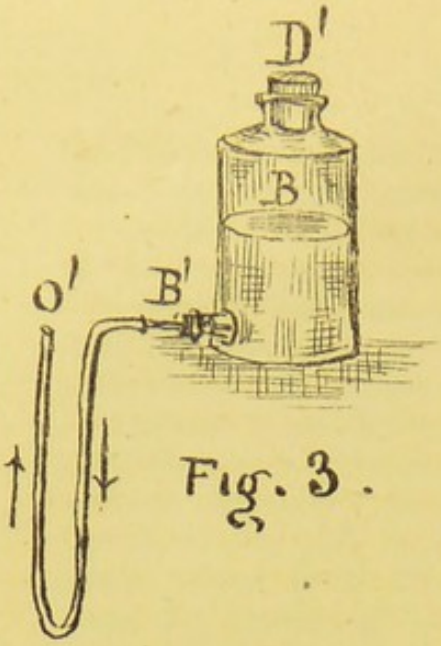


Fig. 3.

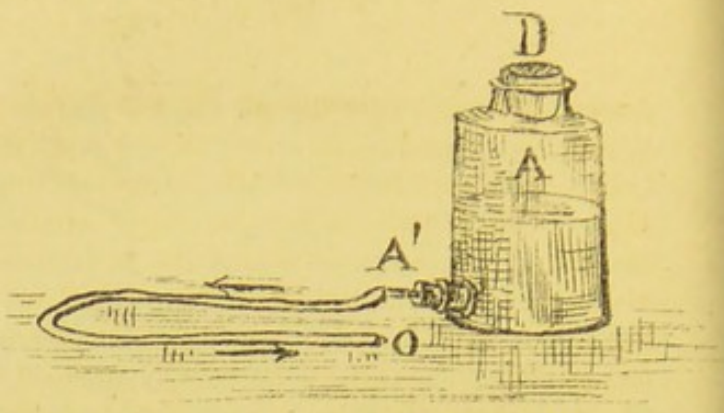


Fig. 2.

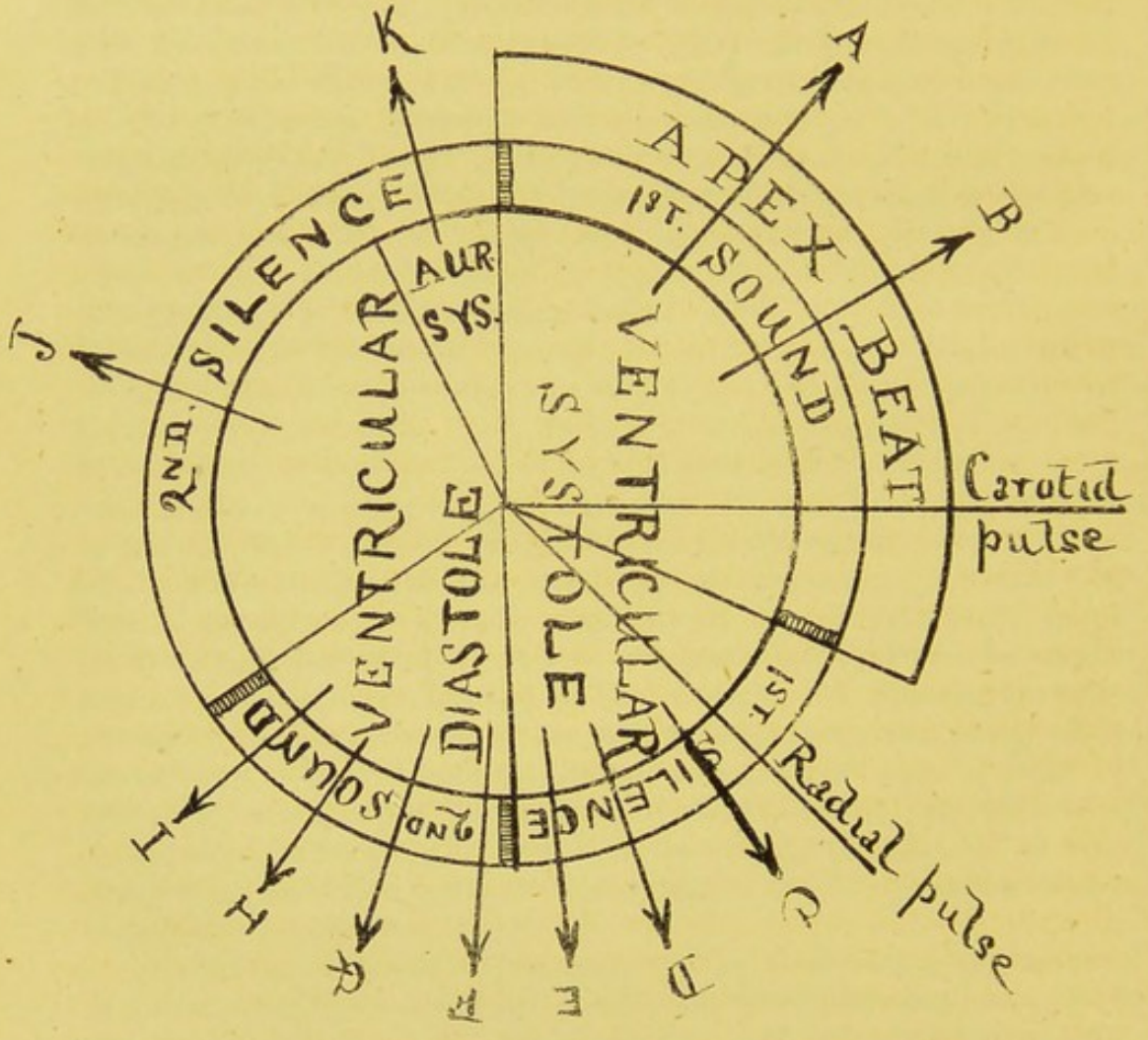


Fig. 4.

lowing simple experiment, in which the propulsive force of the left ventricle is excluded. (Figures 2 and 3.)

A and B are bottles with an outlet and neck at the base A′ and B′, to which tubing of the same length and calibre C and C′ is attached. C is horizontal, C′ is perpendicular. The bottles A and B contain water, and are corked. When these corks D and D′ are removed, water begins to fall in the bottles, and circulates through the tubing, but issues decidedly more forcibly at the orifice O′ of the tubing C′ than at the orifice O of the tubing C, in accordance with that law of hydrostatics which states that the pressure in each layer of fluid is proportioned to its depth, and that the upward pressure of a column of liquid is governed by the same laws as the downward pressure; the depth of the column of fluid in C′ is manifestly greater than in C, and hence the result.

This experiment represents the vascular system in the sheep and in man respectively, excluding, as I have already stated, the heart,—the imaginary points taken being the commencement of the descending aorta, and the entrance into the right auricle of the vena cava inferior. Hence, without considering the effect of the propelling power of the heart in either case, we may conclude that the horizontal circulation of the sheep, on hydrostatic principles, would empty itself less forcibly into the right auricle than the perpendicular circulation of man. As, however, every animal to be healthy must have a circulation adapted in vigour to the necessities of its own body, and to the medium in which it exists, and, moreover, as both the sheep and man are mammalian, and exist in the same medium, the circulation of both must be vigorous for the due maintenance of health. As we have seen, however, the propulsive force intrinsic to the circulation of the sheep is less than that in the circulation of man.

In order, then, to have a circulation equalling in perfection that of the sheep's fellow-mammal man, we must have a greater propulsive force extrinsic to the actual circulation of the former. Hence the preponderance of the muscular development of the left ventricle of the sheep's heart over that of man. But, as I have stated, there are important exceptions to the general horizontality of the circulation of the sheep, and the first of those I mentioned was that portion of the vascular system distributed to the head and neck. Now, to propel blood along the oblique cervical and cranial vessels of the sheep, is manifestly an easier task than effecting the same object in that section of the circulation in man, which is like the rest perpendicular or upright, but where the *vis-à-tergo* is inappreciable in the circulation itself, and where the ventricle has to propel its contents in an upward direction, and against the force of gravitation. This section of the human circulation would, therefore, require a more powerful propulsive organ than the same portion of the circulation of the sheep. The fact, however, is, that the left ventricle of the sheep is more powerful

than that of man, and therefore excessive for this purpose. Excessive force, however, in a portion of the circulation which embraces so vital an organ as the brain, would probably be productive of conditions incompatible with health, as is instanced by the cerebral symptoms exhibited by patients in whom the left ventricle is over compensated. If it is true, therefore, that the power of the left ventricle of the sheep is excessive for the purpose under consideration, and if this is so in all cases in animals of this species, Nature would belie her usual ingenuity were she to neglect the due protection of such an important organ as the brain from this excessive force. But we find that she rises to the emergency with her wonted ability, and by spreading that marvellous net at the base of the brain which extorted the wonder of Galen, when he called this portion of the circulatory apparatus in the sheep the *rete mirabile*, we see her recognising at once the surplus force of the left ventricle, and taking efficient means for the protection of the all-important contents of the cranium. Over the rest of the cervical and cranial surface the excessive force may be harmlessly expended, and is probably lost in contributing to the due propulsion of the general circulation. Granted, then, that we have in the propulsive power of the left ventricle a force sufficient to counterbalance the defective impulse in the circulation in horizontal channels, we may consider those portions of the circulation in which this surplus force is not necessary on account of horizontal impediment. That the more or less perpendicular vessels of the limbs are not materially modified in the sheep, may probably be explained in the same manner as I have explained the expenditure of surplus force in the general circulation of the head and neck; but, when we come to another pendulous and vascular organ of the first importance, viz., the testicles, we can again imagine the injurious influence of a too powerful afferent circulation, and again we see in the long and tortuous spermatic arteries of the ram a safeguard against injury from this cause. To prove the truth of this argument, however, we should find a difference between the nutrient vessels of horizontal and perpendicular organs of equal importance and vascularity, and an examination of the arteries of the kidney in the sheep will convince us on this point. For, whereas the spermatic arteries are, as I have stated, long and tortuous, to diminish by friction over an extensive surface the superfluous impulse from the heart, the renal arteries are proportionally no longer than those of man, and are perfectly straight, showing the necessity for all the impulse possible to drive the circulation aright through the horizontal organ.

So much for the left ventricle. Let us now examine the physical condition of the right ventricle.

The muscular development of the right heart of the sheep is proportionally greater than that of the corresponding chamber in the human heart, and this is probably accounted for by the relative

position of the lungs in the two animals. In the sheep these organs may be considered to be superimposed upon the heart, and to lap it round from above downwards, whereas the lungs in man may be said to be mainly behind the heart, and to surround it from behind forwards. The result of such an arrangement is, that blood propelled by the right ventricle into the pulmonary circulation of the sheep must rise, to some extent, in defiance of gravitation, whereas the pulmonary vessels of man are so placed that a considerable portion of the pulmonary circulation lies below the level of the principal branches of the pulmonary artery, and the blood is assisted instead of impeded in its progress by the law of gravity. Hence, I believe, the proportionately stronger right ventricle of the sheep. But, as I shall presently show, the greater the muscular development of the ventricles, the greater the resiliency of these chambers after systole, and the more powerful their suction force towards the ventricle, provided the capacity of the latter be not at the same time diminished. The influence, therefore, exerted by the more powerful right ventricle of the sheep in suction is greater than that exerted by the thinner right ventricle of man, and any advantage which might be obtained by the latter in this respect, in consequence of its greater capacity, is nullified by its having to deal with a larger quantity of blood than the same chamber in the sheep. The lungs, then, are so placed in this animal as to necessitate by their position a powerful ventricle for the due propulsion of blood through the pulmonary circuit, and by so doing at the same time contribute an absorptive force of greater power than that in man, to assist in conducting a circulation naturally more difficult than in the latter.

What I have stated with regard to the sheep probably applies more or less to the majority of quadrupeds, but, not having personally investigated the matter in other animals, I am unable to state this as a positive fact. We know, however, that the spermatic arteries of the bull are like those of the ram, long and tortuous, and that in many of the lower animals *retia mirabilia* exist. We may, therefore, conclude that in such animals there are also those conditions of the circulation and circulatory apparatus which we have seen require the presence of these bulwarks against excessive impulse in the sheep.

The information derived from this comparison of the vascular system of a biped and a quadruped, appears to me to warrant the conclusion, that the most perfect compensation in cases where the venous circulation is impeded from any cause, whether valvular disease of the right heart or obstruction in the pulmonary circulation arising in the manner already described from such valvular disease, or from an independent chronic lung affection, would be by an accessory development of both the right and left chambers of the heart, the former supplying by suction a *vis-à-fronte* to supplement the great suction force of respiration, and the latter an

increased *vis-à-tergo* to urge forward the sluggish column of venous blood, and overcome the resistance of the obstructed arterioles. But, as we have seen, such a perfect arrangement in the lower animals requires a special provision to be made for it in other portions of the circulatory apparatus and organism generally, which, though capable of being produced by a *vis naturæ*, are beyond the power of a *vis medicatrix naturæ*. And, moreover, it must be remembered that the strong walls of a quadruped's heart do not in any way encroach upon the normal capacity of the chambers contained by those walls, whereas in growth compensatory for disease, we find that hypertrophied walls either encroach materially on the capacity of a ventricle when concentric, or, by contemporaneous dilatation, increase that capacity, both of which circumstances when affecting the left ventricle, as will be shown when considering the state of that chamber, are injurious in cases of valvular disease of the right side of the heart. The right ventricle, from its naturally greater capacity in the human subject than the left ventricle, and its thinner walls, is not so liable to concentric hypertrophy as the corresponding chamber in the left; and its increase of capacity, provided the walls were not degenerated, would not materially influence the efficiency of this chamber, which we at present suppose to be already incompetent from valvular disease; but, on the contrary, if at once powerfully hypertrophied and dilated, would rather from its increased capacity have likewise a greater suction power, and therefore assist rather than impede the circulation of the blood.

Hypertrophy with or without Dilatation of the Left Ventricle.

The analytic table given at page 20 shows us that of twelve cases of valvular disease of the right heart, seven escaped hypertrophy of the left ventricle, whereas in only four was hypertrophy of the right ventricle absent. The table likewise shows us that Dr Tod's case was the only one of the series which exhibited accessory growth of all the chambers of the heart, and that of the other cases which had undergone hypertrophy of the left ventricle, one (Dr Foster's) had an hypertrophied right auricle, without a similar condition of the right ventricle, and in the other (Dr Wilson Fox's) there was an hypertrophied right ventricle, with a normal right auricle, as we may conclude from the condition of the latter not being specially mentioned in an otherwise exhaustive report, while the other cases which exhibited hypertrophy of both the right chambers presented a normal left ventricle and auricle. In other words, we find in these cases of valvular disease of the right heart that the more perfect the compensation in the right chambers from accessory growth of their walls, the less do the left chambers deviate from their normal condition, from which we may

conclude that under such circumstances there is least obstruction in the circulation.

The case recorded by Dr Begbie, in which all the chambers were normal, cannot by any means be regarded as having been so seriously incompetent in the action of the defective valves as the other cases recorded, and since the obstruction to the circulation was slight, we are not surprised to find that secondary changes in the circulatory apparatus were inappreciable.

Had the case survived some years these negative conditions would probably have altered. Although we can thus account in a great measure for the absence of hypertrophy in Dr Begbie's case, it is not so easy to explain the absence of such change in the case recorded by Dr Wilks, where there was not only a very serious valvular lesion, but likewise extensive disorganization of the lungs, which must have maimed the efficiency of the great respiratory aid to the circulation.

Can it have been that the absolute obliteration by disease of a considerable portion of the pulmonary capillary circulation caused a more energetic pressure on the unobliterated or, as it were, collateral portion of the same circulation, allowing a quantity of blood to be driven through that portion when the heart would have been incapable of effecting the same purpose through an extensive capillary network? Be this as it may, I am inclined to regard this case as important evidence of the power of the upward pressure of the venous column in an erect animal, which probably assisted very materially in the propulsion of blood through the right chambers. The point, however, which I am most desirous of investigating is the influence upon a circulation rendered difficult by defective valvular action in the right heart, of hypertrophy, with or without concomitant dilatation of the left ventricle. With this object I shall first consider briefly the function of the healthy ventricle in a normal circulation, and shall have occasion to discuss some points not usually treated in text-books, etc.

There is not much difference of opinion as regards the mechanism and function of the left ventricular systole. That the complete filling of the ventricle with blood is the incentive to its contraction, when the walls, closing on their contents, shut the auriculo-ventricular orifice by means of the mitral valves, the closure of which, and opening of the aortic semi-lunar segments, determine the direction of the blood-current, which passes forward into the aorta till the commencement of the latter is distended with the blood recently occupying the ventricle, is admitted by all, as also is the evident object of the ventricular systole to distribute the aerated and unaerated blood through the systemic and pulmonary arteries respectively. But a similar unanimity does not prevail either as regards the mechanism or function of ventricular DIASTOLE. The following views of the mechanism of this event I transcribe from Dr T. Hayden's excellent text-book of *Diseases of*

the Heart and Aorta recently published:—"The cause of the initiation of diastole has been by some writers considered to be the impetus of the blood propelled into the ventricle by the auricle during contraction of the latter cavity. In refutation of this doctrine, it is sufficient to observe that active contraction of the auricles takes place only at the end of ventricular diastole, and immediately previous to ventricular systole. Longet maintains that the movement of ventricular diastole is due to the twofold cause of the elasticity of the walls of the ventricle, by which, after contraction, they tend to separate, and thus attain a state of equilibrium, as does a caoutchouc bottle, and the influence of a virtual void in the chest created by the retraction of the lungs in expiration. I (Dr Hayden) believe the ventricles, especially the left, possess normally an intrinsic power of expansion in virtue of their elasticity, but I also hold that this intrinsic power is materially supplemented by the extrinsic force of auricular reaction and contraction operating through the medium of a large mass of blood, the mere weight of which contributes in some degree to the expansion of the ventricles. It should be remembered that ventricular diastole cannot in health coincide with expiration oftener than once in every four pulsations of the heart, and it may not even so often. Dr Garrod maintains the existence of an ACTIVE diastole of the ventricles, dependent upon the active turgescence of the walls of the heart, 'consequent on the flow of blood into the coronary arteries immediately after systole,' the auricles from their thinness not being similarly affected. He endeavoured to prove this by injecting water backwards into the aorta of a sheep's heart. He further concludes that during ventricular diastole there is in operation an absorptive force or attraction towards the ventricles. Independently of the objection, that under the influence of the same agency the auricles are supposed to contract, and the ventricles to dilate, it must be remembered that in Brackyn's experiments the alternate action of the auricles and ventricles was maintained without a coronary circulation of any kind."

I have failed to discover any detailed account of Brackyn's experiments, but have carefully repeated Garrod's experiment as related in the *Journal of Anatomy and Physiology* for 1869, vol. iii. p. 391, and have likewise performed some modified experiments on the same principle, which I shall presently relate. The probability of the existence of an absorptive force towards the ventricles had suggested itself to my mind before I was aware of the researches of others on this point, from a study of the case which forms the text of this thesis. The very imperfect action of the right ventricle in completing the pulmonary circuit, coupled with a regular pulse at the wrist, and an almost entire absence of anasarca, led me to suppose that the left ventricle exercised an absorptive influence on the pulmonary, as the right did upon the systemic venous circulation, and it was a desire to determine the

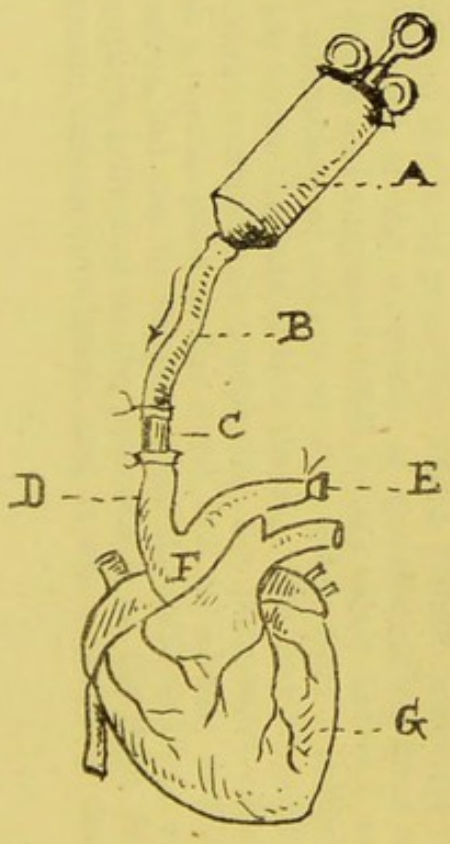


Fig. 5.

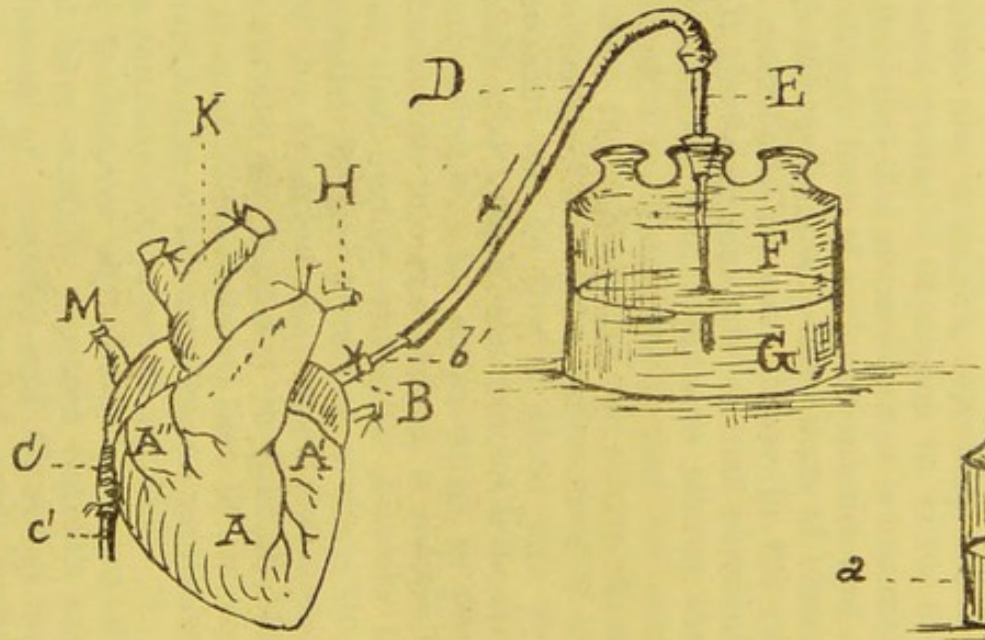


Fig. 6.

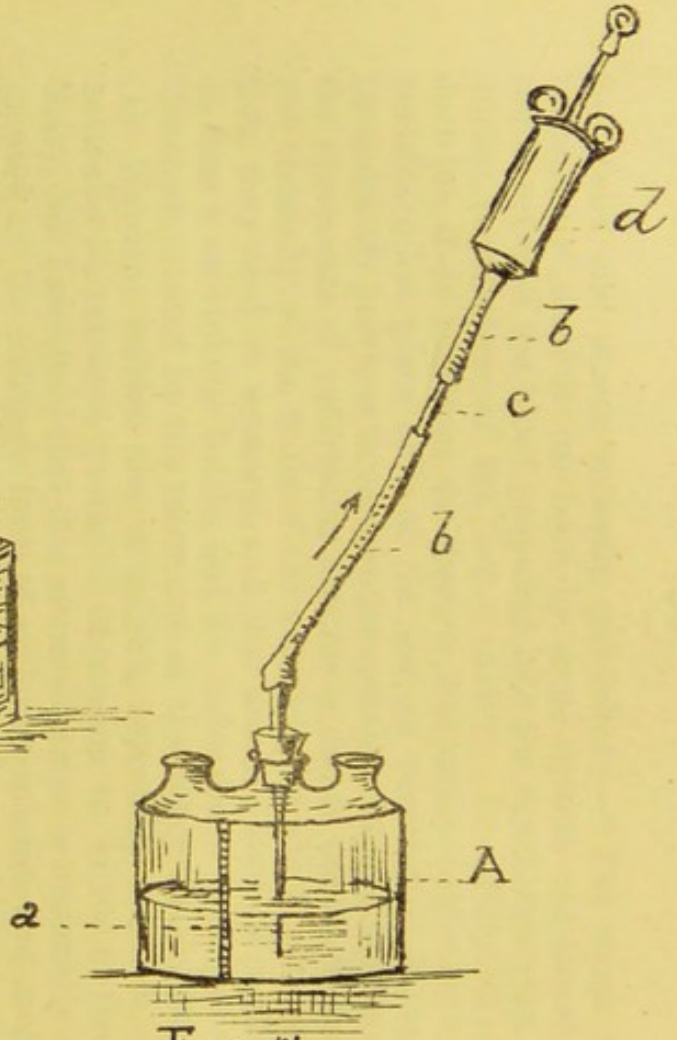


Fig. 7.

truth or falsity of this supposition which prompted me to inquire whether my belief in such an absorptive force was shared by others. Accordingly, having become acquainted with Dr Garrod's views, to which I mentally assented without having tested their truth, I proceeded to repeat his experiment, which I did as follows:—

Experiment No. 2 (Figure 5).—A is a syringe capable of holding 2½ ounces of water; B is a piece of india-rubber tubing connecting the syringe A with a piece of lead tubing C, such as is generally used by gasfitters. The latter (C) connects B and the innominate artery D, which is ligatured to maintain the connexion. E is the arch of the aorta (F), likewise ligatured; G is a sheep's heart. The syringe having been filled, water was gradually injected into the artery. With each syringeful the heart was seen to swell out and expand, and water was observed to complete the coronary circuit, being injected, per aortam, into the coronary arteries, and seen to return, mixed with air, by the coronary veins, from which water escaped when they were pricked, and most forcibly at the time of injection of a fresh syringeful. When the engorged walls were compressed so as to obliterate the lumen of the ventricles, they showed considerable resiliency when released from compression, rebounding to their former size and re-establishing the ventricular cavities. The left ventricle, apparently from its greater thickness, exhibited this property in a higher degree than the right. While water thus injected expanded the ventricles, it also, though to a much less extent, seemed to me to expand the auricles. These appeared to rise partly from elongation of the ventricular portion of the heart, but were also themselves seen to elongate slightly, but only in their more muscular portion (*i.e.*, along the auriculo-ventricular groove), aided possibly by the filling of the coronary sinuses. A transverse section was then made of the heart about its middle, when the following measurements were taken:—

Diameter of lumen of left ventricle,	.	.	.	20 millimetres.
„ „ right „ transversely,	.			10 „
„ „ „ „ antero-posteriorly,				40 „
Average thickness of right ventricular walls,			.	11 „
„ „ of septum ventriculorum,			.	25 „
„ „ of left ventricular walls,			.	20 „

Experiment No. 3.—Water now injected into the coronaries of the divided heart showed that while the capacity of the ventricles was increased, as compared with that observed in systole, the transverse diameter of the walls themselves was also at the same time increased, thus representing a state of dilated hypertrophy.

To ascertain whether the diastolic ventricle exercised any absorptive influence towards itself, and to determine at the same time the comparative absorptive power of the two ventricles, and

the influence on this power of turgescence of the coronary blood-vessels, I devised the following experiment:—

Experiment No. 4 (Figure 6).—A is a sheep's heart, with the left and right ventricles A′ and A″, with glass-tubing *b′* tied into B, one of the pulmonary veins; C is the inferior cava, likewise having a glass-tube tied into it; D is a piece of india-rubber tubing which may connect either B or C with E, a graduated glass pipette passed through a cork into the central aperture of a three-necked jar F containing water G. H, K, and M are the pulmonary artery, aorta, and superior vena cava respectively. H and M may be ligatured at once, and K grasped in the first instance by the forefinger and thumb, so as to cut off the entrance of air into the ventricle during the first step of the experiment, which is as follows:—

If the left ventricle A′ be compressed and then released from pressure, the chamber will resume its original form, and at the same time water will rise in the graduated pipette to the height of about 30 millimetres; with each compression this column will be depressed and again raised on expansion. If the pipette and tubing be then attached to C′, and the right ventricle similarly manipulated, the column of water will rise, but less appreciably.

If we now perform Garrod's experiment, and fill the coronary arteries per aortam with water, and then ligature the aorta, we shall find that compression and relaxation of the left ventricle raises a column of water 120 millimetres high, while if the tubing and pipette be connected with the vena cava inferior, the absorptive power of the right heart is represented by a column of water measuring 30 millimetres, which, though much less than that of the left ventricle, is, under present circumstances, about the same as that resulting from diastole of the left ventricle in an uninjected state. If again the pipette be connected with the aorta and pulmonary artery, and the pulmonary veins and inferior cava ligatured, compression and expansion of the ventricular heart will raise a column of water in the pipette, but less appreciably than under the circumstances already related, and always less in the pulmonary artery than in the aorta.

These experiments tend to prove—(1), that the fibro-muscular substance of the heart has, like all such structures, an inherent resiliency; (2), that this resiliency comes into play during the period of diastole; (3), that this diastole of resiliency has, *per se*, an influence, though comparatively slight, in absorbing fluid towards the ventricles—most powerful by way of the pulmonary veins, and to a less extent by way of the aorta, while the absorptive power of the right ventricular diastole is less evident than that of the left; (4), that such inherent resiliency is increased by injecting the coronary system, when water is raised much more powerfully than when fibro-muscular elasticity alone is active, proving alike the greater resiliency of the ventricular walls, and

(as may be verified by section) a somewhat increased capacity of those chambers. As Dr Norman Cheevers long ago stated in vol. i. of the second series of *Guy's Hospital Reports*, "the coronary arteries have a close resemblance in the arrangement of their tissues to the large superficial veins of the extremities; they indeed appear to possess an intermediate gradation of structure between the vessels of the arterial and venous systems." Such vessels, therefore, are incapable of maintaining a perfect and independent circulation in so active an organ as the heart, whose alternate dilatation and contraction must be regarded as the principal agents in the filling and emptying of the coronary vessels.

Their arterial supply is furnished during the heart's diastole, when blood forced into the aorta rebounds from the elasticity of that vessel upon the aortic segments; they are emptied by cardiac systole compressing the substance of the heart as one squeezes a sponge, assisted by cardiac diastole, which exercises a *vis-à-fronte* in the right heart, and a *vis-à-tergo* at the aortic sinuses of Valsalva, fresh blood entering the coronary arteries, and exercising a capillary pressure on the corresponding veins. If the foregoing statements are capable of proof, the entire sequence of events in the mechanism of the heart's action would be—(1), ventricular systole, with closure of the mitral and tricuspid valves, and opening of the aortic and pulmonary segments, blood the while rushing through the aortic and pulmonary orifices to fill the commencement of the aorta and the pulmonary artery; this coinciding with the apex beat, first sound, and carotid pulse; (2), inherent resiliency of the ventricular heart beginning to assert itself, and tending towards diastole, with partial closure of the aortic and pulmonary valves, and partial relaxation of the auriculo-ventricular valves, and probably the oozing of some blood by its own weight into the ventricles, these events taking place during the first silence and synchronous with the radial pulse; (3), elastic recovery of the aorta and pulmonary arteries from distension, with rebound of a column of blood on to the semi-lunar segments, completing the closure of the latter, and producing the second sound; (4), turgescence of the coronary circulation completing diastole, which is favoured by the fibro-muscular resiliency of the ventricles; (5), opening of the auriculo-ventricular valves, allowing every facility for emptying the auricles by the gravity of the blood and the absorptive power of the ventricles, and supplying at the same time a *vis-à-tergo* and *vis-à-fronte* to empty the coronary veins, these events likewise coinciding as nearly as possible with the second sound; (6), commencing auricular systole under the absorptive influence of ventricular diastole; and (7), active auricular systole completing the ventricular repletion, and thus exciting the succeeding systole, and both occupying the period of the second silence. With reference to the latter event, I would suggest that although we have good reason to suppose that auricular systole is active in

the production of the presystolic bruit, of mitral or tricuspid constriction, as regards the usually abrupt termination of that murmur, the first and softer portion of the bruit, while it is, as it were, gathering strength for its ultimate sharp definition, is a result of the absorptive power of the ventricle towards the completion of diastole, when the coronary turgescence and quadrupled resiliency of the organ suck, as it were, the blood through the constricted aperture.

The annexed modification of Professor W. T. Gairdner's well-known graphic representation of the sounds of the heart, copied from Dr Balfour's *Clinical Lectures on Diseases of the Heart and Aorta*, p. 37, will present the foregoing text more clearly to the eye. (Figure 4.) The barbed radii pointing to alphabetic characters correspond in regular sequence with the events detailed in the text under the consecutive numerals.

I have now endeavoured to prove that there is an absorptive force in the diastolic ventricle, and have likewise tried to indicate a rational mechanism for its production. As Dr Hayden has pointed out in the work already quoted, the diastole of the heart cannot be ascribed in any measure to the thoracic vacuum created by expiration, as this act only coincides with about one of four diastolic movements; and I would suggest further, that in the diastole of the left ventricle we have a counteracting force to the thoracic vacuum referred to. The tendency of the latter is to facilitate the flow of blood from the venous circulation into the lungs, but not to promote directly the entrance of aerated blood into the left heart. The absorptive power of the right ventricle is weak, to avoid causing collapse of the great venous trunks which enter the right auricle.

The absorptive power of the left ventricle, on the other hand, is great, to overcome the retentive power of the thoracic vacuum, which, together with the intervention of the left auricle, saves the pulmonary veins from collapsing under the influence of the powerful diastolic suction of this ventricle.

If the existence of such a power be granted, let us endeavour to determine—(1), what the consequences of its diminution would be upon the pulmonary circulation, and through the latter upon valvular disease of the right heart; and (2), the circumstances which tend to diminish it.

I shall briefly consider the latter first.

The circumstances capable of impairing this force are—(1), diminution of the cavity of the ventricle; (2), increase of the capacity of the ventricle, with loss of power in its walls, which implies valvular incompetency; (3), increased capacity without loss of muscular power, but with valvular incompetency.

The capacity of the ventricle may be diminished, either by hypertrophy naturally produced, which if unaccompanied by dilatation is usually concentric, or artificially by the improper

use of drugs which are found useful in the removal or amelioration of conditions resulting from dilatation, and above all by digitalis. The more detailed discussion of the latter I shall reserve till I speak of treatment. It is scarcely necessary to insist at greater length, that the less the capacity of a suction apparatus, or, in other words, the smaller the vacuum producible by such an apparatus, the less its absorptive power, but to be convinced of this fact the following experiment may be performed :—

Experiment No. 5 (Figure 7).—A is a three-necked jar containing water, into the central neck of which a pipette is passed, through a cork ; the jar is graduated with a millimetre and centimetre scale ; *b* is a piece of indiarubber tubing, the calibre of which is about 5 millimetres, and its length (including 21 centimetres of glass tubing, *c*) 135 centimetres ; *d* is a syringe (*α*) of 2½-ounces (*β*) of 1 ounce capacity. The syringe is held at a higher level than the jar, and slowly exhausted. Exhaustion of the 2½-ounce syringe caused the water to fall 1 centimetre in the jar, and to traverse the whole length of the tubing and pass into the syringe, while the 1-ounce syringe only depressed the level of the water in the jar 3 millimetres, and the portion withdrawn only traversed 110 centimetres of tubing, its highest point being within 8 centimetres of the upper end of the glass tubing C.[1]

Hence, we may conclude a diminished capacity of the left ventricle, notwithstanding an increase in its muscular power, will likewise lessen its power of absorption, and from this cause, as well as from the passage of a smaller quantity of blood into the chamber during diastole, pulmonary engorgement is favoured, and the propulsive power of the right ventricle hampered. It may be argued that such untoward consequences of a diminished capacity of the left ventricle would be obviated by its more frequent and more powerful action, and where we have to deal with an otherwise healthy heart this is probably the case ; but if we have an imperfect right heart and retarded venous circulation, and it is with such cases we are at present concerned, matters are altered. Here the consequences of an energetic and frequent action of the left ventricle would (on account of the imperfect action of the corresponding chamber on the right) tend to fill the coronary circulation more rapidly than it could be emptied, causing an engorgement, having in its train, as we shall afterwards see, gradual dilatation and valvular incompetency of the left ventricle, with an aggravation of the deficiencies in the right heart. When, on the other hand, the capacity of the left ventricle is increased and the power of its walls diminished, that is to say, when it is dilated and its walls degenerated, we cannot have perfect valvular action, especially at the auriculo-ventricular orifice. We can, therefore, easily understand the baneful conse-

[1] Caoutchouc bags are useless for this experiment, as they cannot resist the atmospheric pressure, and when vacuous collapse.

quences of an imperfectly emptied auricle, with its consequent dilatation and pulmonary congestion, or at least impediment; and it cannot surprise us, that the deficient chambers in the right side of the heart, even when hypertrophied, and assisted by the upward pressure of the venous column, fail to surmount such a barrier in the circulation. As regards the third cause of impairment of the absorptive power of the left ventricle, viz., the increase of its capacity *without* loss of muscular power, there is no doubt that such cases of eccentric hypertrophy (or hypertrophy with dilatation) frequently occur, but there is as little doubt that such cases are usually the result of retrograde changes in a heart in which hypertrophy at an early period probably preponderated over dilatation. If such a condition is compatible with perfect valvular action at the aortic and mitral orifices, the absorptive power of the ventricle would be increased, and instead of being detrimental when we have valvular deficiency in the right heart, the logical inference from the preceding argument is, that it would be positively beneficial. But, unfortunately, such a period, if it ever exists *in the cases we have to do with*, would be but transitory, and would soon give place to imperfect valvular action, with dilatation of the auricle, and its sequelæ already mentioned. To illustrate these points more fully, the following experiments may be performed with the simple apparatus described (Figure 8).

A is an ordinary "Higginson's syringe," representing the right ventricle, connected at one end by means of indiarubber tubing with a glass tube G bent at a right angle, which represents the pulmonary artery and its branches, and at the other also by means of rubber tubing with the jar D, where the systemic venous and arterial systems unite. C is a three-necked jar containing water, in which the central neck, K, and the air within the jar, represent the trachea and bronchi, while the water, W, represents the pulmonary circulation. The bent tube, G, passed through a cork in one of the necks, represents the pulmonary artery, while a similar tube, H, issuing from the other neck, represents the pulmonary veins; B, also a "Higginson's syringe," is the left ventricle connected by tubing with H on the one hand and D on the other. D is a jar containing water representing the systemic capillaries, and the source of supply for the water intended to circulate through E, A, L the venous, W the pulmonary, and M, B, F the arterial circulation.

If B be either tied so as to prevent full expansion, or if it be prevented from expanding fully by compression with the fingers, when it is worked so as to imitate systole and diastole, water entering C, either by syphonic force alone or by this in addition to the influence of A worked in like manner to imitate systole and diastole of the right ventricle, accumulates in the jar C more rapidly, tending to fill it, than if B be either unbound, so as to expand fully by its elasticity before being

Fig. 8.

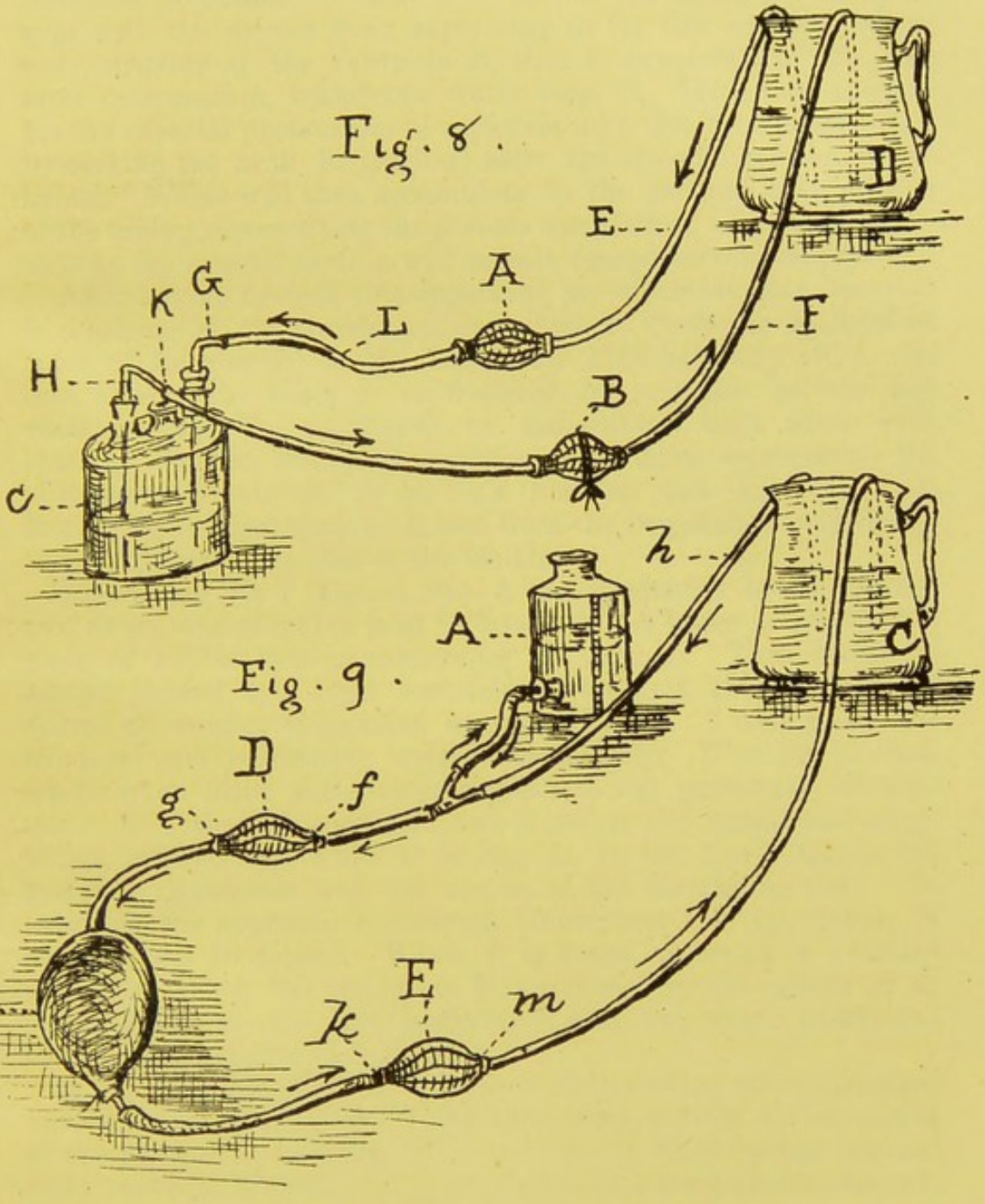


Fig. 9.

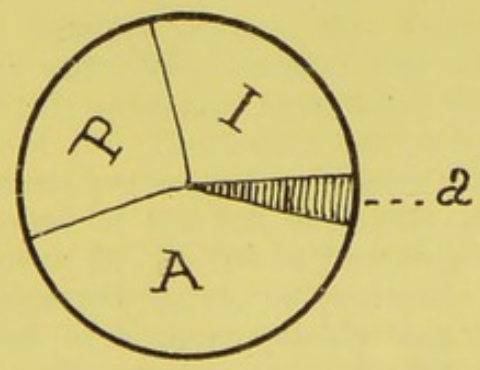
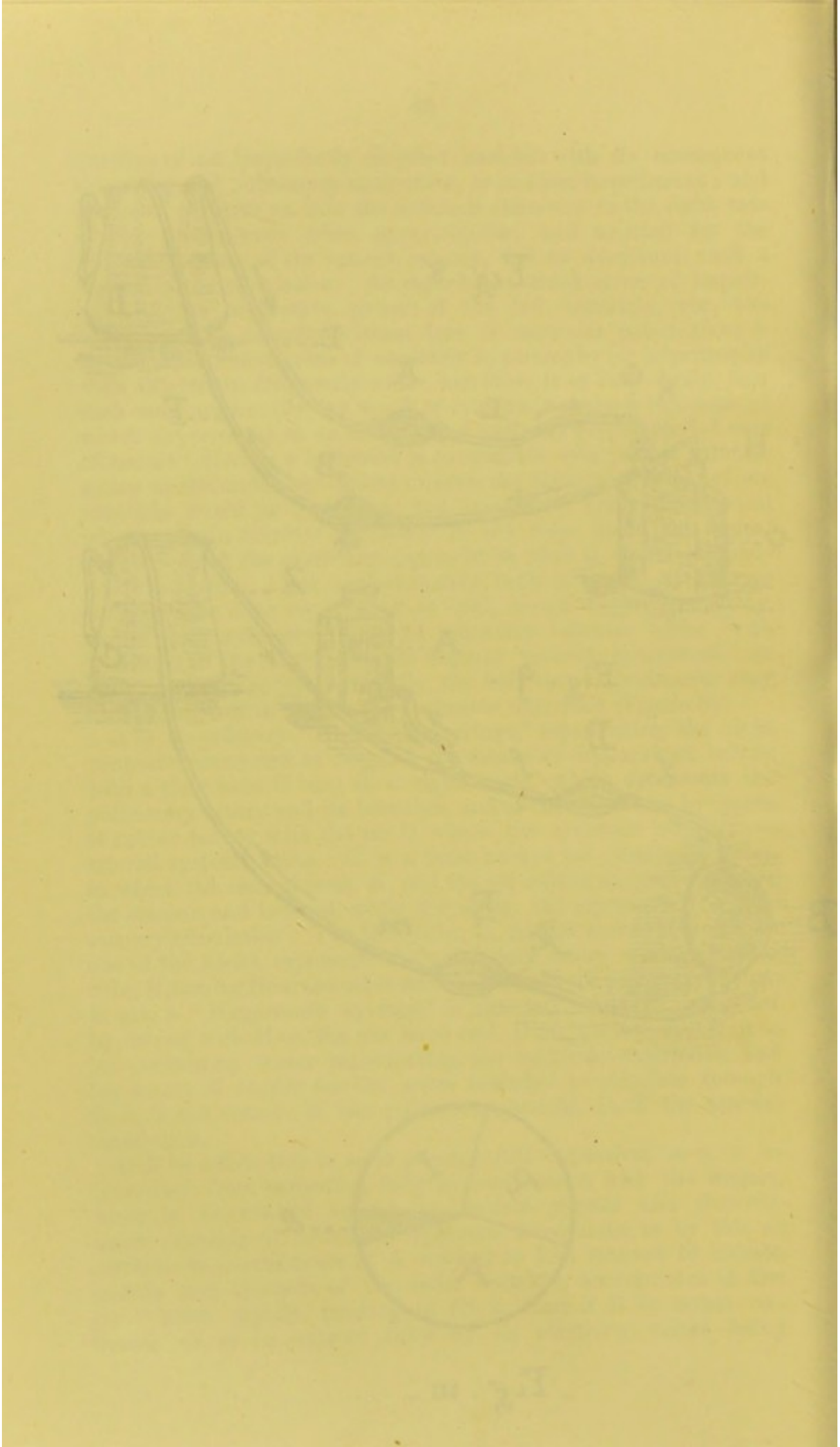


Fig. 10.



squeezed in systole, or than if it be not prevented by compression with the fingers from expanding to its full extent. Moreover, diastole of the ventricle B, that is expansion of the bag after compression, withdraws water from C. With this apparatus the vascular phenomena of asphyxia may also be demonstrated, by corking the neck K (trachea) after the circuit has been established. Water will then accumulate in the jar and that portion of the tubing representing the venous circulation, while that representing the arterial section will remain comparatively empty.

After having devised this apparatus, my attention was directed to a scheme of the circulation, described by Professor Rutherford (now of Edinburgh) in the *Lancet* for 17th February 1872. In that scheme the heart is represented by a single auricle and ventricle, and the capillaries by indiarubber bags filled with sponge. In the following experiments I have represented the pulmonary circulation by an ox's bladder, and mention Prof. Rutherford's apparatus, as it was from the bag-shaped capillaries that I borrowed the idea of the bladder.

Experiment No. 7 (Figure 9).—A is a graduated bottle, having two necks, one of which is at its base; to the latter is attached a piece of rubber tubing connecting it with *h*. The bottle is placed behind the right ventricle D, to mark the amount and degree of venous stagnation or regurgitation. f and g are the tricuspid and pulmonary valves respectively. B is the bladder, which when filled with water represents the pulmonary circulation. E is the left ventricle, and k and m the mitral and aortic valves respectively, while C is like D, in the last diagram, the systemic capillaries, and the source of the circulating fluid. To establish the syphonic circulation throughout the apparatus, A must at first be corked. When B is tense, water in A remains stagnant; when the tension in B is relieved by the action of E, water falls in A; the more perfectly E acts, the more quickly the level of the water in A subsides.

If the ball valve *g*, in the syringe D, representing the pulmonary valves, be removed, B can be emptied more quickly by the action of the perfect left ventricle E, than it can be filled by the maimed right ventricle D, owing to regurgitation into the latter on diastole or expansion, and the consequent withdrawal of part of the water thrown into B, and likewise, because this regurgitation prevents a certain amount of water from behind (the venous circulation) entering D. E has this advantage even when the syphonic action from C is in favour of D. The larger the quantity of water that enters E, and the more perfectly it contracts (that is the greater the capacity and power of the left ventricle), the more apparent is this advantage over D. If the ball valve *f* of the syringe D, representing the tricuspid valve, be removed, and E acts normally, B is emptied by E more quickly than it is filled by D, while under these circumstances regurgitation is very evident in

A; and if the syphonic current *h* be interrupted with this arrangement, while B is tense (the pulmonary capillaries engorged), water flows back into A more abundantly and rapidly than when the valve *g* (pulmonary) alone is deficient under similar circumstances. (A was purposely placed in these experiments at a higher level than B; as otherwise, when the syphon *h* is interrupted another is established from B to A.) By relieving the tension in B, water in A again falls, both when D and E act simultaneously, and when either of them acts alone.

If the valves *g* and *f* (pulmonary and tricuspid) are both removed from D, while E acts normally, B is emptied more effectually than under any other condition of D, while regurgitation in A is even more apparent than when *f* alone is absent. But, though the impulse of the regurgitation into A is more evident, there is no greater accumulation there than when the ball representing the tricuspid valve is deficient. If, on the other hand, the ball K in the syringe E, representing the mitral valve, be removed, while both valves are absent in D, B is filled more quickly per D than it can be emptied per E, owing to regurgitation from E into B (that is from the left auricle into the pulmonary veins and their radicles); but if M, representing the aortic valve in E, be removed while K is perfect, and both valves are absent in D, B can be emptied more quickly per E than it can be filled per D, but less quickly than when both valves in E (k and m) are perfect.

Both valves in D were removed in the latter experiments as representing most nearly the conditions in the case which actually came under my own notice, and the most crippled state of the right ventricle, and therefore affording the best test of the utility of the left.

Duration of the Disease.

A consideration of the length of time for which patients suffering from diseases of the class we are treating of survived, and the degree of embarrassment of the pulmonary and systemic circulation, will throw some light on the comparative efficiency of the compensatory measures adopted by nature in the different cases. We can only estimate the duration of the disease approximately, and in two cases (Ormerod's and Kinglake's) the correctness of even the approximate calculation is a matter of doubt; in the others, however, we are probably near enough the truth for practical purposes. Estimating the duration of the disease in the cases generally, and as accurately as circumstances will allow us, we may consider (as the tables will show) that in three it lasted between two and three years; in one, between three and four years; in one, between five and six years; in one, between six and seven years; in one, between ten and eleven years; in one (my case) between twelve and thirteen years at least, and possibly in a

minor degree from birth, *i.e.*, for twenty years ; in one (Begbie's) it was congenital; and in two, the duration of the disease is not stated, but in one of these (Paget's) it had probably existed for some years if we may judge by the character of the valvular disease, and in the other (Bertin's second case) it had probably a congenital origin.

The analytic table further indicates that where the duration of the disease was greatest the degree of embarrassment in the pulmonary and systemic circulation was least. The only exception to this rule is the case recorded by Dr Wilks, but death in this instance was attributed to disease of the lungs, and not of the heart, and the condition of the latter organ shows that life would probably have been prolonged for a considerably longer period but for the intercurrent affection. Moreover, it is to be noted that, with one exception, where the circulatory embarrassment was moderate, the left chambers of the heart escaped hypertrophy, and vice versa, where such a condition was great, those chambers had undergone accessory development, also with one exception. (I do not here take into consideration the cases marked with an interrogative sign.) The exception in the former instance is Dr Foster's case, and in the latter, Bertin's second case. In Bertin's case, however, the published account of the state of the circulation only applies to the short period during which he was under Bertin's observation, and we may conclude that as the disease was probably congenital, and the patient's occupation that of a stone-mason, his circulatory embarrassment must have been comparatively slight for a considerable period. I have already more particularly discussed at pages 112 and 113 in Journal, August 1879, the question of the comparative influence on the circulation of the different forms of valvular lesion, and need not again enter into this subject.

These facts corroborate the probable correctness of the opinion, that left-ventricular hypertrophy in most instances aggravates the difficulties of a circulation disordered by valvular disease in the right *side of the heart, and that this condition, if in the first instance a consequence of great impediment in the circulation, is in its turn a cause of still greater embarrassment, and not, as one might expect, a beneficial compensation for such obstruction.*

Before summarizing the positions laid down and the points maintained in the foregoing observations, now that I have discussed as fully as it is my intention to do the conditions and consequences of organic valvular disease in the right heart, I shall offer a few remarks upon the subject of functional valvular incompetency, especially of the tricuspid valve, and particularly on that form of incompetency, due to an obstructed pulmonary circulation, resulting from lung disease, and independent of obstructive or regurgitant lesions of the mitral or aortic valves.

Part III.

The competent closure of healthy valves is due to perfect systole of the chamber, at the orifices of which such valves are placed. Incompetent closure, or incompetency of the same, is due to an opposite condition—imperfect systole. The etiology of imperfect systole, therefore, must be the etiology of valvular incompetency. Perfect systole signifies a healthy state of the heart-muscle, and a normal capacity of the ventricular cavity. Perfect systole may likewise exist when the capacity of a ventricle is diminished, as such a condition in an originally normal heart signifies concentric hypertrophy. But, further, even if there be an increase of capacity, and the walls retain a normal vigour, from the universal hypertrophy, of all the constituent elements of the heart, systole may be sufficiently perfect to insure valvular competency. As I have already pointed out, however, the latter condition is transitory, and, as a rule, gives place to progressive dilatation of the ventricle, and the consequences of the latter—imperfect systole and coetaneous incompetency of the valvular guards at its orifices. We have, therefore, so far particularized our proposition, and may state that the etiology of ventricular dilatation is the etiology of so-called functional valvular incompetency.

Although it is my intention to limit my remarks to valvular deficiency in the right heart due to impediment in the circulation on the proximal side of the left heart, there are some general considerations pertaining to dilatation consequent on circulatory obstruction in the arterial heart, which will assist in the elucidation of the causes of dilatation of the right ventricle.

Taking Dr Aitkin's *Science and Practice of Medicine* (ed. 1872, p. 593 et seq.) as supplying a trustworthy exposition of prevalent views on the subject in question, we find he states in vol. ii., at p. 593, the cause of dilatation of the heart cavities to be "over-distension of the heart's fibres;" and again at p. 593, quoting the authority of Dr Fothergill, he says—"The greater the distension, the more frequent and greater are the efforts at contraction. Such increased and frequent contraction tends to increase the arterial distension and tension, and so far relieves systemic symptoms. But increased arterial recoil takes place, propelling more blood into the coronary arteries during diastole, which thus increases and improves the coronary circulation, greatly increasing thereby the nutrition of the heart, and thus promoting compensatory hypertrophy, enabling the muscular walls the better to resist the distending action of the blood (in the ventricle?), or overcome obstruction to its forward flow;" and again, he summarizes the causes of the permanent establishment of dilatation at p. 594 of the same work.

It is not, I need scarcely remark, my intention to dispute the fact, that cardiac dilatation is frequently associated with one or

other, or one or more, of the conditions enumerated, but to suggest, as a causal element in the production of dilatation, the active operation of an agency not sufficiently indicated by Dr Aitken, but discussed at considerable length in this thesis; namely, the active or passive, or both active and passive, turgescence of the coronary bloodvessels.

I admit that over-fulness of the ventricles tends to distend those cavities by stretching, and especially, from its greater thinness, the right ventricle; but although this may be the initial event where the circulation is obstructed either from the disease of the respiratory or circulatory apparatus, it is of short duration, and the dilatation which we detect at the bedside, either in acute or chronic disease, is, I would suggest, to be accounted for otherwise. When, from such circulatory impediment, we have repletion of the ventricles, the process differs according as the repletion is first experienced by the left or the right ventricle. When the former is the case, I would read the passage I have quoted from Dr Aitkin's work as follows:—"The more frequent and greater are the efforts at contraction, such increased and frequent contraction tends to increase the arterial distension and tension, and so far relieves systemic symptoms. But increased arterial recoil takes place, propelling more blood into the coronary arteries, *causing a more perfect diastole,* which together (the increased recoil and more perfect diastole) increase and improve the coronary circulation, relieve pulmonary stasis by means of the absorptive force of a more perfect diastole, and diminish in turn the repletion of the right ventricle, while the more efficient action of the latter supplies a *vis-à-fronte* for emptying the coronary veins in aid of the *vis-à-tergo* for the same purpose, supplied by the increased coronary arterial fulness. These circumstances combined greatly increase the nutrition of the heart, and promote an hypertrophy which enables the muscular walls the better to resist not only the distending action of the blood in the ventricles, but also the more powerfully distending force of an increased impulse in the coronary circulation. Moreover, if dilatation be superadded to hypertrophy, it must be regarded as a partial diastole becoming persistent under the influence of a permanently-increased coronary circulation; and further, such a condition of concomitant dilatation, so long as the walls of the ventricles remain healthy and their valves tolerably perfect, aids the emptying alike of the coronary, pulmonary, and systemic venous circulations by the absorptive force of a more powerful diastole, while the greater power of the systole of an hypertrophied heart assists in overcoming obstruction to the forward flow of the blood."

When the pulmonary circulation is obstructed on the proximal side of the left heart from any cause, such as bronchitis, the right ventricle is the first to feel the consequences of such an obstruction, in over-distension of its cavity, which, if continued for a suffi-

cient length of time, results in tricuspid incompetency, and still greater embarrassment of the systemic and pulmonary circulation. The mechanism of ventricular dilatation, when the initial event is repletion of the *right heart*, differs from the mode of its production in valvular disease of the left. I cannot better illustrate this than by criticising the statements of Dr G. W. Balfour on the subject in one of the most interesting chapters of his recent work on *Diseases of the Heart and Aorta*. As an example of curable tricuspid regurgitation, Dr Balfour mentions a case of acute bronchitis with symptoms of a greatly embarrassed venous circulation, in which not only was there tricuspid, but, in Dr Balfour's opinion, mitral regurgitation, both of which ultimately disappeared, and in their removal digitalis appeared to be of great service. This curable mitral regurgitation Dr Balfour considers as having resulted from "febrile muscular relaxation" (p. 185), and in a foot-note (pp. 182, 183) expresses surprise at Gerhardt's ignoring tricuspid regurgitation, and regarding the systolic mitral murmur under such circumstances as "a sign of local anæmia," "produced in some extraordinary manner by the defective blood-supply in the left side of the heart and arteries." I would interpret these physical signs thus:—An obstructed pulmonary circulation, the result of bronchitis, caused (α) distension of the chambers of the right heart by excessive accumulation of blood within them; (β) as in all cases of asphyxia, there was diminished transmission of blood to the left chambers; (γ) the distended right chambers caused stasis in the coronary venous system, both on account of the obstruction to the escape of blood into the right auricle, and because of the imperfect *vis-à-tergo*, resulting from the smaller quantity of blood thrown into the aorta by the left ventricle; (δ) the latter condition (stasis) produced engorgement of the cardiac walls, and counteracted systole to some extent, by favouring dilatation or diastole; (ϵ) imperfect systole rendered the perfect closure of the tricuspid valves difficult, and hence conduced to regurgitant incompetency; (η) a similar process of cardiac engorgement in a high degree had a similar influence on the closure of the mitral segments, regurgitation through which was in addition favoured by the scarcity of blood in the left ventricle already referred to, and very appropriately described by Gerhardt as a "local anæmia."—(*Lehrbuch der Auscultation und Percussion*, 1871, p. 264.)

The active influence for evil, of which an impeded current in the coronary veins is capable, is argued by the fact, that while the great systemic veins emptying themselves into the right auricle are unprovided with valves, the coronary veins *are* so protected at their very entrance into the main coronary sinus. When this barrier, however, is overleapt by excessive retrograde pressure, there are no further valvular impediments to encounter, and the obstructed current forthwith begins to exercise its pernicious influence.

Experiment No. 8.—If the process of rendering the coronary circulation turgid be reversed by distending the right chambers of the heart by way of the superior vena cava, after ligaturing the inferior cava and pulmonary artery it will be found much more difficult to effect that object than by injection per aortam; and even when the nozzle of the syringe is inserted into the coronary sinus, considerable resistance is offered to the water entering the coronary veins. Such an obstruction is, however, soon overcome under the influence of continued pressure, and the coronary veins filled with water.

I have recently had under my care a case which illustrates these remarks on the mechanism of tricuspid incompetency, and is one of a class which must be familiar to all. It was that of a bricklayer, Charles Coulson, 40 years of age, who had for some years suffered from chronic bronchitis and emphysema, at intervals aggravated by more acute symptoms. During his last illness his symptoms were more severe than on any previous occasion, and the right half of his body, towards which side he inclined, became highly anasarcous, while the left half remained for long emaciated, and free from dropsy. His urine, though frequently below the average normal quantity, was free from albumen. The physical signs of the lungs and heart were those of an advanced stage of bronchitis with emphysema. While such was his general condition, the point I wish to mention particularly I have noted in my case-book on the 25th of January 1878 as follows:—The stethoscope revealed a distinct systolic bruit, loudest immediately to the left of the sternum, between the fifth and sixth ribs, which was less distinctly heard in the epigastrium.

The jugulars were at the same time tense and the face suffused.

℞ Tinct. digitalis ʒi.
Sp. ammon. aromat, ʒij.
Sp. æth. chlor. ʒij.
Aq. camph. ad. ℥vi. Misce.

Sig. One tablespoonful to be taken every four hours; also, ℞ Sp. vini gall. ℥ss., occasionally.

Next day, the rhythm of his heart's action, which had hitherto been regular but quick, changed. The diastolic pause was distinctly prolonged, and the unmistakable bruit of the previous day had disappeared.

He continued to take digitalis, and on January 27th the bruit was still absent, and the rhythm of the heart as before. Again, on Jan. 31st, I find in my note-book: "Looks better, breathes more easily, bruit still absent. The pulse at the wrist is 72 per minute, and is regularly intermittent, and of equal strength. That is, there is an appreciable pause after every second beat, and the force and volume of the pulse are equal. Two beats and a pause at the

wrist correspond with three sounds at the heart; thus, lup, dŭp, dūp; the last "dup" is prolonged in proportion to the preceding "lup, dup." This I take to be reduplication of the first sound, and prolongation of the diastolic pause. "Omit digitalis." And on 1st. Feb., "Pulse and heart correspond; no prolongation of diastole; no bruit; otherwise in *statu quo*."

From this date his condition varied. One day the anasarca was greater, and the pulse weaker than on another. The pulse temporarily increased in force under the influence of a more frequent repetition of the digitalis, and the heart's sounds when examined were usually free from bruit. On one occasion a systolic bruit was thought to have been detected in the epigastrium, at the point of the ensiform cartilage. The absence of bruit, at this period of the case, was with greater probability attributable to pericardial effusion, with impotent systole, than to absence of tricuspid regurgitation.

The left half of the body also became anasarcous, but much less so than the right. Finally, on Feb. 14th, 1878, with a regular soft pulse of 120, he gradually lost consciousness, his pupils becoming insensible and his breathing slower, until he died at 7 P.M. the same evening. I examined the contents of the thorax, twenty-six hours after death.

Rigor mortis was not well marked. The body was anasarcous, and the right half much more so than the left half. Subcutaneous fat was deficient. Towards the posterior half of the body the subcutaneous textures were markedly œdematous. The cartilages of the ribs were not ossified. On raising the sternum, the lungs collapsed very slightly, and the edges of opposite lungs were in close apposition and adherent, completely covering the heart, except a small portion of it lying opposite the 4th and 5th intercostal spaces, on the left side, and extending as far to the right as the middle line of the sternum. The lungs were universally adherent to the costal pleura, and likewise to the pericardium covering the heart and large vessels. They were emphysematous in a high degree, and the pleural cavities contained little or no fluid, as all available space was occupied by distended and adherent lung.

The pericardium contained about half-a-pint of straw-coloured serum, but exhibited no evidences of recent inflammation. The visceral pericardium on the anterior surface of the heart was thickened and opaque over a considerable extent of surface.

The coronary veins were intensely engorged, full and prominent, especially on the posterior surface of the heart. The heart itself was much enlarged, and the right auricle was prominent, full, and distended to the size of a good plump kidney, and contained a huge black clot, as also did the right ventricle. The left ventricle contained a small dark clot, and the left auricle was empty.

The heart weighed 18 ounces.

The circumference of the right auricle was . . 20 ct.m.
The greatest height of the right auricle from the attachments of the tricuspid segment was . . 7 ct.m.
The thickness of the right auricular wall was . . 3-5 m.m.
The circumference of the tricuspid orifice was . . 14 ct.m.
The breadth and length of the anterior, internal, and posterior segments of the tricuspid valve were respectively, 6 ct.m. × 2½ ct.m; 4½ ct.m. × 2 ct.m.; and 3 ct.m. × 18 m.m.
The length of the right ventricle perpendicularly from the attachment of the tricuspid to the apex was . 8 ct.m.
And obliquely from the attachment of the pulmonary valve to the apex, ... 10½ ct.m.
The thickness of the muscular wall of the right ventricle, excluding the pericardial textures, was at the base, ... 10 m.m.
At the middle, ... 10 m.m.
At the apex, ... 5 m.m.
That of the anterior wall of the conus arteriosus was 11 m.m.
The circumference of the pulmonary orifice was . 9 ct.m.
And the breadth and length of the pulmonary segments, ... 3 ct.m. × 15 m.m.
The circumference of the left auricle was . . . 11 ct.m.
The thickness of the left auricular wall was . . 2-4 m.m.
The circumference of the mitral orifice was . . 11 ct.m.
The breadth and length of the aortic and ventricular segments of the mitral valve respectively were, 4 ct.m. × 3 ct m.; 7 ct.m. × 7-10 m.m.
The length of the left ventricle from the attachment of the mitral valve to the apex was . . . . 8 ct.m.
And from the aortic valve to the apex, . . . 8½ ct.m.
The circumference of the aortic orifice was . . 8 ct.m.
The breadth and length of the aortic semilunar segments were . . . . . 28 m.m. × 16 m.m.
The thickness of the muscular wall of the ventricle was at the base, ... 2 ct.m.
At the middle, ... 13 m.m.
At the apex, ... 8-10 m.m.

Coronary Arteries.

Circumference near origin, ... 11 m.m.
Circumference of coronary sinus in the right auricle near its origin, ... 25 m.m.
Circumference of a coronary vein near its entrance into sinus and for 4½ ct.ms. of its course, . . 5 m.m.

The endocardium and serous lining of the coronary arteries were spotted here and there with opacities, and the opening of

the coronary arteries into the sinuses of Valsalva were rather more gaping than usual, while the lining of the aorta at the mouths of these showed evidences of endarteritis.

On *microscopic examination*, many muscular fibres from both ventricles exhibited well-marked granular fatty degeneration, and there was an evident increase of the interstitial fibrous textures of the heart.

The diagnosis of dilated hypertrophy of the heart, with regurgitation through the tricuspid orifice, from pulmonary emphysema and bronchitis, was made during life. The former condition may be verified by comparing the measurements given, with the dimensions of an average normal heart, according to Bizot, at page 27. The situation of the systolic murmur, together with the systemic venous engorgement, appeared to justify a diagnosis of the latter, but there was room to doubt its correctness (1), on account of the disappearance of the bruit towards the close of life, and (2) because of the absence of a distinct regurgitant impulse in the distended cervical veins. The necropsy, however, explained these points satisfactorily. The very considerable serous effusion into the pericardium, together with the increasing feebleness of the cardiac systole, were sufficient to mask the former sign, while the enormously dilated right auricle was a physical obstacle to the propagation of the latter into the jugular veins.

An examination of the exact measurements of the heart, detailed in the account of the necropsy, shows, that while the pulmonary, mitral, and aortic orifices had an extent of valve exactly equivalent to the dimensions of the openings which it was necessary to close, the circumference of the tricuspid orifice exceeded its complement of valve by half a centimetre, and this deficiency is accounted for by a valveless interval measuring about five millimetres between the anterior and internal segments, thus (Figure 10):—A, is the anterior; I, the internal; and P, the posterior segment; while α is the valveless interval between A and I.

As the tricuspid and mitral are not so nearly circular as the pulmonary and aortic orifices, we cannot expect their diameters, as represented by the aggregate of the lengths of the different segments, to bear so mathematically exact a proportion to the circumferences of the former, as do the similarly estimated diameters of the pulmonary and aortic orifices to the latter. It is probable, nevertheless, that the length and breadth of the valves of the mitral orifice were sufficient during life to insure competency, and moreover the physical sounds of the heart gave no indication of mitral deficiency. In the absence of any signs of recent pericarditis, we must therefore assume that the systolic bruit heard during life, immediately to the left of the sternum and in the epigastrium, was significant of tricuspid regurgitation.

But again, the breadth and length of the tricuspid segments, from their hypertrophy, simultaneously with the rest of the heart,

were probably sufficient to close the right auriculo-ventricular opening (except the valveless interval of 5 m.m.), provided cardiac systole was sufficiently complete. The presence of so slight a valvular deficiency as that between the anterior and internal segments would scarcely of itself be sufficient to produce a regurgitation capable of originating a diagnostic bruit, as the chink must have been still narrower during systole; if indeed we can regard this deficiency as an abnormal condition, for some, and among them great names, consider that a salutary and natural regurgitation sometimes takes place through the tricuspid orifice.

Dr Robert Adams (*Dublin Hospital Reports,* vol. iv.), commenting on a case of contraction of the left auriculo-ventricular opening, refers at page 437 to a statement of John Hunter's, that the valves of the right side of the heart do not so completely close the arterial and auricular openings as those of the left side, and then says, "I look upon this difference in the valves of the right and left sides of the heart to be a natural provision to allow of a partial reflux into the right auricle on those occasions when, from any cause, the passage of the blood through the arterial opening is retarded. Such a provision was absolutely necessary in the right or pulmonary ventricle, as various causes must momentarily retard the passage of blood through the lungs."

Mr Thomas Wilkinson King likewise supports this theory, in the *Guy's Hospital Reports,* Nos. IV. and XII.

Although few will now be found who credit these views, especially as regards a normal insufficiency at the pulmonary orifice, it is not improbable, that in a case such as the present some tricuspid regurgitation may take place and be natural, and presumably therefore salutary, in an over-distended state of the ventricle. The valveless space at the junction of the anterior and internal segments had doubtless been stretched to the extent of 5 m.m. by, and simultaneously with, the universal dilatation of the auriculo-ventricular orifice and the neighbouring chambers; but there must originally have been a break in the continuity of these segments at this point. We can therefore easily understand that in a similar case (and in all instances if this is a normal and constant condition) a temporary dilatation of the right heart, however caused, may in like manner produce a temporary gap at this point sufficient to permit a transitory regurgitation through this orifice, and, of course, that a permanent dilatation of these chambers would result in the establishment of a permanent gap and a persistent regurgitation.

Now as to the cause of the tricuspid regurgitant bruit clinically detected in this case, was it due to the over-distension by blood within it of a chamber weak in muscular fibre, or to engorgement of the coronary venous system, or to both? And what assistance in the elucidation of this point is given us by the action of digitalis in abolishing the bruit?

The greatly increased diameter of the muscular walls of the right ventricle would have led one to suppose that that chamber was supernaturally strong, had the textures not been submitted to a microscopic examination, which proved the condition to be really one of pseudo-hypertrophy, as a great part of the increase in the thickness of the walls was due to an excessive production of fibrous connective tissue. The latter, as the result of long-standing "congestion of the heart," was in itself a proof of coronary engorgement. Moreover, the fact of the right auricle, coronary veins, and right ventricle being intensely distended with blood at the necropsy, and likewise the fact of the right auricle and coronary sinus being in a state of persistent dilatation, argued that an over-accumulation of blood in the right heart and coronary veins was probably a more or less persistent condition. We must therefore infer that both the degenerate condition of the muscle of the ventricle, and the engorgement of the coronary veins, probably contributed to the over-dilatation of the right auriculo-ventricular orifice at the time when the tricuspid bruit was noted. That, however, the latter condition was principally operative in the establishment of permanent dilatation, is highly probable; for not only would the impeded coronary circulation exercise, as has been shown, a diastolic influence, but, by the production of an excess of intermuscular connective tissue, it would indirectly promote the ultimate degeneration of the muscular fibres, the systolic influence of which would, in their degenerate state, the more easily succumb to the dilating influence of the blood within the ventricle, and to that of the engorged coronaries. The presence of an excessive and active coronary engorgement at one period of the case, probably before the hypertrophied left heart had degenerated, may be inferred from the dilated and atheromatous mouths of the coronary arteries, and the patchy opacity of their serous coats.

The disappearance of the bruit under the influence of digitalis, and the simultaneous but transient relief afforded to the patient, further prove the dependence of the bruit upon imperfect systole, from the causes indicated. Digitalis, by squeezing the heart muscle, perfected the tricuspid valvular action, abolishing bruit, tended to assist the evacuation of the coronary veins, and to empty both ventricles and the coronary arteries, thus affording relief to the lungs, and promoting for the time an improvement in the action of other organs. If our physiological argument is correct, such a beneficial prolongation of the diastolic pause must signify an increase in the absorptive power of ventricular diastole, the existence of which we endeavoured to establish.

I shall now conclude this portion of the subject by briefly recapitulating the physiological and pathological positions which it has been my endeavour to maintain in the preceding pages. The physiological propositions were as follows:—

A (1.) That i is more difficult to propel a horizontal than a perpendicular circulation.

(2.) That the horizontal circulation of the sheep may be taken as a type of a difficult or impeded circulation in an upright animal.

(3.) That the provision made by nature for the propulsion of such a circulation is, THEORETICALLY, the true type of the most perfect compensation in a circulation obstructed by disease of the right heart in man; but that there are circumstances which preclude a beneficial result from the adoption in disease of the plan of this natural provision in its entirety.

(4.) That turgesence of the coronary circulation has an influence in the production of diastole, as demonstrated by Garrod's experiment.

(5.) That diastole is thus in part an active event.

(6.) That active diastole has an absorptive influence towards the diastolic chamber.

(7.) That diminution of the capacity of a chamber diminishes its absorptive power.

(8.) That cardiac systole and diastole in the manner explained in the context are mainly concerned in the propulsion of the coronary circulation, while the latter at the same time contributes, as already stated, a force in aid of the production of cardiac diastole.

The pathological propositions were:—

B (1.) That there are two classes of cases of valvular disease of the right heart, distinguished according to the order in which the pathological changes in the circulatory apparatus follow one another; that in the one, obstruction to the pulmonary circulation and hypertrophy of the right ventricle is the final, and in the other the initial event.

(2.) That the more perfect compensation is in the right chambers from accessory development, the less do the left chambers deviate from their normal condition.

(3.) That diminution of the capacity of the left ventricle by hypertrophy, in cases of dextral valvular disease, increases the embarrassment of the pulmonary circulation primarily, and of the systemic venous circulation secondarily.

(4.) That ventricular dilatation with imperfect systole is the cause of valvular insufficiency.

(5.) That active or passive engorgement of the coronary circulation is a force of the first importance in the production of permanent dilatation of a ventricle, which is in fact a condition akin to *persistent partial diastole.*

PART IV.—TREATMENT.

THE points which I purpose considering under this heading, with as much brevity as possible, are:—

(1.) The position most appropriate for a patient suffering from "dextral" valvular disease.

(2.) The temperature and atmosphere in which such a patient should, if possible, be placed.

(3.) The most suitable diet, including the question of stimulants.

(4.) The desirability of venesection in some cases.

(5.) External remedial or alleviative applications.

(6.) The administration and object of internal remedies, with special reference to the indications and contra-indications for the employment of ammonia and digitalis.

α. The position in which the patient is most comfortable is probably, in all cases, an appropriate adaptation of the economy to the exigencies of each individual case. A more or less upright position of the upper half of the body (orthopnœa) may, however, be regarded as that which is most favourable to the transmission of blood from the systemic venous system through the right heart to the lungs; but this may vary in degree, according to the seat of the valvular lesion (whether tricuspid, or pulmonary, or both), and the extent to which the valves are rendered incompetent, and also, according as we have to do with incompetency from organic lesion, or functional insufficiency from lung disease. In Sir James Paget's case of pulmonary regurgitation, the recumbent position was easily maintained, as there was evidently no considerable regurgitation into the large veins; but in most instances, and especially where an impeded pulmonary circulation is the *origo mali*, or where there is any serious imperfection of the tricuspid valve alone, or in conjunction with pulmonary insufficiency, some degree of orthopnœa is a result of the condition. This fact merely signifies that in this position the great suction force of respiration can be most effectively employed from the possibility of fixing the respiratory muscles so as to act more energetically, and from avoiding that compression of the thoracic walls by the weight of the body against resistant surfaces, such as the bed, which is calculated to impede the full expansion of the chest. We may likewise see in the emphysematous condition of the upper lobes of the lungs, in some cases of dextral valvular disease, an expression of exaggerated effort on the part of those organs, and therefore conclude that we should place the body in that position in which they may best continue their excessive labour with the least exertion. It is, however, possible that this emphysema may also to some extent be a consequence of the usually congested and more or less inefficient condition of the lower lobes.

β. The indications to be fulfilled in respect to temperature and air may be shortly stated to be the maintenance of such a degree of warmth as is necessary to secure at least a normal cutaneous circulation, and thus to prevent excessive blood pressure in the deeper vessels and heart. The great atmospheric necessity is, of course, a due oxygenation of the blood in the impeded pulmonary circuit—a condition best fulfilled by as free ventilation as is compatible with the maintenance of moderate warmth

γ. A light, nutritious diet, given in small quantity, to facilitate digestion by an alimentary canal, whose functions are always more or less impaired by a chronic passive engorgement, will best suit most of such cases. As in most instances of any species of diseased heart, a restriction of fluid food is advisable, lest the bulk of the blood circulating in the vessels be too suddenly increased thereby.[1] In disease of the right side of the heart it is especially necessary that too large a quantity of fibrinogenous articles of diet should be avoided, to diminish as far as in us lies the possibility of coagulation in the right chambers of the heart, or in the systemic venous or pulmonary arterial system. Alcohol, in one form or another, but especially brandy, from its twofold property of invigorating the action of the heart, and maintaining a more or less dilated condition of the capillaries, is indispensable in the treatment of "dextral" valvular incompetency, from whatever cause arising. We must, however, be on our guard against any excess in the administration of so highly carbonaceous a substance as alcohol to a patient whose blood is already surcharged with carbonic acid from imperfect aeration.

δ. Judicious venesection, now happily resuscitated from long and culpable neglect, may prove of the greatest importance in valvular disease of the right heart. Mr Kinglake's withdrawal of 1248 ounces of blood from the system of his patient by 4-ounce venesection, on 312 separate occasions, during the space of two years, is perhaps as good an example of the bloodthirsty therapeutics of our ancestors as any on record; yet, if we accept his authority, the patient seems to have been greatly benefited thereby. A less frequent and less copious relief of tension to an intensely engorged venous circulation would probably render useful service still.

ε. By external remedial or alleviative applications, I mean poulticing, counter-irritation, or derivative treatment generally, for the intercurrent congestion of various organs (lungs, kidneys, etc.), of such frequent occurrence during the course of the cases in question. The external application of extract of belladonna appeared to relieve the præcordial anguish of the patient under my care.

ζ. For the rest, normal secretion and exertion must be favoured as far as possible, and, if arrested, an endeavour made by recognised means for their re-establishment. It must always be borne in mind that the most delicate index to the cardiac condition is the state of the urinary secretion; in combating an embarrassment of the latter, therefore, we must endeavour to regulate the former.

[1] Care must be taken, however, not to render the blood too viscid by too great a restriction of WATER. In all cases of engorged right heart, whether from pulmonary or cardiac disease, water appears to have a beneficial action by promoting fluidity of the blood, and the consequences of this—easy transmission through the vascular system, and improved excretion, especially by the kidneys.

This, however, is sometimes beyond our power in the first instance, and can only be effected after the cardiac repletion has been relieved by purgative evacuants and lumbar counter-irritation, to remove renal congestion, or, if necessary, by venesection. Dr B. W. Richardson's well-known investigations on the cause of the coagulation of blood, though not universally endorsed, are of great practical importance, if it be true that the fluidity of the blood is favoured by the volatile alkali ammonia. Dr Richardson, however, pointed out in a paper "On the Antiseptic Properties of Ammonia," in the *British Medical Journal* for 3d May 1863 (Ranking & Radcliffe's *Half-Yearly Abstract of the Medical Sciences,* vol. xxxvii. p. 337), "that this alkali had in addition the power of arresting the oxidation of tissues, and even prevented the action of ozone." "Hence (*loc. cit.*) long exposure to ammoniacal vapour, by arresting oxidation, produced extreme anæmia and a low depraved condition of the system altogether, with reduced respiration, reduced appetite, reduced muscular power, and reduced energy."

In disease of the right heart, unfortunately, whether organic or functional, we have to do both with a condition of imperfect oxygenation of the blood and with conditions favourable to its coagulation. Of the two evils, therefore, we must choose the least, according as the patient appears at different times to suffer most from pulmonary dyspnœa or cardiac venous engorgement. When the former is the case, ammonia may be given sparingly or withheld, and when the latter, it may be administered more liberally.

The undoubted efficacy of chlorate of potash in maintaining intrauterine life in some instances, and its chemical property of disengaging oxygen, render it rational to suppose that its combination with ammonia may restrain to some extent the influence of the latter in preventing oxidation, while its influence in maintaining the fluidity of the blood would not be interfered with.

In short, the prevention of spontaneous coagulation of the blood, and its due arterialization, are the great desiderata, and whatever is now known to promote these conditions, or whatever may hereafter be found to have such an influence, will be found useful in the class of cases which we are discussing.

The question of the employment of DIGITALIS in valvular disease of the right heart demands fuller consideration than the preceding well-known and almost generally observed rules of cardiac therapeutics. Drs C. Hilton Fagge and Thomas Stevenson, in a paper entitled "On the Application of Physiological Tests for Certain Organic Poisons, and especially Digitaline," published in the *Proceedings of the Royal Society of London*, vol. xiv. p. 272, state that, after poisoning with digitaline, helleborus, and scilla, the ventricle remains "rigidly contracted and perfectly pale after it has ceased to beat," and the fact of death "in systole," after doses of digitalis, has been corroborated by many other competent observers. Dr George W. Balfour, in the work already quoted, says, at p. 308,

"Digitalis, while it slows the heart's action, at the same time increases the force of the ventricular systole, this dilatation lessening *pari passu* with the increased action of the drug, therefore, as a rule, with an increase of the dose. In aconite poisoning, or in threatened asystole, the aortic pressure falls because the over-distended ventricle is unable to contract upon its contents, each contraction only succeeding in expelling a small quantity of blood off the top of the distended ventricle; in digitalis poisoning, on the other hand, the aortic pressure falls because the over-contracted ventricle permits but little blood to get into it, and can consequently send but little forward."

While there is thus unanimity as regards the physiological action of digitalis on the heart, there is a most confusing variance of opinion among recognised authorities as to the employment of digitalis in disease even of the left heart, while there seems to be no scientific basis whatever for its administration in organic disease of the right heart. For example, from the passage last quoted from his work, at p. 308, Dr Balfour derives his warrant for the use of digitalis in aortic incompetency: "A disease," he says, "in which we often obtain the most brilliant therapeutic results from the use of this drug." The prolonged diastole he considers compensated by the diminished ventricular capacity.

Dr Balthazar Foster, on the other hand, in his *Clinical Medicine*, p. 97, states that "The diminution of the frequency of the heart's beats under digitalis means an increase of the period of the dilatation of the ventricles. Pulse traces readily show this. It is during this very period that aortic insufficiency produces its ill effects, and it is consequently not hard to understand why digitalis acts injuriously. . . . There is one condition that warrants its use—that of over compensation."

It is not my intention to criticise these discordant views, though my own experience, such as it is, agrees with that of Dr Balfour, but merely point them out to show that if such opposite opinions can be maintained with regard to so comparatively common a lesion as aortic regurgitation, it is little wonder that knowledge of the effect of digitalis in the much rarer instances of organic valvular disease of the right heart can scarcely be said to exist.

Dr J. Milner Fothergill (*Digitalis, its Mode of Action, etc.*) says, "In valvular disease of the right side we do not know any peculiarities connected with the pulmonary orifice; disease here is rare, and the same indications and contra indications would exist as in aortic disease."

In tricuspid regurgitation the same author states, "The administration of digitalis is apparently of no benefit whatever; the increased action of the left ventricle is too far distant to be of any avail. The action seems lost over the length of the systemic circulation, and in the dilatability of the veins." And again (*op. cit.*), "In tricuspid obstruction, digitalis and all other cardiac neurotics would be use-

less for the same reason." I do not dispute with Dr Fothergill the fact of the inutility of digitalis under such circumstances, but, as I shall show later, I cannot altogether agree with his theory of this inutility.

Dr Balthazar Foster, in his *Clinical Essays*, at p. 103, also says, "Two cases in which digitalis failed me unexpectedly were examples of mitral stenosis, associated with a similar lesion in the right side of the heart." As many cases of mitral stenosis might be cited, in which the administration of digitalis was beneficial, we may conclude that the disturbing element in these instances was the dextral disease.

As this question of the advisability or unadvisability of the employment of digitalis in the treatment of organic valvular disease of the right heart is of the greatest practical importance when such, happily rare, cases are met with, I shall now add the following particulars of its use and apparent effects in the case of E. W. G. M., and if we compare the periods during which the drug was administered with corresponding dates in the clinical progress of the case, the inference is highly significant of its evil effects. From the absolute want, so far as I know, of published clinical experience on the part of others in this respect, I had no sources of information whence I could have learned, not only the inutility, but, as I now think, the positive danger of giving digitalis under these circumstances. It must be remembered that I was then under the impression that the case was one of double pulmonary lesion, associated with mitral regurgitation, and therefore argued that as the latter was almost invariably benefited by the action of digitalis, and as, like Dr Fothergill, I regarded the pulmonary lesion as analogous to double aortic disease, which I had likewise known to be benefited by that drug, I concluded that the union of two disorders benefited by one and the same agent would result in benefit to both on employing the said agent; and so convinced was I of the correctness of this argument, that I attributed the subsequent phenomena of retrograde congestion to increasing dilatation of the right heart, and as a further indication, rather than otherwise, for the employment of still more of that opponent of dilatation—digitalis, though my occasional abandonment of the drug sufficiently shows that I entertained doubts of its beneficial action. I accordingly prescribed five minims of the tincture every four hours from the 20th of May till the 2d of the following June, when, as will be seen in the context, ecchymotic purpura appeared, and the digitalis was stopped. This phenomenon soon disappeared, and on June the 19th, thinking that five minims had possibly been too small a dose, I increased it to seven and a half minims every three hours, and continued it till the 24th of June. After resuming it on this occasion, the purpura at first continued to fade, and I was inclined to regard this as evidence of a beneficial influence exercised by the drug, but it again returned, and I reduced the dose to

five minims every three hours, and ultimately combined it with ten minims of liquor secale cornuta on the appearance of the hæmoptysis referred to in the clinical history of the case.

Subsequent reflection upon the entire course of the case leaves little doubt on my mind, and will, I think, convey the same impression to the mind of any one who peruses this paper, that the retrograde phenomena which I attributed to the progressive overthrow by disease of the barriers to such engorgement, in a case so fatally maimed in the venous heart, were in reality precipitated by the mistaken employment of a drug most potent for good in many forms of heart disease, but likewise, like all such, most potent for evil, when from insufficient knowledge its energies are misdirected. Let us now try to discover the most feasible explanation of the pernicious influence exerted by digitalis on a circulation embarrassed in consequence of organic valvular disease in the right heart. As my personal experience is limited to the case of E. W. G. M., my argument will, of course, be founded chiefly upon it; but, as both Drs Fothergill and Foster speak of the inutility of the drug in the case of dextral complications, it is not unreasonable to suppose that my remarks may have a more general significance, notwithstanding the limited data from which I argue.

Were the effects of the drug due to its action upon the right or the left ventricle, or both?

There is little doubt that the supra-sternal systolic pulsation, noted in the clinical history of my case, was due to powerful regurgitation into the innominate veins by way of the superior vena cava, for it is scarcely possible that even a *dilated* pulmonary artery could have exhibited visible pulsation in this situation.

This pulsation, it will be remembered, was greatly subdued after digitalis had been employed for some days, although the distress of the patient was not proportionately relieved. The subdual of this regurgitant pulsation, it is rational to suppose, was the result of diminished regurgitation, for the effect of digitalis is not to lessen the force of cardiac systole, and this is the only alternative by which the phenomenon could have been explained. We know that in the later stages of many cases of tricuspid regurgitation individual pulsations are lost in a general turgescence of the cervical veins. Had this been the case in the present instance, the site of the formerly pulsating vessel would probably have been occupied by a distended trunk, and accompanied by the turgid venous branches and suffused face characteristic of such a condition. This, however, was not evident. The effect of digitalis, then, in so far as it diminished regurgitation from the right ventricle (if I am correct in supposing it did so), cannot be regarded as having been DIRECTLY conducive of the phenomena of serious retrograde congestion which supervened, and therefore we may be allowed to doubt whether digitalis, in so far as its effect upon the condition of the right ventricle was concerned, had any important share in the production of the

untoward circumstances which followed its administration. Dr Fothergill considers the inutility of digitalis, under the circumstances we are discussing, to be due, as has already been stated, to the increased action of the left, being at too great a distance from the surcharged right ventricle. The left ventricle, however, we must remember, while the rapidity of its pulsations may be increased by digitalis, has a diminished capacity from the same cause, and the extreme effects of such a condition cannot better be demonstrated than by the following experiment in Dr Fothergill's work already quoted. At p. 3, after stating that half a dram of digitalis was given to two sparrows, he says, "On opening them immediately after death the left ventricle in each was found firmly contracted, the lungs so congested as almost to be hepatized, the right ventricle full of blood. It was evident that the condition of the lungs and right ventricle was due to inability to drive the blood into the contracted left ventricle." A minor degree of left ventricular contraction would doubtless have shown the same effects to a less extent. It is true that the "inability to drive blood into the contracted left ventricle" is a feasible explanation of the co-existent retrograde engorgement, but on this hypothesis the influence of the left ventricle is purely *passive*. Can it, on any supposition, exercise an *active* influence? I have endeavoured to prove by experiment, in the physiological argument, that the left ventricle in particular exercises a considerable absorptive power by way of the pulmonary veins, and have likewise tried to show, both by experiment upon the sheep's heart, and by placing a ligature round the bag representing the left ventricle in the indiarubber schema of the circulation, that the absorptive power of the left ventricle is in direct proportion to its capacity, *i.e.*, a diminution of its capacity and absorptive power, and *vice versa*. A diminution of its capacity and absorptive power, moreover, was found to favour retrograde engorgement, the removal of which was effected by increasing the capacity, and with it the absorptive power. The effect of digitalis upon the heart is analogous to the effect of the ligature upon the caoutchouc bag. The capacity of the former is thereby diminished, and with it, we may conclude, its absorptive power. By having a diminished capacity the left ventricle may be considered conducive of engorgement passively; by a necessarily simultaneous diminution of its absorptive power, it must therefore exercise a similar influence ACTIVELY.

But, again, in the pathological argument it was pointed out that the circulation was most embarrassed when hypertrophy of the left ventricle co-existed with dextral valvular lesion. Hypertrophy of the left ventricle, if unassociated with mitral insufficiency, is probably in most of these cases unassociated with any considerable degree of ventricular dilatation, and may, under such circumstances, be associated with some diminution of ventricular capacity. Such a condition is more likely to accompany

left ventricular hypertrophy in cases of dextral valvular disease, than in those cases in which disease of the valves of the arterial heart constitutes the chief lesion; for, in the former, there is not only the long tract of systemic venous and arterial vessels intervening between the hypertrophied left ventricle and the site of the capital defect, and which, doubtless, offer a considerable resistance to the retrograde pressure from the venous, and thus share the work of the arterial heart, but the defective pressure in the pulmonary circuit, from the dextral valvular incompetency, causes less blood to enter the left ventricle. This chamber, then, under these circumstances, is protected in a twofold manner from a dilating influence, which is unfettered, and in fact exaggerated, in cases of sinistral disease.

It is rational, therefore, to suppose that hypertrophy of the left ventricle, arising from disease of the pulmonary and tricuspid valves, must in most instances, and for a considerable period, be unassociated with dilatation, and probably coupled with diminution of the capacity of that chamber. The result of this, as we have seen, would be to aggravate the distress of the patient, and if our argument be valid, Dr Fothergill's dictum, that accessory growth is our warrant for the employment of digitalis, would be an instance of "imperfect induction," for we have here an exception to the rule, as the systolic influence of digitalis, by still further reducing the capacity of the ventricle, would merely facilitate retrograde engorgement.

Finally, when we consider that, in consequence of dextral valvular deficiency, one of the forces for propelling blood through the pulmonary circuit is seriously impaired (and results, as we have endeavoured to show at page 24, in one form of pulmonary congestion), it would evidently be mischievous to diminish the size of an undilated left ventricle by digitalis, the effect of which would be, as is shown by Dr Fothergill's experiment on the sparrow, and by the statements of others, to render it more difficult for blood to pass through the lungs from the venous to the arterial heart. Whether digitalis would be of service where the left ventricle is dilated in connection with a dextral lesion, it is difficult from lack of experience to say, but the failures I have referred to when it has been used with a sinistral, coupled with a dextral valvular defect, permit us to doubt whether, under any circumstances, this drug can be employed with benefit when we have an organically disabled venous heart.

A left ventricle, then, of normal capacity appears to be the most desirable associate of a right ventricle maimed as to its valves; because in most cases it signifies that the compensatory changes in the venous circulation have been adequate to the necessities of the economy, while at the same time its own peculiar functions have in no way been deranged by any deviation from its normal capacity and power. How delicately the balance is adjusted may be

gathered from the unmistakable manner in which it was upset by the comparatively small and carefully administered doses of digitalis employed in the case of E. W. G. M.

From the foregoing argument it is now permissible to infer that the systolic influence of digitalis upon the left ventricle is, if not the only, probably the chief element of danger in the exhibition of that drug in cases of organic valvular disease of the right heart; and additional probability is lent to this view by the fact that pulmonary hæmoptysis was noted for the first time, during the course of the case reported by me, while the patient was under the influence of digitalis.

Functional valvular incompetency of the tricuspid valve is the result, as we have seen, of over-dilatation; and digitalis (the *rationale* of the action of which, under such circumstances, has been already indicated), in so far as it can correct such a condition and restore the competency of otherwise healthy valves, is a remedy of great value, and, carefully administered, may prolong life for a time after what may be considered (especially in chronic cases) the knell of dissolution has been sounded by the appearance of tricuspid regurgitation.

The therapeutic conclusions, then, at which we have arrived are briefly as follows:—

(1.) That a more or less orthopnoeic position is best suited to many cases of organic valvular disease of the dextral valves; but that there are exceptions to this rule, and that the latter will probably most frequently be constituted by cases of pulmonary valvular lesion, and especially by cases of pure pulmonary regurgitation, just as we find a recumbent position best adapted to the analogous instances of aortic regurgitation.

(2.) That the cutaneous circulation must be maintained by adequate warmth, and arterialization of the blood by as free a ventilation as is compatible with the maintenance of sufficient heat.

(3.) That food must be given to patients from dextral disease under the same restrictions as in the case of other cardiac sufferers, but that fibrinogenous material must be even less consumed than in other cases, from the greater liability to spontaneous coagulation, and that alcohol, in one form or another, is absolutely necessary, but must be cautiously administered.

(4.) That venesection is calculated to render signally good service in the retrograde plethora due to dextral valve lesions.

(5.) That external applications must be employed in such cases, under the same circumstances as in other forms of cardiac disease.

(6.) That the use of ammonia, from properties peculiar to it, is indicated, but that asphyxial conditions may render its combination with chlorate of potash or some other oxygenator advisable.

(7.) That the employment of digitalis is not only useless in cases of organic disease of the dextral valves, but fraught with a

danger which cannot be exaggerated, and that the chief cause of its pernicious influence is probably its systolic action upon the left ventricle.

(8.) That in functional valvular disease of the right heart, arising from ventricular dilatation, and especially in functional tricuspid regurgitation, digitalis carefully administered may prove very beneficial by diminishing the capacity of the ventricle, and restoring or improving the competency of valvular action.

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