semblance to another which I described and figured in the *Transactions of the Pathological Society*, vol. xxxix, 1888, p. 200. In that instance inflammatory changes in the appendages had set in seven years previously.

It is evident that an operation was even more urgent in this case than in the first. I have seen neglected cases where the papillomatous masses have passed beyond the limits of the cyst and invaded the pelvic viscera, so as to be irremovable. These cases ended fatally, whilst by timely interference in the case here described the patient was restored to perfect health.

ON THE ACTION OF DIGITALIS IN CASES OF AORTIC REGURGITATION.

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FROM Corrigan down to the present day many practitioners and authors have objected to the use of digitalis in cases of patency of the aortic valve from a theoretical idea that this drug, by prolonging the diastolic pause, favours the regurgitation of the blood into the ventricle, and so tends to fatally embarrass the heart's action. It is, however, nearly fifty years (1844) since the late Professor Easton, of Glasgow, pointed out that when the heart beats less frequently there is less opportunity for regurgitation, and not greater.¹ This is obvious enough: when a heart beats forty times a minute there are only forty regurgitations per minute; when the heart beats ninety times a minute there must be of course also ninety regurgitations. But it is argued that though the regurgitations are fewer, they are more prolonged, therefore greater in amount and more dangerous to the maintenance of the integrity of the circulation. But a prolonged diastole does not necessarily signify an increased reflux. Within the vascular system there is neither air nor a vacuum, but one unbroken column of blood extends from any given point within it back to itself again. The heart is the only part of this system endowed with the power of active movement, and upon it the circulation of the blood depends. When the left ventricle of the heart contracts it propels its contents into the aorta, that vessel—already full of blood—by virtue of its elasticity expands and receives this further supply thrown into it. When the systole of the ventricle is completed and its diastole commencing there is developed "a negative pressure in the rear of the column of blood driven from the ventricle into the aorta with great suddenness and rapidity."² This negative pressure follows the blood past the semilunar valves, and in the normal state it in part at least determines the closure of these valves. This negative pressure, which is greatest within the ventricle, is largely due to the return of the ventricle from its contracted to its normal form. At the end of the ventricular systole the segments of the mitral valve form a depending cone with its apex directed to the apex of the ventricle. This cone is full of blood in free communication with the auricle now fully distended,³ and at the moment of diastole the sucking influence of the negative pressure within the ventricle opens up its apex, the blood closely follows the expanding wall of the ventricle, and gently floats up and into apposition the segments of the mitral valve. At the moment of the ventricular diastole the systole of the aorta takes place, this vessel returns to its normal dimensions. In the normal man the systolic compression of the blood within the aorta helps to close the semilunar valves, but the whole amount received from the heart is propelled onwards. When the aortic valve is incompetent, the systole of the aorta, coupled with the negative pressure within the ventricle, determines a certain amount of regurgitation which can never be equal to the amount propelled, otherwise the circulation would at once come to a standstill. This reflux has been calculated as amounting to one-third of the whole ventricular contents,⁴ and is probably usually considerably

less than this. Under the influence of the aortic systole and of the intraventricular negative pressure the blood from the aorta and from the auricle closely follows the wall of the ventricle during its expansion, fills up its cavity, and floats up and into apposition the segments of the mitral valve. So soon as the ventricle has returned to what Foster calls its normal condition its active expansion ceases, and a period of rest or quiescence sets in, during which the blood within its cavity, in the aorta, and in the auricle comes under the influence of hydrostatic laws only.

The most important of these laws in regard to aortic regurgitation, is that to which the name of its earliest enunciator, Pascal, has been given. "Pressure exerted anywhere upon a mass of liquid is transmitted undiminished in all directions, and acts with the same force on all equal surfaces, and in a direction at right angles to those surfaces." This law has a probable influence in promoting the gradual dilatation of the left ventricle when the aortic valve is incompetent, but it acts very slowly, as one of the corollaries of Pascal's law is that what is gained in power is lost in velocity. There is also another and from the present point of view a more important corollary to Pascal's law, and that is that the pressure transmitted by a liquid is proportional to the extent of its surface. Now the area of the mitral opening may be taken at 1.25 square inches, and the area of the abnormal aortic orifice at 0.25 of a square inch, never more, often very much less. The ratio of the respective surfaces of these openings is thus as 5:1.⁵ From the mere surface point of view, therefore, the blood column in the auricle is four equivalents in excess of what is necessary to counterbalance the blood column in the aorta, and it acts perfectly favourably because there is a third corollary from Pascal's law which states that the upward pressure of liquids at any point is governed by the same laws as the downward pressure. We may, however, suppose the pressure of the aortic blood column to be equivalent to the blood pressure on the sound aortic valve which the ventricle has to overcome before the segments of that valve are thrown open and the blood projected into the aorta, equal therefore to a pressure of 250 m.m. Hg., or 3 metres of blood. But then we must also remember that from the absence of capillary resistance and from the non-existence of vasomotor nerves within the lungs, the mechanical effect of the systole of the right ventricle makes itself felt from pulmonary artery to left auricle, and this has been calculated at one-fourth of the force of the left ventricle, 60 m.m. Hg., or $\frac{1}{4}$ of a metre of blood. These figures multiplied by five to bring them to the proper ratio give more than enough of upward pressure—300 m.m. Hg., or $\frac{3}{4}$ metres of blood—to prevent any regurgitation from the aortic orifice. But if we consider that the filling up of the ventricle detracts somewhat from the intrapulmonary pressure, and suppose that the blood passes through the mitral orifice at the pressure of only 6 inches of blood, as Davies has done,⁶ then we must also allow for the reduction of blood pressure by regurgitation from the aortic column, and this coupled with the patency of the valves, would seem to reduce the pressure in that column to that of about 18 inches of blood or the height of a column of blood reaching from the base of the heart to the top of the head. But the pressure at the mitral orifice—6 inches—multiplied by five, its proportional area, gives a relative pressure of 30 inches at the mitral orifice, much more than sufficient to prevent all regurgitation through the aortic orifice during the quiescent period of the diastole.

A slow pulse, therefore, not only gives fewer opportunities for regurgitation, but though it is indubitably accompanied by a prolonged diastole the regurgitation is not thereby increased, the reflux is actually diminished by a slowing of the heart's action, and the balance of the circulation made more equitable. The action of digitalis therefore in slowing the heart is not hurtful in aortic regurgitation but beneficial. But apart from its action in slowing the heart—which is a mere accidental or secondary effect—digitalis possesses pre-eminently the properties belonging to what is called the digitalis group of remedies. It increases the elasticity of the muscular tissue so that this extends and contracts more completely and perfectly, and as all the blood passes more frequently through the heart than through any other muscle,

¹ *Monthly Medical Journal*, July, 1844, p. 634. Easton goes fully into the question, and his paper is well worth studying.

² Foster's *Physiology*, 6th ed., p. 248.

³ Pettigrew, On the Relations, Structure, and Functions of the Valves in the Vascular System of the Vertebrata, *Transactions of the Royal Society of Edinburgh*, vol. xxiii, p. 799.

⁴ Davies, *The Mechanism of the Circulation through Diseased Hearts*, London, 1889, p. 51.

⁵ Davies, *op. cit.*, p. 48.

⁶ *Loc. cit.* The calculations in this book are by Dr. Haughton, of Trinity College, Dublin.

this action is specially exerted upon it, and is manifested at a time when the other muscles are practically uninfluenced.⁷ The importance of such an action on a failing heart cannot be overestimated; in all cases of ruptured compensation in mitral incompetence we are accustomed to rely upon it with the utmost confidence, and are rarely disappointed. Why should we have misgivings in aortic incompetence? In the early stages of aortic incompetence the heart is well fed, every part of it is flushed with blood which, from the increased size of the blood wave and the position of the coronaries, must be at first at all events at an abnormally high pressure, the nutrition of the heart is specially well provided for, there are no symptoms, and no treatment is required. When from any cause however the compensation is ruptured, an aortic heart will be found as amenable to the beneficial influence of digitalis as any other failing heart, but larger doses are required, but little influence is produced by less than three times as much as would suffice for a mitral heart. Even should the pulse under treatment become abnormally slow, which is not at all usual and certainly not needful to secure benefit, we may rest assured that excessive regurgitation is not then promoted, and though sudden death is not at all unlikely to happen in a badly compensated aortic heart, whether it is treated with digitalis or not, digitalis is never to blame for this. On the contrary, the judicious use of digitalis is the most efficacious treatment in all cases of failing heart, whether that failure be accompanied by aortic or mitral regurgitation. In failure dependent on arterio-sclerosis alone, the tonic influence of digitalis on the heart is hindered unless we combine it with some drug which unlocks the arterioles, and so prevents an increase of the blood pressure, already abnormally high.

REMARKS ON SOME EFFECTS OF THE INTRA-PERITONEAL RUPTURE OF ABDOMINAL CYSTS.¹

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As I have recently had occasion to perform ovariectomy on three consecutive patients in whom the cyst had ruptured previous to operation, it occurred to me that this subject would be an interesting one for us to consider this afternoon. Abdominal cysts may burst into the peritoneal cavity as a result of injury, such as a blow or a fall, in consequence of pressure during labour, or from axial rotation. Frequently the rupture cannot be ascribed to any of these causes; it is then said to be spontaneous. The results following intra-peritoneal rupture vary according to the nature of the cyst. When large parovarian cysts burst, the accident is indicated by sudden acute pain in the belly, followed by disappearance of the swelling. The patient then begins to pass large quantities of urine. In the course of a few hours the bland fluid characteristic of these cysts is absorbed and rapidly excreted. In several reported cases parovarian cysts have burst, refilled, and burst again, the process being repeated several times in a year over a period of several years, the accidents merely causing temporary inconvenience. In ovarian adenomata (multilocular glandular cysts) the fluid is thick and tenacious like mucus. When these rupture the effects are somewhat different, as the following case shows.

M. N., single, aged 48 years, came under my care with a large ovarian tumour. The swelling of the belly had been noticed two years, but had not caused any inconvenience until a few weeks before I saw her, when it commenced to be painful; coincidentally with the onset of pain, the belly had enlarged more rapidly. On physical examination the tumour did not yield the classical signs of ovarian tumour, and there was a peculiar area of dulness in the left loin. A few weeks later ovariectomy was performed, and I found a large ovarian adenoma surrounded by several pints of colloid material, which had escaped into the peritoneal cavity through a rent, 12 centimetres long, in the upper surface of the tumour. The tumour was removed in the usual way, and the peritoneal cavity freely irrigated with warm water at 110° F., and, after much patient sponging, the cavity was cleansed and the wound closed. The woman made an uninterrupted and speedy recovery.

An interesting feature in the case was this: The amount of colloid material free in this woman's belly was many times

greater than the capacity of the ruptured loculus; indeed, it exceeded the total bulk of the tumour. A careful survey of all the facts of the case led me to the conclusion that the cyst had been ruptured several weeks prior to the operation, and the fluid secreted by the glands in the walls of the ruptured loculus had continued to escape into the general peritoneal cavity, where it accumulated, as it was too viscid to be absorbed. The accession of pain probably indicated the occurrence of rupture, and the increase in the size of the belly was due to the accumulation of the escaping fluid, and not to actual growth of the tumour. It is difficult to ascertain with precision the length of time this colloid material will remain in contact with the peritoneum without causing grave changes. In January, 1892, I removed an ovarian adenoma weighing over 60 lbs. from a woman, aged 68. In this case, some of the loculi had ruptured, and a quantity of free colloid stuff had escaped into the general peritoneal cavity, and had probably been there many weeks; yet this old patient recovered without any hesitation.

There is a variety of cyst which arises in that portion of the ovary known as the paroöphoron, in which rupture is followed by curious changes. This is illustrated by one of my cases.

A single woman, aged 28, had noticed for two years a slowly increasing prominence to the right side of the belly. It was pedunculated and freely movable, but irregular in outline; a second tumour could be felt lying to the left of the uterus but not appreciable above the pelvic brim. On opening the abdomen, a large amount of dirty fluid escaped and two large cysts were found. The largest of these had ruptured, and huge warty masses projected from the ruptured loculi. The cyst on the left side had not ruptured and was removed entire. The pelvic peritoneum was dotted with small warts similar to those within the cyst; here and there warts were present on the serous coat of the intestines, on the mesentery and omentum. I regret to add that this patient died five days after the operation.

It has, of course, long been known that when papillomatous cysts of the ovary burst the fluid distributes itself over the peritoneum, and practically sows broadcast cells detached from the warts within the cysts; these are deposited on the peritoneum and ultimately grow into warts. The cysts from this case illustrates very well the way in which the cells become disseminated. In the smaller cysts the warts make their way through the cyst walls by pressure, and having protruded therefrom—as shown in Fig. 1—the emancipated

Fig. 1.—Portion of a warty (paroöphoritic) cyst, to show the warts protruding through the cyst walls.

portions grow luxuriantly, and the cells on the free surface of the warts, detached by the intestines rubbing against them, are disseminated throughout the peritoneal cavity, like so much thistledown over a field of corn. The second and more serious method of dissemination is when a large wart-containing cyst bursts, and these cysts have a natural

⁷ Stockman, *New Official Remedies*, London, 1891, p. 58.

¹ Read before the Thames Valley Branch of the British Medical Association, at Kingston.