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.

tendency to rupture, for the warts growing within so distend the loculi containing them that this persistent pressure interferes with the nutrition of the cyst wall and causes portions of it to necrose and finally yield, allowing the cell-laden fluid to rush out.

In the tumours removed from the patient whose case was last related every stage of the process could be made out, and on the wall of one of the cysts we noticed a line of demarcation around the necrosing segment of the cyst wall.

Hydatid cysts of the liver or omentum give rise to effects somewhat similar to paroöphoritic cysts when they rupture. The natural tendency of hepatic hydatids, especially when they contain large number of secondary cysts is to enlarge until they burst. This event may be prematurely induced by a blow or fall; failing this, the intracystic pressure induced by the multiplication of the daughter cysts leads to the same effect as the growth of warts in paroöphoritic cysts, namely, necrosis of the cyst wall and rupture.

Intraperitoneal rupture of hydatid cysts may be followed by death in a few hours from shock or hæmorrhage; with this we are not concerned to-day. Two results deserve mention. One is the appearance of the hydatid rash, a species of urticaria, which may last for about two days or less; it is intensely itchy and accompanied by high temperature, abdominal pain, and tenderness. Occasionally it occurs after puncture of an abdominal hydatid. Krabbe, in 1866, wrote that the effusion of hydatid fluid into the peritoneal cavity almost always provokes a transient urticaria, and Finsen (1869), in his valuable account of the *Echinococcus Disease of Iceland*, incidentally records several cases to illustrate this clinical phenomenon. The most graphic record runs thus:—Paul Helgason, aged 12 years, had for many years a large tumour in the right hypochondrium, extending to the umbilicus. The lad received a blow from a cow's horn upon the belly, which caused the tumour to disappear; almost immediately afterwards the body was covered by a papular eruption like an urticaria, but it soon disappeared.

The second effect of intraperitoneal rupture of hydatid cysts of which I wish to speak is this: there is reason to believe that when the brood capsules are distributed over the peritoneum many of them become attached to, and grow upon, the omentum and the serous membrane lining the recesses of the pelvis.

Three years ago I operated upon a man who had a large hepatic hydatid, and at the time of the operation the omentum was beset with innumerable specks, varying in size from a millet seed to a cherry stone, and at that time I expressed the opinion that the mother cyst had leaked or else another cyst had ruptured into the peritoneal cavity, and the brood capsules had become transplanted on to the serous surface of the viscera. Several months after the large cyst had been successfully treated the man presented himself with his belly full of numerous rounded bodies, and it gave me the notion of a sack filled with oranges. I reopened the abdomen, and found that many of the cysts, which at the time of the operation were no larger than cherry stones, had grown into cysts some of them as big as the closed fist, while others were as large as walnuts. To remove these was an impossible task, so I excised one and closed the wound. Suppuration occurred among these cysts, and pus flowed freely for several weeks from the peritoneum. The tumours disappeared from the abdomen, the sinus closed, and the patient was discharged. Two years later the man accosted me in the street at Stratford, fat, healthy, and quite a different being. The peritoneal suppuration had killed the hydatids.

With the facts of this remarkable case on my mind, it was but natural that I should feel interested in a case mentioned by Graham.² The patient, a woman, died from a suppurating hepatic hydatid. A cyst in the under surface of the liver had been tapped three months previously. At the *post-mortem* examination the omentum, the spleen, mesentery, and folds of the broad ligaments (Fig. 2) were studded with pearly white tubercles, the size of sago grains. These, when examined microscopically, presented either a shrivelled cyst wall or hooklets. Graham states that in all the similar cases he has seen there was a history of puncture or aspiration of a liver cyst. These facts seem to show that the offspring of the brood capsules may become transplanted on to the peritoneum, and in this respect they resemble the cells from paroöphoritic cysts.

Perhaps the most remarkable example of the dissemination of growths over the peritoneum is that which occurs in connection with ovarian dermoids. Several cases have been recorded in which large dermoids have existed in the ovaries, and at the *post-mortem* examination small dermoids have been

found attached to the omentum, liver, or mesentery. Cases of this kind have been described by Moore,³ Hulke,⁴ Matthews Duncan,⁵ and others. It is now established beyond doubt that ovarian dermoids may, in consequence of axial rotation or dragging on their pedicles, become detached from the uterus and be transplanted to the omentum or some neighbouring part; with these I am not concerned to-day. It is an equally established fact that when eyelashes and fragments of conjunctival epithelium are accidentally conveyed into the anterior chamber of the eye, they sometimes engraft themselves upon the iris and give rise to cysts. These accidentally transplanted eyelashes have been actually seen growing upon the iris.

Fig. 2. The posterior surface of the uterus and broad ligaments beset with minute hydatid cysts (after Graham).

There is reasonable evidence in favour of the belief that, when ovarian dermoids rupture, the dissemination of the epithelial cells and developing hairs may produce a crop of minute dermoids on the peritoneum. For instance, Kolaczek⁶ reported a case in which Martini removed from a single woman, 40 years of age, an ordinary ovarian dermoid as large as a man's head; its surface was perfectly smooth. After the escape of some ascitic fluid the peritoneum was seen to be dotted with small yellow knots; many of them were furnished with a small tuft of light hair, which projected into the peritoneal cavity. Many of the hairs were plucked out, and their nature confirmed by the microscope.

Fraenkel⁷ has recorded the following valuable case: A woman, 37 years of age, had complained of an abdominal tumour for two years. Nine months before operation she fell from a vehicle; this accident was followed by increase in the size of the tumour and peritonitis. At the time of the operation the peritoneal cavity contained dirty fluid mixed with hairs. The tumour was a dermoid which had ruptured into the peritoneal cavity. The surface of the peritoneum was strewn with small pedunculated dermoids, and some larger ones were found in the neighbourhood of the liver, diaphragm, and mesocolon. Here and there isolated hairs were found growing on the omentum. Fraenkel, in the same paper, describes another similar case. Grawitz⁸ has also published a case of the same nature. These observations of Kolaczek, Fraenkel, and Grawitz are very valuable, for they indicate, in no uncertain way, that multiple dermoids on the peritoneum and under surface of the liver are produced in a manner identical with the warts so frequently seen in the peritoneum in cases where warty cysts are growing in connection with the ovaries.

Apart from its clinical interest and importance, the question of the occurrence of secondary growths upon the peritoneum after the rupture of abdominal cysts is one which has a wide and important bearing in relation to the distribution of malignant tumours. This, however, is far too large a subject to admit of discussion to-day, but I will insist that the facts mentioned in this paper may be urged as additional reasons to those usually given when advocating the early removal of ovarian cysts.

³ *Trans. Path. Soc.*, vol. xviii, p. 190.

⁴ *Loc. cit.*, vol. xxiv, p. 157.

⁵ *Trans. Obstet. Soc. Lond.*, vol. xxiv, p. 318.

⁶ *Virchow's Archiv*, Bd. 75, s. 399.

⁷ *Wien. med. Wochenschr.*, 1883, p. 865.

⁸ *Virchow's Archiv*, Bd. 100, p. 262.

² *Hydatid Disease*, 1891, p. 86.