STRANGULATION RESULTING FROM DISTENTION OF HOLLOW VISCERA.

ITS BEARING UPON APPENDICITIS, STRANGULATED HERNIA AND GALL-BLADDER DISEASE.

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SEVERAL times during the past four years I have made the assertion that distention of the appendix back of an obstruction in any part of its lumen would interfere with its circulation even to the point of arresting it entirely.

The following experiments upon dogs were undertaken to demonstrate this fact.

The placing of an electric light bulb in the lumen of the appendix or the intestine made it possible to directly observe the circulation under the microscope and opened up a wonderfully interesting and inviting field for study.

After proper etherization a loop of intestine was withdrawn through an abdominal incision, a canula tied into one end of it and a small electric lamp into the other. The whole was then brought under the microscope and the circulation in the wall of the gut studied under different pressures by water introduced through the canula. The degree of pressure was noted on a mercury manometer, placed in circuit with the pressure bulb. The same experiment was done on the appendix and the same results obtained.

The following are the principal observations as they relate to this study.

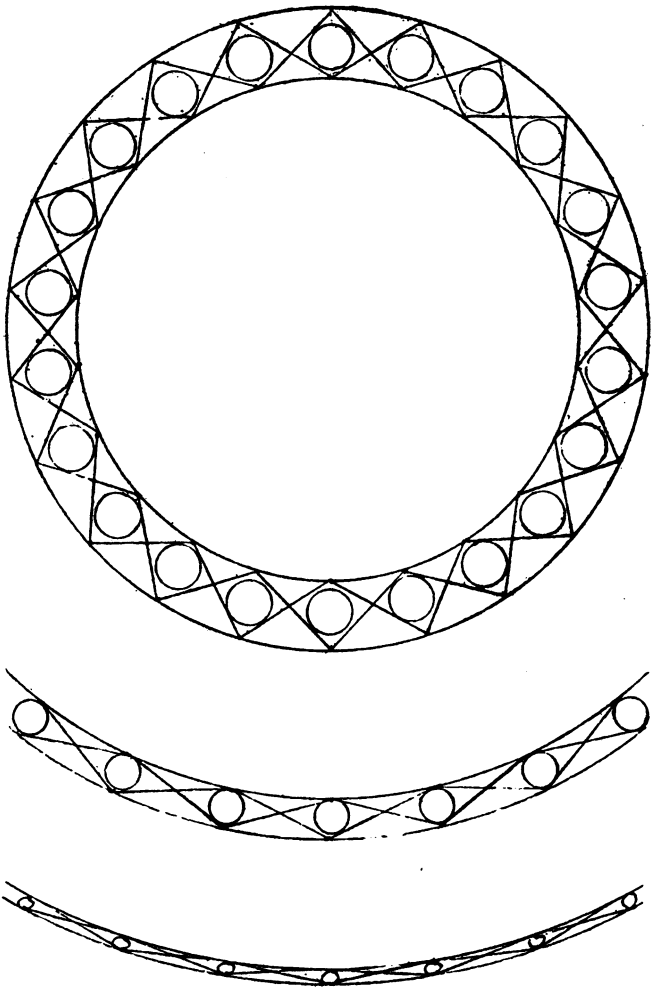
At 30 millimeters mercury pressure we found that some capillary streams were arrested.

At 60 mm. many small veins had their currents arrested, and in most of them the stream was so slow that individual corpuscles could be seen.

At 90 mm. a most interesting study presented itself. All blood streams were moving slowly and many were not moving at all. One was impressed with the difficulties encountered by the circulation in its attempt to find some point of least resistance through which the stream might insinuate

itself. Currents would at one time go in one direction and the next moment in the opposite. At the point of branching of vessels, streams were seen going in all sorts of directions.

FIG. 1.



Schematic representation of constriction of the blood-vessels in the walls of a distended intestine or appendix. (*a*) Normal. (*b*) Moderately distended. (*c*) Fully distended. The tense tissues in the thinning wall blot out the space for blood-vessels.

At 130 mm. all circulation had ceased—the corpuscles standing still in the blood vessels. Many vessels, however, still carried the impulse of the heart beat—the corpuscles moving to and fro, but retaining their relative

positions in the field. This pulsation was seen in some of the largest and the most superficial arteries after the pressure was carried up to 250 mm. (Fig. 1).

Some ecchymotic hæmorrhages were noted as early as 50 mm.

Gut kept at 80 to 90 mm. pressure for an hour was enormously "congested;" that is, the apparent number of veins seen on the serous surface was greatly increased and their caliber was also greatly increased.

Evidently there was great damming back against the aggressive high pressure in the arteries and, in its effort to find exit into the open veins beyond, the stream had been forced into many unfrequented paths.

Pulsation in the veins was noted frequently at the comparatively lower pressures and constantly at high pressures, illustrating the fact that capillary and venous pressures are relative—being the result of the arterial impulse as resisted by the obstruction to the venous return. The more firmly the column of blood is held the more directly and positively the impulse is carried.

That effusion accompanies such obstruction and congestion is so obvious from daily clinical observation that I shall not stop to discuss it here. Enough to say that drops of fluid were observed standing upon the surface of the gut like beads of perspiration. When we remember how quickly exposed peritoneal surfaces become dry and note that the exposure in this case was open to the dry air of Southern California the evidence of effusion becomes still more conclusive.

The demonstration seems complete that distention of gut (or of other hollow viscera) interferes with the circulation in its walls, and allows infiltration and effusion to take place into its walls and lumen and any other open space which may come within its influence.

The return circulation is retarded at comparatively slight pressures. Effusion follows, as in all obstructions to venous flow. As the average venous pressures in the intestines under ordinary circumstances probably varies from 4 to 10 mm. mercury, any pressure beyond that will offer some resistance to the return current. Tiegerstedt (Text-book on Physiology) gives pressures in a number of veins corroborating these

figures. This effusion into the tissues of the walls and into the lumen of the gut increases the distention. When the distention reaches the point where it produces a pressure in the intestine equal to that in the arteries, circulation is entirely arrested.

This mechanical interference with the circulation explains a number of surgical conditions.

In the early pathology of acute appendicitis it is responsible for most of the initial changes.

In connection with the roll of the ball valve in the etiology of appendicitis, I have discussed it in So. Cal. Practitioner, Sept., 1903, Jour. Am. Med. Association, March 26, 1904, and in the ANNALS OF SURGERY, March, 1905.

Acute appendicitis always finds a constricted or kinked appendix. Fecal matter, whether in the form of a concretion or not, lodges behind this constriction, forming a ball valve. Secretion behind the obstruction soon produces some distention, and pressure upon the circulation. This means effusion and more pressure and the vicious circle is established. Effusion and pressure mean infection as soon as resistance is sufficiently lowered by interference with the blood supply, and the minute traumatisms and ecchymotic hemorrhages have made the entrance of germs sufficiently easy.

If the plug is fortunately driven out early through the constriction by the rising pressure the ensuing inflammation may be very slight. But if it remains the lumen of the appendix beond the constriction becomes a veritable abscess cavity and pressure continuing to rise, necrosis or gangrene of the wall is certain to follow.

In strangulated hernia it is a very important consideration. The first step in the process is just enough obstruction at the ring to prevent the easy return of the hernial mass which has been crowded into the sac with perhaps more than its accustomed abdominal pressure. This obstruction is sufficient to retard the venous circulation. This results in effusion into the sac and into the incarcerated gut or any other organ which may be present.

This effusion increases the pressure in the sac and con-

tents, and continues until the height of the arterial pressure is reached when obviously no more fluid can be pumped into it and strangulation is complete.

Any gut present in the sac will suffer the greatest injury on account of the pathogenic germs, always present, attacking the tissues as soon as the resistance is sufficiently lowered by the poor blood supply to allow infection to take place.

In the gall-bladder it often determines the pathological condition. From this cause, in a distended gall-bladder, with an obstructed cystic duct *in the presence of germs* to produce the infection, empyema or gangrene will result. The firmness of the gall-bladder wall offers much greater protection to its circulation than does the intestine, or the appendix, nor is *complete* obstruction so common—the concretion does not commonly form in the very mouth of the duct as it does in the appendix and consequently does not make such a perfect ball valve.

In common duct obstruction infection is much less likely to take place because of the ability of the liver to take up fluids from the bile ducts and thus to keep down the pressure.

The question has often presented itself to me whether this same circulatory interference were not an important factor in gastritis and in ulcer of the stomach. It is conceivable that a heavily loaded stomach might have sufficient interference with its two exits, the œsophagus and the pylorus—to allow sufficient distention to take place to materially interfere with the circulation in the walls of the stomach, particularly the mucosa.

Minute traumatisms and ecchymotic hemorrhages which are early features of this distention should receive due consideration in this connection.

In all abscesses this effusion and pressure upon blood vessels is very marked. The location of the most vulnerable blood vessels in the abscess wall being the first to succumb, determines the direction in which the pus will burrow.

Thus is explained the greater likelihood of an abdominal abscess rupturing into the bowel than anywhere else.

If a considerable surface of gut goes to make up a part

of the wall of an abscess the blood vessels in that wall will have only the thin membrane like intestinal wall to protect them from pressure. There is no possibility of their being backed up behind by exudate as that is a mucous surface.

As the abscess wall is put upon the stretch the effect upon the intestinal blood vessels is the same that it is when the pressure is from within the gut. By stretching, all interstices in the wall are blotted out and circulation is out of the question. The effect is the same as the steady pull upon the broad ligament when operating in the pelvis. As long as it is kept taut there is no bleeding, but when this tension is relaxed the blood vessels reopen and hemorrhage appears.

The vessels in the abdominal wall, for instance, have thick cushions of tissue to resist this stretching and consequent obliteration, with many blood vessels to reinforce those in the abscess wall.

With the help of the ubiquitous omentum the peritoneal exudate which makes so large a proportion of the wall of abdominal abscesses can go on indefinitely backing up with more exudate to take the place of the gradual necrosis going on "at the front."

Many other conditions which are affected by this circulatory disturbance from distention might be discussed. This is not an attempt at making this an exhaustive catalogue. I will only suggest for consideration the following:

Intestinal distention in typhoid fever with its greater danger of perforating ulcers and hemorrhage. Its importance in all forms of ileus only casually suggested by Kocher,[1] but really one of the greatest factors in the multiform pathology of that condition. Intra-cranial pressures, so beautifully demonstrated by Cushing.[2] Overdistentions in the urinary tracts, bladder, ureters and kidneys. Effusions into joints, with or without pathogenic germs being present. Fluids in tumors. And even in excessive uterine distention in pregnancy

[1] Ueber Ileus, Mitth. aus der Grenzgebieten der Medizin und Chur., vol. iv, 1899.

[2] Am. Jour. Med. Sci., September, 1902.

this mechanical burden upon the circulation should be given due consideration.

In the past few years much work has been done upon absolute pressures in the blood vessels, but outside of these there is still a wide field for investigation. The hydraulics of the body are worthy of much greater prominence than have been given them.

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