SOURCE CONTROL

A century of source control to manage peritonitis of colonic origin

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The force behind the source: Colonic bacteria

The large bowel contains up to 400 different bacterial species with concentrations of $10^{12\text{-}13}$/ml of feces. All require an intact intracolonic environment to prosper and stay in balance for the benefit of the host [1 2]. The colon wall has a mature host defense system to keep bacteria in check by preventing their transmural migration into the peritoneal cavity. Structural bowel wall changes such as diverticula and inflammatory bowel disease and cancer interfere with the colonic wall's ability to contain bacteria and a perforation may result. Similarly impaired blood supply may diminish oxygen and nutrient supply to cellular structures of the colonic wall and

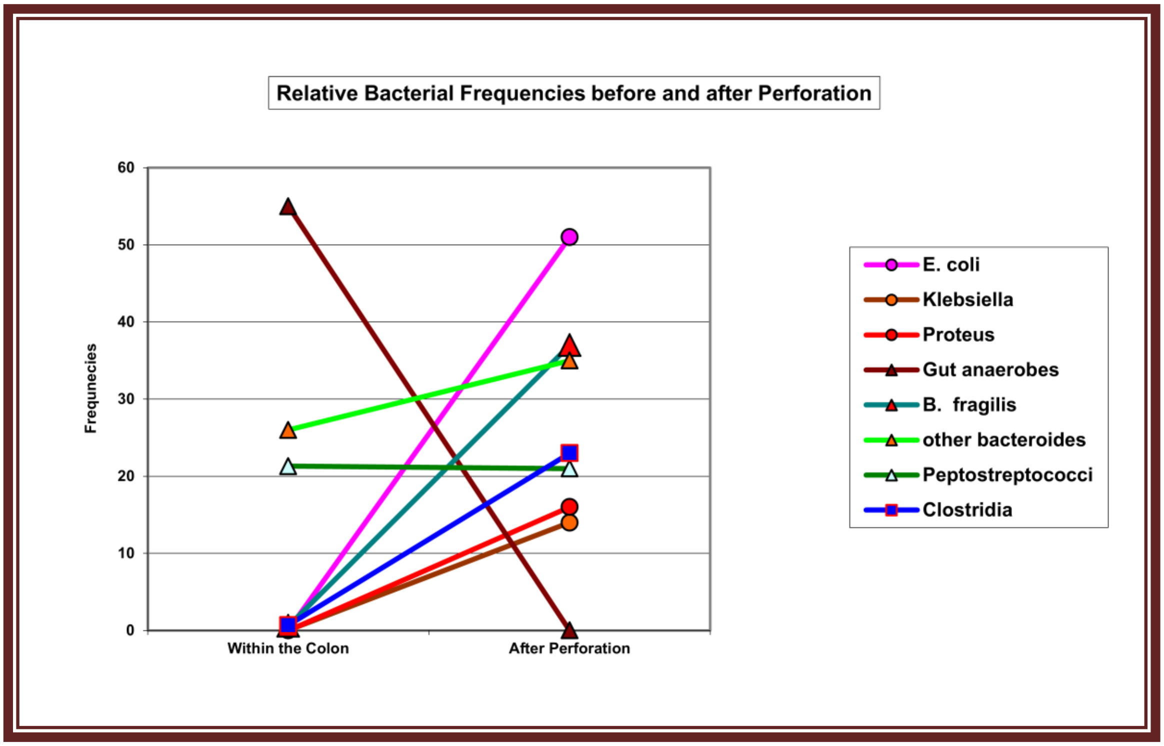


Figure 1: Survival of the fittest

impair colon function in general and cellular defense in particular. Perfusion deficits may progress to franc ischemia when several factors add up. Such scenario may be seen when an inflammatory edema increases intra-abdominal pressure, reduces venous and arterial blood flow and surgical sutures strangulate perianstomotic arterioles [3]. Rather than act as a bacterial barrier and defense against bacterial invasion, the ischemic colon wall now serves as nutrient for bacterial growth fostering their access to the peritoneal cavity. Figure 1: Relative frequency of colonic bacteria within the normal colon and compared to bacterial frequencies within the peritoneal cavity following perforation.

Only few of these bacteria, however, can survive outside the colon following perforation or migration through a diseased and defenseless colon wall. A comparison of bacteria from the healthy colon and the peritoneal cavity after colon perforation identifies bacterial survivors that adapt to the new environment. (Figure 1) Comparing their relative numbers may give a clue as to bacterial pathogenicity. *E. coli* e.g. makes up less than 0.1 % of the intracolonic flora, colonic; following perforation it is the most commonly isolated bacterium accounting for more than 50% of the isolates. Conditions outside the colon favor its growth producing huge amounts of endotoxin when attacked by powerful peritoneal and systemic defenses. Small amounts of endotoxin may kill the host. *Bacteroides species* on the other hand are more common intracolonic microorganisms. They are, however, similarly often present in cultures taken from the peritoneal cavity after perforation indicating mere survival and not special affinity to the extra colonic environment. For the growth of this organism conditions inside and outside the colon resemble and it may grow within the peritoneal cavity and cause disease. *Bacteroides*, however, may be less dangerous for the host than *E. coli,* because it does not find the same optimized living conditions outside the colon as *E. coli* does. In peritonitis *E. coli* is isolated 300 times the rate of its intracolonic concentration.

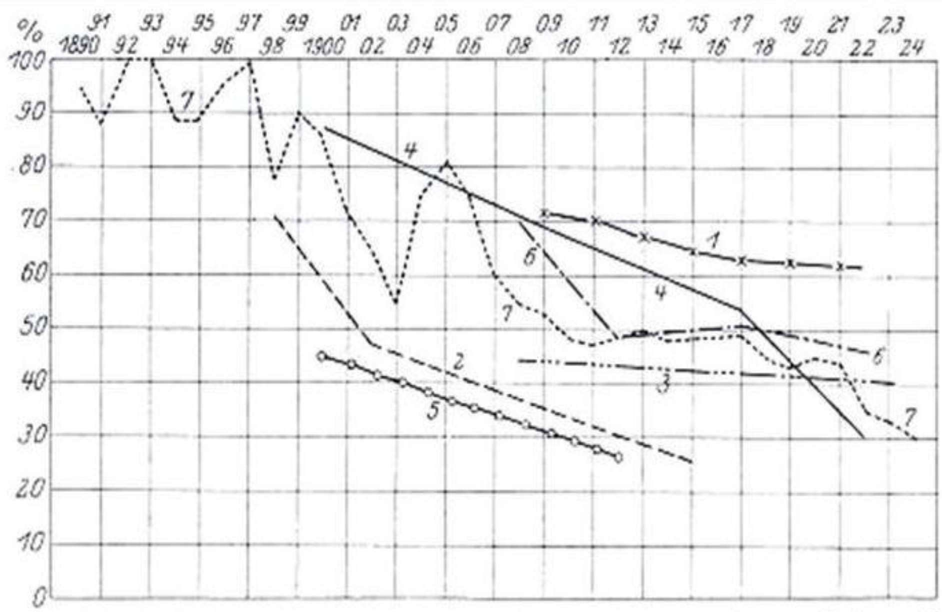
The pathogenic potential of colonic bacteria that adapt well to the extra colonic environment, therefore, is the force that causes disease upon perforation. The perforation itself is the source in this process. It must be closed or eliminated to save the patient. When this was not possible about 100 years ago 90 to 100 percent of all patients with perforations died. [4]

The therapeutic effect of source control

The therapeutic effect of source control may be best assessed when looking at conditions where source control was the only available therapeutic option and other means of therapy such as antibiotics and fluid resuscitation were not developed. Towards the end of the 19th century we learned to access the abdominal cavity surgically to treat intra-abdominal infections [5]. Dramatic therapeutic successes were seen and gradually more abdominal procedures were introduced and performed. Surgeons experienced the benefits of closing the source of infection when they operated to treat intra-abdominal infections. In 1902 Rehn mandated "radical elimination of the cause (source) of peritonitis [6], and W Nötzel reported in 1909 a dramatic improvement of mortality that was attributed to radical source control as mandated by his teacher Rehn [7] . His reports include 449 operations for peritonitis from 1891 to March 1909 of which 178 were cured, a 38% mortality. All patients with diffuse suppurative peritonitis including the most desolate cases were treated without exception. Considering that no antibiotics were available, the fluid resuscitation was in its infant shoes and meaningful medical and critical care therapy was not developed yet, one must conclude that source control and maybe purging the abdominal cavity from pus (they used copious intra-operative irrigation with saline) were the single most important factors that improved outcome by a 62% mortality reduction. Other institutions gradually applied the source control philosophy. Their successful efforts to close the source of infection surgically were rewarded by ever decreasing mortality rates (Figure 2). Kirschner, in 1926, presented an impressive statistical analysis of 5468 patients with peritonitis of 7 major institution from 1890 to 1924. [4] The mortality curves indicate a parallel decline in all 7 hospitals alike (Figure 2).

Figure 2: Mortality of 5468 cases peritonitis from 1890 to 1924

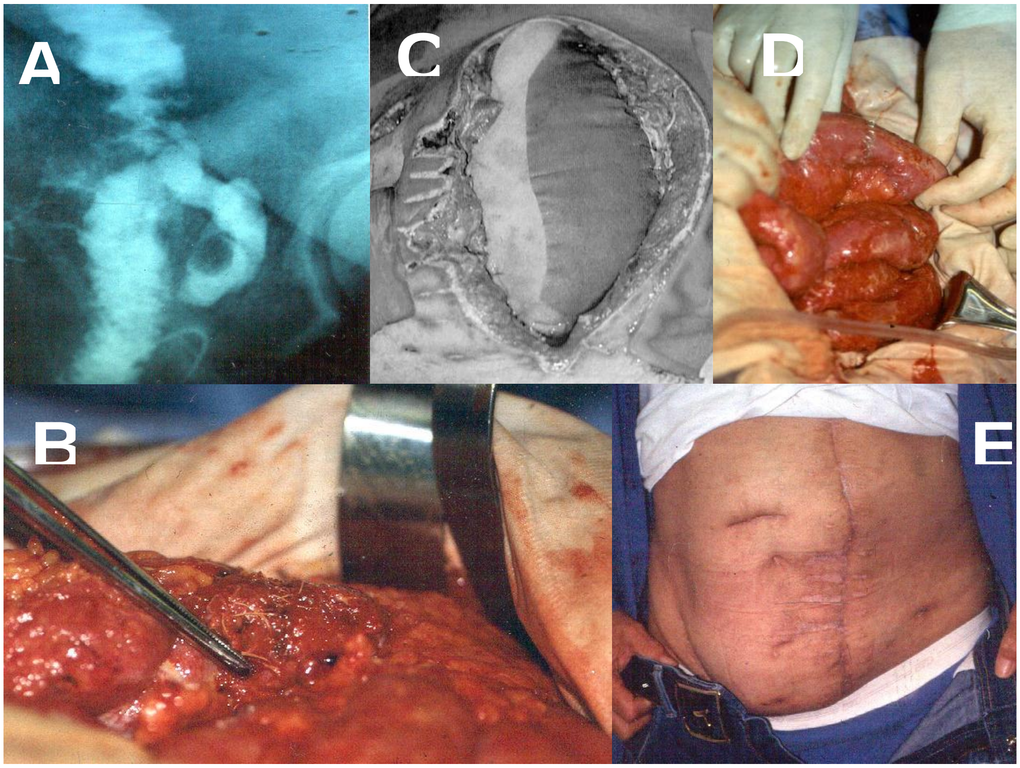
.During this time period the principle of source control was introduced and increasingly applied clinically. [4]



Tab. 18. Sterblichkeit 7 verschiedener Kliniken zu verschiedenen Zeiten (5468 Fälle).

1. *Braun*, Zwickau. ×—×—×
2. *Heidenhain*, Worms. - - - - -
3. *Kappis*, Hannover. -···-···
4. *Kirschner*, Königsberg. ———
5. *Körte*, Berlin. o—o—o—
6. *Schöne*, Stettin. ·—·—·—·
7. *Sudeck*, Hamburg. ··········

Figure 3: Source control under difficult circumstances with STAR (Staged abdominal repair)



This 62 y o man with macronodular liver cirrhosis developed diffuse peritonitis from an anastomotic leak following sigmoid colon resection. A colostomy had been performed and the leak was sutured in an attempt to control peritonitis. The infection, however, continued due to continuous bacterial flow through a persistent anastomotic leak (a). The colostomy did not prevent peritonitis and the patient was transferred with multiple retention sutures cutting through the edges of a pararectal incision. Upon exploration we found multiple small bowel perforations (injuries from retention sutures), diffuse peritonitis and a leaking colon anastomosis. We resected the small bowel and resected the leaking anastomosis sutured two new anastomoses during STAR entry #1 and closed with the artificial bur (Wittmann Patch) after debridement and copious irrigation with R/L. At STAR entry # 3 on the third day, the colostomy was taken down (B) and anastomotic healing controlled (D). Picture C shows the artificial bur in place neutralizing increased intraabdominal pressure and also sowing the previous colostomy site in the upper left corner. D shows the patient after recovery.

When the source of peritonitis is the colon

During the historic period when the source control principle was introduced, colon origin peritonitis had the worst outcome compared to peritonitis from other sources such as a perforated appendix or perforations of the upper GI tract. The colonic origin mortality was 100% from 1895 to 1914. It decreased in the to 68% and 50% from 1915 to 1919 and 1920 to 1924 respectively while in the latter period peritonitis with the appendix and upper GI tract had decreased to 20% and 42.2% respectively [4] emphasizing the importance of source control. While an appendectomy permits a safer source control, it is surgically more difficult to close or eliminate a perforation of the retroperitoneally fixed upper GI-tract or the colon. The technique exteriorizing the colon perforation was not developed yet and colostomies as a source control measure were introduced during second world war. Also the relatively higher amount of bacteria perforating into the peritoneum after colon perforation over upper GI-tract perforations may have contributed to the higher mortality.

Source Control: refocus on a solved problem

Following the discovery of the importance of source control and its clinical application about 100 years ago, the source control idea to become a self-evident feature of daily praxis and teaching and source control discussion went out of focus.

Recent developments in surgery and clinical research, however, showed that the faded idea of source control had detrimental consequences for the interpretation of clinical trials. It became evident that is very difficult to value outcomes in trials testing new antimicrobials [8] and new immunological strategies [9] [10] [11] without precise definition of source control. This problem prompted researches and surgeon under the leadership of John Marshall to refocus on source control. [12].

How is a source of colonic origin controlled

Any colon wall pathology leading to free bacterial flow into the peritoneum can be classified into three fundamentally different types of colon perforation:

1. Transmural Traumatic perforation following accidental and non-accidental injury
2. Perforation following localized necrosis from disease
3. ischemic necrosis due to impaired vascular flow

The choice of therapy depends on the general condition of the patient, the inflammatory response of the peritoneum, the level of increased intra-abdominal pressure and the pathology itself.

Treatment options available to eliminate any colon perforation are

1. Simple suture closure of the perforation
2. Resection of the diseased colon segment with concomitant anastomosis
3. Resection of the diseases colon wall with delayed anastomosis
4. Exteriorization of the colon segment that is perforated
5. Resection of the perforated colon segment without anastomosis and formation of a terminal colostomy with or without a mucous fistula.
6. STAR (Staged abdominal Repair) to secure source control by daily inspection during abdominal reentries

The goal of any of these procedures is to securely prevent the perforation from permitting further bacterial flow into the peritoneal cavity. If abdominal hypertension prevents fascial closure at the initial operation, or if sepsis has created the situation of hemodynamic instability that allows for emergency measures only, a damage control procedure may be a meaningful initial step. The source can be eliminated by simple resection and closing the transected colon openings by stapler or simple ligature temporarily. Establishing of intestinal continuity can be deferred to be done during subsequent abdominal reentries some 24 hours periods later when conditions have improved. If, at that point there are still concerns about healing of anastomosis without leak, re-explorations can be done the ensure a proper healing process and if necessary correct emerging problems early

The newly developed STAR procedure that uses a artificial bur device as a temporary fascial prosthesis permits fascial expansion and temporary abdominal closure at the same time to allow for and planned abdominal reentries and finalization of repair and final fascia closure.

The method offers the additional advantage to be able to inspect the abdominal cavity and check that the source has been closed permanently . If necessary defective closures can be repaired before major bacterial masses regain access into the peritoneum (Figure 3). [13-16]

Table 1

Possible source control procedures for the three types of colon perforations. There are no prospective randomized trials to guide the surgeon's choice for any particular procedure. In recent publications, however, many surgeons seem to abandon operations

Table 1: Source control operations for colon perforations

Operation\Pathology	Traumatic perforation following accidental and non-accidental injury	Perforation following localized necrosis such as diverticulitis	Transmural ischemic necrosis due to impaired vascular flow
Simple suture closure of the perforation	yes	possible	no
Resection of the diseased colon segment with concomitant anastomosis	yes	yes	yes
Resection of the diseases colon wall with delayed anastomosis	yes	yes	yes
Exteriorization of the colon segment that is perforated	possible	possible	difficult
Resection of the perforated colon segment without anastomosis and formation of a terminal colostomy	possible	possible	possible
STAR procedure with multiple abdominal reentries and final fascial closure	yes	yes	yes

with exteriorizations and formation of colostomies because those procedures add numerous additional problems associated with the colostomy and the eventual take down of the colostomy. Also, bedsides patient disgust for colostomies, it may be difficult to prevent stool from leaking from a colostomy into the abdominal wound which matters most when planned relaparotomies or STAR is required. It seem to be a much better idea to favor a primary anastomoses and check out the healing process at subsequent abdominal entries and close the abdomen only when source control is certain. [4 17]

For the conduct of clinical trials testing newer non operative therapies, source control criteria of table 1 may be used to assess outcomes more effectively. It is not possible to favor one of the mentioned procedures over the other and further research may help to clarify optimal operative management. The pivotal point of success, however, is a permanently close infectious source within the colon wall. Post surgical follow/up information must be included in the study design to document the safe closure of the source or a recurrent leak.

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