

World Journal of Surgery

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## World Progress in Surgery

## Intraabdominal Infections—Introduction

Before operative therapy was generally used, about 90% of all patients with intraabdominal infection died from sepsis. This outcome might be regarded as the natural course of the disease. When principles of surgical management were established and became commonly utilized, the mortality dropped below 50% in large series [1]. The improved survival of 40–50% must be credited to operative management alone since, during the first 3 decades of this century, efficacious antibiotics were not available and effective critical care treatment was not possible.

Fleming discovered penicillin in 1929 [2] and its later introduction into clinical medicine led to dramatic therapeutic improvements in surgical infections [3]; however, in subsequent decades, the mortality risk of intraabdominal infection was not affected by antibiotic therapy and the average mortality reported remained unchanged until the 1970's [4] (Fig. 1). In the past 10 years, better survival rates have been reported. It is difficult, however, to attribute the most recent improvement to one specific therapy since several new supportive techniques for the care of patients with serious intraabdominal infection have been recently introduced. These advances include new operative techniques (Tables 1, 2) [5-30], more potent antimicrobials, new concepts of hemodynamic, respiratory, and renal support guided by direct measurement of cardiac performance, and new radiographic techniques for localizing and treating (nonoperatively) abscesses.

Despite advances, mortality from many forms of intraabdominal infection remains unacceptably high. Substantial differences between conventional and more recently developed therapies have not been found in randomized prospective studies. It has become apparent that approaches for managing patients profoundly ill from intraabdominal infection requires further critical review and that new methods for analyzing the results of various therapeutic interventions must be found.

With this background, an international congress on intraabdominal infections was organized in Hamburg in 1987, supported by the Surgical Infection Society (SIS) and the Paul Ehrlich Society. Surgeons from all continents came to review the current status of definitions as well as statistical techniques and severity-of-illness scoring systems, to allow for more sophisticated analysis of results. Also analyzed were all new and innovative operative techniques which had been developed because of the failure of accepted therapies to greatly alter outcome. Additionally, a broad range of subjects were presented on all aspects of intraabdominal infection including diagnosis, pathogenesis, bacteriology, inflammation and immunology, animal models of intraabdominal sepsis, intensive care, multisystem organ failure, antimicrobial therapy, and nonoperative treatment for intraabdominal abscess.

Participants in the session on "Definitions and Risk Factor Analysis and Severity Scoring: Foundation for Research and Clinical Trials" continued the discussion at subsequent meetings. The results of these conferences are presented in the first article of this Progress Symposium. Although the APACHE II score is difficult to assess and is not specific for intraabdominal infections, it was recognized as the most widely accepted prognostic index. While other more specific scores may be easily assessed, and have been validated in large patient populations and shown to exhibit the same prognostic value as APACHE II, preference was given to the SIS-modified APACHE scoring system. Its use was recommended for better comparison of critically ill patients and as a baseline reference for future studies. During these discussions, it became obvious that the predictive power of scoring systems are limited and further improvement may not be possible. Other yet unknown techniques may be necessary to accurately measure the biological variances seen with intraabdominal infection.

In the second article of this symposium, the authors address new types of intraabdominal infections now being seen, which are difficult to understand [31, 32]. They have defined them as tertiary peritonitis. During the Hamburg meeting, it was not possible to develop a more practical classification system to include all aspects of this disease such as chemical peritonitis, intraabdominal abscess, spontaneous peritonitis, traumatic peritonitis, serofibrinous peritonitis, tertiary peritonitis, etc. Until a better nosologic answer is found, perhaps the classification of peritonitis given below might be utilized:

- I. Primary Peritonitis
  - A. Spontaneous peritonitis of childhood
  - B. Spontaneous peritonitis of adult
  - C. Peritonitis in patients with CAPD (continuous ambulatory peritoneal dialysis)
  - D. Tuberculous peritonitis
- II. Secondary Peritonitis (Acute Suppurative)
  - A. Perforation peritonitis (spontaneous acute)
    - 1. Gastrointestinal tract perforation
    - 2. Bowel wall necrosis
    - 3. Pelviperitonitis
    - 4. Peritonitis after translocation of bacteria
  - B. Postoperative peritonitis
    - 1. Leak of an anastomosis
    - 2. Leak of a suture line
    - 3. Stump insufficiency



Table 1. "Open abdomen technique" for treating advanced diffuse peritonitis.

Author	Year	Died/total	Mortality (%)
Champault et al. [6]	1979	16/27	59
Guivarch et al. [7]	1979	4/16	25
Steinberg [8]	1979	7/14	50
Hay et al. [9]	1979	34/64	53
Duff and Moffat [10]	1981	7/18	39
Maetanai and Tobe [11]	1981	2/13	15
Doutre et al. [12]	1982	16/29	55
Anderson et al. [13]	1983	12/20	60
Broomé et al. [14]	1983	14/30	47
Hollender et al. [15]	1983	7/22	32
Andrus et al. [16]	1986	21/34	62
Mughal et al. [17]	1986	5/18	28

**Fig. 1.** Mortality of intraabdominal infections: Mean of 76 studies (from D.H. Wittmann, Habilitation, Hamburg University Medical School, Federal Republic of Germany, 1984).

Table 2. Planned relaparotomies/etappenlavages for intraabdominal infections and pancreatitis.

Author	Year	Device used for temporary abdominal closure	Interval between relaparotomies (hr)	Died/total	Mortality (%)
Hay et al. [9]	1979	Marlex <sup>®</sup> mesh	24	9/26	35
Fagniez et al. [18]	1979	Polyurethane foam	Variable	21/70	30
Goris [19]	1980	Marlex <sup>®</sup> mesh	Variable	13/26	50
Kerremans and Pennickx [20]	1982	Retention sutures	48	15/39	38
Teichmann et al. [21]	1982	Retention sutures	24	4/21	19
Wouters et al. [22]	1983	Marlex <sup>®</sup> mesh	Variable	4/20	20
Penninckx et al. [23]	1983	Retention sutures	48	9/31	29
Stone et al. [24]	1984	Zipper	Variable	7/36	19
Bartels et al. [25]	1985	Retention sutures	48	14/46	30
Muhrer et al. [26]	1985	Vicryl <sup>®</sup> mesh	48	11/27	41
Heddrich et al. [27]	1986	Marlex <sup>®</sup> + zipper	48	2/10	20
Teichmann et al. [28]	1986	Slide fastener	24	14/61	23
Garcia-Sabrido et al. [29]	1988	Zipper-mesh	24	15/64	23
Wittmann [30]	1990	Velcro <sup>®</sup> (burr-like)	24	28/117	24

- 4. Other iatrogenic leaks
- C. Posttraumatic peritonitis
  - 1. Peritonitis after blunt abdominal trauma
- 2. Peritonitis after penetrating abdominal trauma
- III. Tertiary Peritonitis
  - A. Peritonitis without pathogens
  - B. Peritonitis with fungi
  - C. Peritonitis with low-grade pathogenic bacteria
- IV. Intraabdominal Abscess
  - A. Intraabdominal abscess with primary peritonitis
  - B. Intraabdominal abscess with secondary peritonitis
  - C. Intraabdominal abscess with tertiary peritonitis

Most surgeons refer to peritonitis as an intraabdominal infection due to a perforation of an intestinal organ. Intraabdominal infection and peritonitis, however, are not synonymous. Although commonly used to describe a suppurative intraabdominal process, "peritonitis" actually means inflammation of the peritoneum, or of a part thereof, which may not necessarily be due to infection. The term "intraabdominal infection" implies an infectious disease process and requires identification of the causative infecting microorganism. The body's response to intraabdominal infections is the same as that for peritonitis. Thus, peritonitis should be regarded as a general class which includes the specific entity, intraabdominal infection.

Intraabdominal infections are not solely a local disease, but affect the entire body with subsequent organ system dysfunction. These pathophysiologic responses of the host to the inflammatory and bacterial challenges are addressed in the articles by Hau, Christou, Runcie and Ramsay, and Offenbartl and Bengmark. The important pathogenic issue of adherence of bacteria to peritoneal cells is highlighted in the contribution by Edmiston and associates. Consequences of organ function, although a major issue during the Hamburg congress, are still poorly understood and these 5 contributions deal with current concepts.

Four further articles address therapeutic issues: the present concepts of antimicrobial therapy, the classical concepts of operative therapy, nonoperative management of intraabdominal abscesses, and experience with the more aggressive operative management of etappenlavage. During the Hamburg congress, it became obvious that the open abdomen technique for treatment of advanced diffuse suppurative peritonitis carries a risk of too many complications [6–18]. Planned relaparotomies with various devices for temporary abdominal closure seems to be the answer for the subset of patients with advanced infections

causing grave damage to distant organ systems [19–32]. The Surgical Infection Society and associated individual groups are presently working on improving these techniques.

Hopefully, methods of measuring the true benefit of these procedures will be utilized to find a definitive answer in the near future. This will then be the time that a second international meeting on intraabdominal infection should be organized. Answers to questions raised during the Hamburg congress will then be ready for critical review. Studies of managing patients profoundly ill from intraabdominal infection should be able to define the ultimate goal of treatment which, at the present time, seems unlikely to be a zero mortality.

> Dietmar H. Wittmann, M.D., Ph.D. Guest Editor

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