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Bacteremia due to *Acinetobacter* Species Other than *Acinetobacter baumannii*

Summary: The objective of this study was to describe the clinical features, possible predisposing factors and treatment outcomes associated with bacteremia du to *Acinetobacter* species other than *Acinetobacter baumannii*. A review of laboratory and medical charts over a period of 18 months revealed 61 cases of bacteremia due to *Acinetobacter* species other than *A. baumannii* occurring in 59 patients. Six of these were considered not significant. Fifty cases represented catheter-related bacteremia, one case was associated with meningitis following brain surgery, and four cases could not be classified. Clinical courses wre usually benign: all but four patients were cured, but death was not related to *Acinetobacter* bacteremia in any case. Therapy included catheter removal alone (32.8%), appropriate antimicrobials alone (12.7%), or both (49.1%). Plasmid analysis showed distinct patterns in all strains isolated from different patients and did not reveal any epidemiological relationship among cases. *Acinetobacter* species other than *A. baumannii* are clinically significant organisms with limited pathogenic potential. They are almost exclusively involved in devise-related bacteremia. Clinical and epidemiological features of infections due to these organisms are clearly distinct from infections due to *A. baumannii*.

Introduction

Acinetobacter baumannii (formerly *Acinetobacter calcoaceticus* var. *anitratus*) is well recognized as an important cause of infectious morbidity and mortality in hospitalized patients, mainly affecting patients with impaired host defenses in the ICU setting. Resulting illnesses include meningitis, pneumonia, peritonitis, urinary tract infection, and bacteremia [1–8]. Hospital outbreaks have been associated with contaminated ventilators and pressure transducers [3, 7]. Multidrug resistance is common among these organisms and often leaves few therapeutic options [9, 10]. Currently, little is known about the clinical significance and hospital epidemiology of *Acinetobacter* species other than *A. baumannii* (formerly *Acinetobacter calcoaceticus* var. *lwoffii*). These organisms are considered to be part of the indigenous flora of human skin and have only rarely been implicated as a cause of infection in humans. We conducted a detailed study of 61 consecutive cases of bacteremia caused by *Acinetobacter* spp. other than *A. baumannii*. We describe possible predisposing factors, clinical and bacteriological features, and outcome. Plasmid DNA analysis was used in an attempt to identify possible cross infections due to these organisms. These cases, to our knowledge, constitute the largest series of bacteremia cases caused by these organisms reported to date.

Patients and Methods

Patient selection and evaluation: The Institute of Medical Microbiology and Hygiene, University of Cologne, serves two large teaching hospitals and ten smaller hospitals. The study period extended from 1 July, 1990 through 31 December, 1991. A case of *Acinetobacter* bacteremia was defined as a patient from whose blood any species of *Acinetobacter* other than *A. Baumannii* was isolated in one or more blood cultures collected when clinical evidence of infection was present. Second episodes were included, provided more than 3 days had elapsed since resolution of signs and symptoms of the previous episode. Cases were identified through review of the laboratory charts. The medical records of all case patients were reviewed. The following clinical and epidemiological data were recorded: date of birth, sex, dates of admission to hospital and discharge, clinical service and ward location, admission diagnosis, all important diagnoses and underlying conditions, dates and sites of isolation of *Acinetobacter* spp., dates, sites and species identification of other isolates obtained from any body site, the number of blood culture bottles drawn and the number of positive bottles, invasive procedures including surgery performed within 4 weeks before the acquisition of *Acinetobacter* spp., exposure to indwelling catheters, number of days with fever and bacteremia before and after catheter removal and before and after initiation of antibiotic therapy, dose and duration of precedent antimicrobial and heparin treatment, and outcome in relation to catheter removal and initiation of appropriate antimicrobial therapy. Underlying diseases were classified according to the criteria of *McCabe* and *Jackson* [11] as fatal (death anticipated during hospitalization), ultimately fatal (death anticipated within 5 years), and nonfatal.

Definitions and clinical parameters: Fever was defined as a temperature > 38°C rectally. Neutropenia was defined as a neutrophil count less than 0.5×10^9/l. Leukocytosis was defined as a total leukocyte count greater than 12×10^9/l, and leukopenia as a count less than 2×10^9/l. Thrombocytopenia was defined as a

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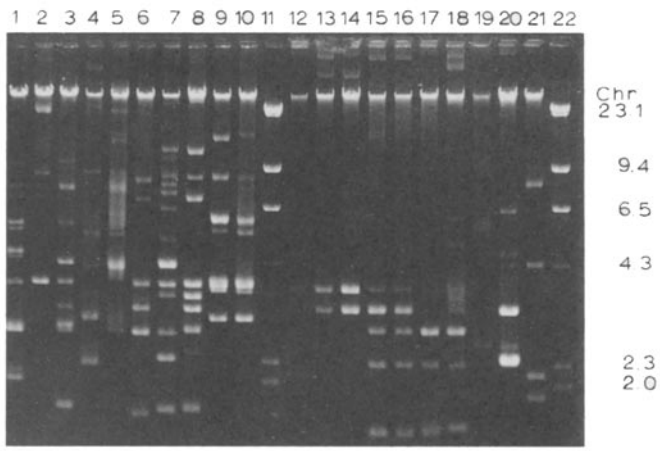

Chr = chromosomal DNA.

Figure 1: Plasmid DNA profiles of *Acinetobacter* isolates obtained from blood cultures and intravascular catheters. Lanes: 1 to 8, *Acinetobacter lwoffii* isolates obtained from different patients; 9 to 10, *Acinetobacter lwoffii*, blood culture and central venous catheter isolate from patient A; 12 t 20, *Acinetobacter* species 10 isolates; 12 to 14, isolates recovered from two blood cultures and a peripheral line from patient B; 15 to 18, blood culture and central venous catheter isolates obtained from patient C during two consecutive bacteremic episodes (see text for details); 19 to 20, isolates recovered from different patients; 21, *Acinetobacter haemolyticus* isolate; 11 and 22, molecular size marker, in kilobases (Hind III digest of lambda DNA).

platelet count less than 125×10^9/l. Sepsis was defined according to criteria published by the American College of Chest Physicians/Society of Critical Care Medicine consensus conference as the systemic response to infection manifested by two or more of the following conditions: temperature > 38°C or < 36°C, heart rate > 90 beats/min, respiratory rate > 20 breaths/min, and total leukocyte count > 12×10^9/l or < 4×10^9/l [12].

Colonization and infection were defined according to previously published criteria for the study of nosocomial infections [13]. Catheter-related bacteremia (definition modified from *Raad* and *Bodey* [14] was defined as a primary bloodstream infection in which clinical evidence implicates the catheter as the source of infection. The clinical evidence could be one of the following: (1) an exit site infection due to the same organism as that isolated from the bloodstream or (2) resolution of the clinical sepsis within 48 h of catheter removal while the patient is receiving no active antimicrobials or after an unsuccessful trial of active antibiotics for at least 72 h. Probable catheter-related bacteremia was defined as the isolation of *Acinetobacter* from one or more blood cultures from a patient with clinical manifestations of sepsis and no apparent source for the sepsis except the catheter. Probable catheter-related septicemia was also considered if the same organism was cultured from the catheter tip as from the bloodstream, but the above mentioned clinical criteria for definite catheter-related bacteremia were not met, because therapy included catheter removal and appropriate antimicrobials at the same time.

Prior antibiotic therapy was defined as the receipt of a systemic antimicrobial agent for at least 48 h within the preceding 10 days. The empirical antimicrobial treatment was considered appropriate if the organism was susceptible *in vitro* to at least one of the drugs administered. Cure was defined as complete resolution of signs and symptoms of infection. Death was considered related to *Acinetobacter* bacteremia if the patient had died within 72 h after a blood culture positive for *Acinetobacter* spp. had been obtained.

Microbiological methods: Blood samples for culture were drawn by the clinical staff and inoculated into a two-bottle blood culture system. A 5-ml volume of blood was injected into each of two 50-ml bottles, one vented and one unvented. Subcultures and Gram-staining were performed routinely on days 1 and 7 and if bottles showed turbidity. Catheter tips were cultured by placing the distal portion of the catheter in trypticase soy broth (TSB) for up to 7 days. *Acinetobacter* spp. were presumptively defined as gram-negativ, oxidase-negative, nonmotile, nonfermenting coccobacilli. Identification at the genus level was confirmed by the transformation assay of *Juni* [15]. Species identification was done with the simplified identification scheme of *Bouvet* and *Grimont* [16], including growth in TSB at 37°, 41°, and 44°C, acid production from glucose, gelatin hydrolysis, and carbon source assimilation tests. A standardized microtiter broth dilution method was used for susceptibility testing as described previously [9]. Plasmid DNA analysis was performed as described by *Hartstein* et al. [17] with minor modifications.

Results

Study Population

A review of the laboratory charts revealed 68 consecutive cases of bacteremia due to *Acinetobacter* spp. other than *A. baumannii*. Seven cases were excluded from further consideration because medical records were not available or incomplete. Of the remaining cases, six (9.8%) were considered not significant because fever was found to be due to other underlying conditions and there were no other clinical signs or symptoms to suggest infection. Thus, a total of 55 episodes of true bacteremia occurred in 53 patients within the 18-month study period. The majority of cases was observed in three hospitals: 20 and 13 cases, respectively, occurred in two tertiary care centers, 11 cases in a community hospital; the remaining cases occurred in seven community hospitals with one to four cases in each hospital. Thirteen of these cases have been described in a previous report [18]. Two patients experienced a second episode of *Acinetobacter* bacteremia due to the same species, 3 and 4 days after resolution of fever in the previous episode. For the purpose of this study these episodes were considered separate cases although bacteriological relapse could not be ruled out. The demographic features and clinical characteristics of these patients are shown in Table 1. Continuous intravenous infusion of heparin was given to 35 patients (63.6%) for different reasons including thromboembolic disease in 12, coronary heart disease including myocardial infarction in seven, cardiac valve replacement in four, bypass surgery of angioplasty in three, and rheumatic heart disease with atrial fibrillation in two patients. None of the patients were mechanically ventilated or treated in an intensive care unit. Bacteremia occurred more than 72 h after admission and was thus considered hospital-acquired in all but one episode.

Microbiological Data

Acinetobacter johnsonii (14 isolates), *Acinetobacter* species 3 (12 isolates), and Acinetobacter *lwoffii* (ten isolates) were the pathogens most frequently encountered in the 55 cases of *Acinetobacter* bacteremia (Table 2). A total of 11 episodes of *Acinetobacter* bacteremia were polymicrobial in nature with pseudomonas, enterococci and coagulase-negativ staphylococci cultured most frequently as copathogens. *Acinetobacter* spp. were recovered from more than one blood culture in 20 episodes (36%) with a mean of 1.6 positive cultures per episode (range, 1–5). The mean duration of bacteremia was 1.2 days. Catheter tips were cultured in 26 episodes. In 17 patients *Acinetobacter* spp. were cultured from one or more blood cultures and from the catheter tip concomitantly. Antimicrobial susceptibility patterns and plasmied profiles for corresponding isolates were identical. Plasmid profiles from a blood culture and from a central venous catheter obtained from patient C during the first bacteremic episode were identical, whereas the profiles from the corresponding isolates recovered during the second bacteremic episode differed slightly (Figure 1, lanes 15 to 18). On the other hand, plasmid profiles of strains recovered from different patients were all distinct (Figure 1). No temporal or spatial relationship could be established among our patients that would be suggestive of an epidemic situation. *Acinetobacter* spp. were not isolated from any other clinical specimen submitted for culture in these patients.

Antimicrobial susceptibility testing revealed that all strains were suceptible to amoxicillin (clavulanate, the aminoglycosides, ciprofloxacin and imipenem. With the exception of *Acinetobacter* species 3 the majority of strains were also susceptible to ampicillin, broad-spectrum penicillins and third generation cephalosporins.

Clinical Features

All but three patients had fever at the time a positive blood culture as drawn. The mean temperature was 39.6°C (range, 37.9–41.0°C). The mean duration of fever was 2.3 days (range, 1–10 days); 28 patients (50.9%) experienced chills. The mean interval from admission to the hospital to the first episode of *Acinetobacter* bacteremia was 19.2 days (range, 1–85 days). At the time of bacteremia central venous catheters were in place for a mean of 9.9 days (range, 4–22 days), whereas peripheral lines were in place for a mean of 7.3 days (range, 3–19 days). The mean leukocyte count was 10.4×10^9 leukocytes/l, with leukocytosis in 19 patients and leukopenia in four patients. In all these patients, leukopenia was due to the underlying disease. Thrombocytopenia was present in 13 patients, in seven of these thrombocytopenia was due to underlying malignancy or antineoplastic chemotherapy. Clinical sepsis was evident in all but one episode, with only one patient showing signs of septic shock requiring vasopressors and intensive care management. The clinical courses were not significantly influenced by the type of *Acinetobacter* spp. involved.

Table 1: Demographic features and clinical characteristics of 53 patients with 55 episodes of bacteremia due to *Acinetobacter* species other than *Acinetobacter baumannii*.

Feature: unit of measure	Value	
Age: years		
Mean (range)	55	(3–82)
Sex: no. (%) of patients		
Male	30	(56.6)
Female	23	(43.4)
Length of hospital stay: days		
Mean (range)	43	(11–140)
Severity of underlying disease: no. (%) of patients		
Fatal or ultimately fatal within 5 years	15	(28.3)
Nonfatal	38	(71.7)
Tumor diagnosis: no. (%) of patients	11	(20.7)
Hematologic malignancy	4	(7.5)
Solid tumors	5	(9.4)
Intracerebral tumors	2	(3.8)
Vascular diseases: no. (%) of patients	38	(71.7)
Cardiovascular	24	(45.3)
Cerebrovascular	2	(3.8)
Thromboembolism	12	(22.6)
Intravascular catheters: no. (%) of episodes	51	(92.7)
Central venous catheters	22	(40.0)
Peripheral catheters	29	(52.7)
Heparin treatment: no. (%) of episodes		
Low dose s. c. heparin (≤15,000IU/day)	12	(21.8)
Low dose i. v. heparin (<20,000 IU/day)	7	(12.7)
High dose i. v. heparin (≥20,000 IU/day)	28	(50.9)
Diabetes mellitus: no. (%) of patients	13	(24.5)
Renal impairment: no. (%) of patients	5	(9.4)
Prior surgery: no. (%) of patients	12	(22.6)
Prior antimicrobial therapy: no. (%) of patients	15	(28.3)

Management of Infection and Outcome

Eighteen episodes (32.7%) of *Acinetobacter* bacteremia were considered definite catheter-related infections with only one episode showing clinical signs of insertion-site infection. Thirty-two episodes (58.2%) were considered probably catheter-related, of these five showed clinical signs of exit-site infection. One episode represented meningitis due to an indwelling ventricular catheter. Four episodes (7.3%) could not be classified.

Table 3 shows the therapeutic approach to the management of infection. Antimicrobial therapy was initiated in 44 cases (80%) and was considered appropriate according to *in vitro* results in 34 cases (61.8%). Catheter removal was documented in 45 cases (81.9%). In some patients with peripheral lines catheter removal was not properly

Table 2: Species involved in 55 episodes of *Acinetobacter* bacteremia.

Species	No. (%) of episodes	
Acinetobacter haemolyticus	3	(5.4)
Acinetobacter johnsonii	14	(25.5)
Acinetobacter junii	4	(7.3)
Acinetobacter lwoffii	10	(18.2)
Acinetobacter species 3	12	(21.8)
Acinetobacter species 6	1	(1.8)
Acinetobacter species 10	4	(7.3)
Acinetobacter species 12	4	(7.3)
Acinetobacter strains ungrouped	3	(5.5)

documented. Nine patients (16.4%) were cured with catheter removal alone without antimicrobial therapy; catheters were removed in another nine patients who were treated with antimicrobials that were not considered appropriate. Twenty-seven patients (49.1%) were treated with catheter removal and appropriate antimicrobials, in six of these patients antimicrobial therapy was initiated and intravascular lines were changed at the same time. Seven patients (12.7%) were successfully treated with appropriate antimicrobials with the catheter left in place. In the remaining three patients data on insertion and removal of intravascular access devices as well as on antimicrobial therapy in relation to outcome were inconclusive.

All but four patients were cured. Cure was achieved after a mean of 18 h after initiation of therapy (catheter removal and/or administration of appropriate antimicrobials). Four patients died 5, 7, 22 and 40 days, respectively, after *Acinetobacter* bacteremia was documented, accounting for an overall mortality rate of 7.3%. Since all patients survived at least 72 h, *Acinetobacter* bacteremia was not considered to have contributed to death in any case.

Discussion

The genus *Acinetobacter* has recently undergone significant taxonomic reorganization that allows a deeper insight into the epidemiology and clinical impact of the different members of the genus [16, 19–21]. A total of 17 DNA-hybridization groups (genospecies) were identified including the named species *A. baumannii, A. calcoaceticus, Acinetobacter haemolyticus, A. johnsonii, Acinetobacter junii,* and *A. lwoffii.* At our institute *Acinetobacter* spp. represented the second most common gram-negative pathogens isolated from blood cultures, accounting for 8.1% of all blood culture isolates during the study period; 43% of these were *Acinetobacter* spp. other than *A. baumannii* [22]. *A. baumannii* (formerly *A. calcoaceticus* var. *antitratus*) is an established agent of nosocomial pneumonia [2, 3, 23], wound infection [24], and catheter-related bacteremia [7, 25]. *Acinetobacter* species 3 (also formerly *A. calcoaceticus* var. *anitratus*) has only recently been implicated as the cause of nosocomial infections, but clinical details were not given in the report by *Dijkshoorn* et al. [26]. The other members of the genus (formerly designated *A. calcoaceticus* var. *lwoffii*) were only infrequently associated with human disease. Isolated reports have documented *Acinetobacter calcoaceticus* var. *lwoffi* as the causative agent in peritonitis [4], prosthetic valve endocarditis [27], urinary tract infections [6], and septicemia [28–30]. *Ng* et al. [29] reported an outbreak of septicemia due to *A. calcoaceticus* var. *lwoffii* in a neonatal intensive care unit. Contamination of intravenous nutritions fluids was strongly suspected but could not be proven. However, all these reports were based on the old taxonomy. Nevertheless, the ability of *Acinetobacter* spp. to contribute to a patient's morbidity and mortality has been questioned because of their apparent lack of virulence, and as part of the normal flora of human skin [31], these organisms are often dismissed as commensals or contaminants by both clinicians and microbiologists. Thus, it is currently not known which of the recently defined *Acinetobacter* spp. may be involved in human infectious diseases, nor if infections due to any of these species show characteristic clinical features or are associated with certain risk factors.

Age, sex, severity of underlying disease, antineoplastic chemotherapy, neutropenia, or immunosuppression were not considered major predisposing factors to *Acinetobacter* bacteremia for this series; only 11 patients (20.7%) were immunosuppressed by either malignancy or antineoplastic chemotherapy. Others have found *Acinetobacter* bacteremia in cancer patients and considered indwelling central venous catheters to be the major predisposing factor leading to *Acinetobacter* bacteremia [28, 30]. However, in these reports only cancer patients were studied. Interestingly, both previous studies did not find an association of bacteremia with the presence of neutropenia but with recently administered antineoplastic chemotherapy. The major predisposing factor among our patients was the presence of an intravenous line. In 51 of 55 cases (92.7%) patients had either a central venous catheter or a peripheral line. One patient each had a Broviac catheter and a hemodialysis shunt, respectively. In our series, another striking observation was the fact that more than 60% of affected patients were treated with intravenous heparin infusions via the offending catheter. We have currently no explanation for this finding and it may only be coincidental. Further studies are required to assess the role of heparin administration as a possible risk factor for the development of *Acinetobacter* bacteremia. Only 15 patients (28.3%) had received prior antibiotic therapy, and none of the patients was mechanically ventilated or treated in an intensive care unit. By contrast, the predisposing factors that have been suggested as major determinants for the development of *A. baumannii* bacteremia included treatment in an intensive care unit, prior major surgery, mechanical ventilation, hyperalimentation, prior broad-spectrum antimicrobial therapy, and intravascular catheters [7, 32].

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The clinical presentation of *Acinetobacter* bacteremia in our patients was usually benign and not significantly influenced by age, nonfatalis versus fatal or ultimately fatal underlying disease, monomicrobial or polymicrobial bacteremia. Clinical features were not helpful in distringuishing infections caused by one *Acinetobacter* spp. from those caused by another. All but one patient showed clinical signs and symptoms of sepsis, but only one patient presented with signs of septic shock. Complicated courses such as hematogenous spread to other organs were not observed. Overall mortality was low (7.3%), and no death was considered to be directly attributable to *Acinetobacter* bacteremia. Similar clinical courses of only a few days' duration, absence of cardiovascular compromise, and prompt response to catheter removal and antimicrobial therapy were also reported by *Fuchs* et al. [28] and *Rolston* et al. [30]. This favourable outcome is consistent with the observation of *Elting* and *Bodey* [33] who found that 95% of patients with central venous catheter-related infections due to *Xanthomonas maltophilia* and non-aeruginosa *Pseudomonas* spp. responded to therapy. In contrast, the overall mortality documented for *A. baumannii* bacteria in some series ranges from 22 to 43% [5, 7], and is even higher in ventilator-related nosocomial pneumonia due to *A. baumannii* [2].

Fifty of 55 episodes [90.9%) were considered definite or probable catheter-related infections. This rate is remarkably similar to the rate of 80% seen by *Rolston* et al. [30]. The number of definite device-related infections might have been even greater if a semiquantitative culture method for catheter tips had been used. Unfortunately, peripheral catheters were only infrequently submitted for culture. Two patients had two separate episodes of bacteremia due to *A. johnsonii* and *Acinetobacter* species 10, respectively. In both cases central venous catheters were removed at the time a positive blood culture was drawn but were immediately replaced. Thus, these new catheters might have been seeded during the first bacteremic episode and served as a source for the second episode of bacteremia despite appropriate antimicrobial therapy. In one patient who had an indwelling ventricular catheter after brain surgery for astrocytoma, blood culture and CSF repeatedly yielded *Acinetobacter* species 3. Clinical improvement was delayed until a 7 days' course of antimicrobial therapy with a β-lactam had been completed and the intraventricular catheter had been removed. This episode was considered to represent *Acinetobacter* meningitis.

Table 3: Approaches to the management of 55 episodes of Acinetobacter bacteremia.

Antimicrobial therapy	No. (%) of episodes Catheter removed	Catheter not removed	Total
Appropriate	27 (49.1)	7 (12.7)	34 (61.8)
Not appropriate	9 (16.4)	1 (1.8)	10 (18.2)
None	9 (16.4)	2 (3.6)	11 (20.0)
Total	45 (81.9)	10 (18.1)	55 (100)

Poor documentation of catheter insection and removal resulted in four episodes that could not be classified. Of note, there were no clinical or microbiological data suggestive of any other type of primary infection like pneumonia, urinary tract infection, wound or soft tissue infection.

All isolates were shown to be distinct by plasmid DNA analysis and there was no temporal and spatial relationship between patients. Though all but one episode were considered hospital-acquired, organisms possibly resident on human skin even before hospitalization may become adherent to indwelling plastic catheters and eventually invade the bloodstream.

In conclusion, our data suggest that most bacteremias caused by *Acinetobacter* spp. other than *A. baumannii* represent true bacteremia, are catheter-related, and respond readily to catheter removal, irrespective of the appropriateness of antibiotic therapy. Although *Acinetobacter* spp. other than *A. baumannii* similar to coagulase-negative staphylococci or non-aeruginosa pseudomonas, are often considered relatively avirulent bacteria, they may behave as opportunists in the presence of indwelling medical devices and cause invasive disease. Unlike previous reports, for patients in this series neither severity of underlying disease, immunosuppression, nor treatment in an intensiv care unit appeared to be major predisposing factors. Patient-to-patient transmission or infection from a common source was not observed. All these features demonstrate considerable differences compared to the clinical presentation and the hospital epidemiology of *A. baumannii.*

Acknowlegement

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Zusammenfassung: Bakteriämie durch *Acinetobacter* Spezies, die nicht der Spezies *Acinetobacter baumannii* zuzuordnen sind. Während eines Zeitraumes von 18 Monaten wurden in zwei universitären Krankenhäusern und acht kleineren Krankenhäusern bei 59 Patienten in 61 Fällen positive Blutkulturen mit *Acinetobacter* Spezies gefunden, die sich nicht der Spezies *Acinetobacter baumannii* zuordnen ließen. Sechs Fälle wurden als nicht signifikant eingestuft. In 50 Fällen lag eine katheterassoziierte Bakteriämie vor, in einem Fall eine Meningitis nach einem neurochirurgischen Eingriff, vier Fälle konnten nicht sicher klassifiziert werden. Die klinische Symptomatik war meist von hohem Fieber ohne ernsthafte Komplikationen gekennzeichnet. Nur vier Patienten starben während des Krankenhausaufenthaltes, in keinem Fall stand die Bakteriämie in ursächlichem Zusammenhang mit dem Tod des Patienten. Die Therapie bestand in der Entfernung des Katheters ohne antibiotische Therapie (32,8%), adäquater antibiotischer Therapie ohne (12,7%) oder gleichzeitig mit Entfernen des Katheters (49,1%). Die Plasmidanalyse aller Bakterienisolate ergab unterschiedliche Muster und damit keinen Hinweis auf einen epidemiologischen Zusammenhang zwischen den Fällen. *Acinetobacter* Spezies, die sich nicht der Spezies *A. baumannii* zuordnen lassen, sind, wie aus der vorgelegten Übersicht hervorgeht, ganz überwiegend für gutartig verlaufende katheter-assoziierte Bakteriämien verantwortlich. Ihre Pathogenität und epidemiologischen Charakteristika unterscheiden sich daher erheblich von Infektionen mit *A. baumannii.*

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Book Reciew

J. E. Banatvala (ed.)
Viral Infections of the Heart
257 pages, numerous tables and figures
Edward Arnold, London, Boston, Melbourne, Aukland 1993,
ISBN 0-340-55737-0
Price: £ 50.00

This monograph presents a scientifically-oriented summary of the state of the art in a field that has developed considerably in recent years, when molecular biology technology became accessible. The editor, an internationally renowned clinical virologist with a long-term special interest in enterovirus-produced myocarditis, has collected a number of leading theorists and clinicians with various areas of specialization in the field for the different chapters. He has indeed been successful. The contributions are of high quality, in some cases eminently so, particularly *Sally Huber's* chapter on the complicated immunological mechanisms in the pathogenesis of Coxsackie myocarditis in animal models.

Several chapters are significant from a pedagogical point of view, and are typically colored by the different cultural backgrounds of the authors. For example, the Germans – *B. Maisch, M. Herzum* and *U. Schönian* – lucidly present richly detailed material regarding the pathogenesis of viral heart diseases in humans, complementing their contribution with many tables and figures. In the reviewer's opinion, however, the chapter that deals with "The Clinical Spectrum of Viral Heart Disease" is rather insubstantial with respect to acute infectious myopericarditis. From the point of view of the infection specialist, very little has been said about various viral infectious diseases partially manifesting themselves as myopericarditis. Most likely, this is because the authors are cardiologists, whose interests naturally focus on so-called lymphocytic myocarditis, which is defined on the basis of the histophathological findings of endomyocardium biopsy and thus represents patients with myocarditis who often come for a cardiological examination because of persistent arrhythmia or cardiac insufficiency. Moreover, information on the usefulness of myocardium-specific enzymes and troponin T in diagnosing myocarditis is, unfortunately, totally missing. The kinetics of creatine kinase, isoenzyme MB, in relation to the development of ECG changes in cases of acute infectious myocarditis have, for example, been thoroughly investigated by Finnish reseachers. HIV-associated heart disease, infections of the heart in heart-transplant patients and the conceivable role of viruses in the pathogenesis of arteriosclerosis are treated in separate chapters. Repetitions, which are unavoidable in a book where each of the various chapters has been written by a different author, are not at all distracting. All of the chapters are furnished with complete lists of references. Within this rapidly developing area, monographs age quickly, and so their purpose is often questioned. If this book, however, comes into use quickly, it should have good changes of playing a useful role, especially for clinical specialists in charge of patients with myocarditis and cardiomyopathy, but also for representatives of the "service disciplines," such as clinical microbiologists, physiologists, immunologists and pathologists. Other comparable, concise presentations of such a high standard are unknown to the reviewer.

In the preface, the editor expresses the hope that this book will even be useful to general practitioners. The reviewer is more inclined to think that is should be recommended as stimulating advanced reading material for all interested physicians.

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