Anesthesiology
1998; 88:679–87

Perioperative Risk of Bradyarrhythmias in Patients with Asymptomatic Chronic Bifascicular Block or Left Bundle Branch Block

Does an Additional First-degree Atrioventricular Block Make Any Difference?

Albrecht Gauss, M.D., Christine Hübner, M.D.,* Peter Radermacher, M.D.,† Michael Georgieff, M.D.,‡ Wolfram Schütz, M.D.**

Background: **The incidence of perioperative bradyarrhythmias in patients with bifascicular or left bundle branch block (LBBB) and the influence of an additional first-degree atrioventricular (A–V) block has not been evaluated with 24-h Holter electrocardiographic monitoring. Therefore the authors assessed the rate of block progression and bradyarrhythmia in these patients.**

Methods: **Patients (n = 106) with asymptomatic bifascicular block or LBBB with or without an additional first-degree A–V block scheduled for surgery under general or regional anesthesia were enrolled prospectively. Three patients were excluded. Of the 103 remaining, 56 had a normal P–R interval and 47 had a prolonged one. Holter monitoring (CM2, CM5) was applied to each patient just before induction of anesthesia and was performed for 24 h. The primary endpoint of the study was the occurrence of block progression. As secondary endpoints, bradycardias <40 beats/min with hemodynamic compromise (systolic blood pressure <90 mmHg) or asystoles >5 s were defined.**

Results: **Block progression to second-degree A–V block and consecutive cardiac arrest occurred in one case of LBBB without a prolonged P–R interval. Severe bradyarrhythmias with hypotension developed in another eight patients: asystoles >5 s occurred in two cases and six patients had bradycardias <40/min. Pharmacotherapy was successful in these eight patients. There was no significant difference for severe bradyarrhythmias associated with hemodynamic compromise between patients with and without P–R prolongation ($P = 1.00$).**

Conclusions: **In patients with chronic bifascicular block or LBBB, perioperative progression to complete heart block is rare. However, the rate of bradyarrhythmias with hemodynamic compromise proved to be relevant. Because an additional first-degree A–V block did not increase the incidence of severe bradyarrhythmias and pharmacotherapy by itself was successful in nearly all cases, routine prophylactic insertion of a temporary pacemaker in such patients should be questioned. (Key words: Anesthesia; asystole; atrioventricular block; bifascicular block; bradycardia; bundle branch block; complete heart block; dysrhythmias; heart arrhythmias; Holter electrocardiography; pacemakers.)**

This article is featured in "This Month in Anesthesiology." Please see this issue of ANESTHESIOLOGY, page 4A.

CHRONIC bifascicular bundle branch block or left bundle branch block (LBBB) can progress to complete heart block (CHB) with the risk of sudden perioperative death.[1–3] In the perioperative period, different factors can induce a prolongation of atrioventricular (A–V) conduction and increase the risk for bradyarrhythmias.[4–8]

The rate of block progression, however, in cases of bundle branch block (BBB), seems to be low,[9] although one recent report of five patients with perioperative progression of asymptomatic bifascicular block has presented evidence to the contrary.[1] The limitations of previous studies were the retrospective design in many trials, the limited number of patients investigated, and the absence of continuous electrocardiographic (ECG) monitoring.[4]

Further, it is unknown whether an additional first-degree A–V block in patients with bifascicular block or

* Staff Anesthesiologist.

† Professor of Anesthesiology.

‡ Professor of Anesthesiology; Chairman, Department of Anesthesiology.

Received from the Department of Anesthesiology, University of Ulm, Ulm, Germany. Submitted for publication May 13, 1997. Accepted for publication October 29, 1997.

Address reprint requests to Dr. Gauss: Department of Anesthesiology, University of Ulm, Steinhoevelstr. 9, D-89075 Ulm, Germany. Address electronic mail to: albrecht.gauss@medizin.uni-ulm.de

LBBB increases the risk of block progression.[10] Finally, prophylactic insertion of a temporary pacemaker is controversial in patients with bifascicular block or LBBB: although Atlee[11] and Roizen[9] do not recommend prophylactic insertion of temporary pacing wires, other authors recommend use of a temporary perioperative pacemaker for all patients with bifascicular or LBBB and additional first-degree A-V block.[1,10,12,13]

The aim of our study was to assess prospectively the incidence of block progression and severe bradyarrhythmias in patients with bifascicular BBB and LBBB using continuous ECG (Holter) monitoring. In addition, we tested the hypothesis that a first-degree A-V block in patients with bifascicular block or LBBB increases the incidence of block progression and bradyarrhythmia.

Materials and Methods

Study Population

The study protocol was approved by the ethics committee of the University of Ulm. After obtaining written informed consent, 106 consecutive patients with asymptomatic chronic bifascicular block or LBBB with or without prolongation of P-R interval, who were scheduled to undergo surgery under general or regional anesthesia, were prospectively enrolled in the study between April 1991 and September 1994. The diagnosis of a BBB and the duration of the P-R interval were assessed by a 12-lead ECG that was obtained in median four days before operation. The patients were separated into two groups, one with normal and a second with prolonged P-R intervals. Patients with BBB and atrial fibrillation were assigned to the group with a "normal" P-R interval. If P-R prolongation with bifascicular block or LBBB was detected before operation, an overdose of digitalis glycosides was excluded by measuring serum glycoside concentration, and the necessity of a concomitant medication with beta-adrenergic blocking agents was questioned. If either of these agents was withdrawn, normalization of the P-R interval was controlled by subsequent 12-lead ECG, and patients with secondary normalized P-R interval went in the group with "normal" P-R interval. Asymptomatic BBB was defined as the absence of any signs of dizziness or syncopes.

Three patients were excluded: In one case the Holter ECG was technically inadequate, and two patients were transferred to monitored anesthesia care after the Holter ECG had been applied and thus did not receive either a general or regional anesthesia. Thus 103 patients (56 with normal and 47 with prolonged P-R intervals) met the criteria for the final evaluation.

For general anesthesia, either inhalation anesthesia (enflurane and nitrous oxide and oxygen; fraction of inspired oxygen = 0.3; induction with 5 mg/kg thiopentone or 0.3 mg/kg etomidate combined with 0.1-0.2 mg fentanyl or 0.5-1 mg alfentanil) or neuroleptanalgesia (5-10 μg/kg fentanyl and 0.1 mg/kg midazolam) was used. Succinylcholine at 1.5 mg/kg or 0.1 mg/kg vecuronium were given to facilitate tracheal intubation. Spinal anesthesia was performed with 17.5 mg bupivacaine 0.5% (isobaric solution). The median duration of surgery was 108 min (range, 10-665 min). After surgery, 51% of the patients were transferred to an intensive care unit and 49% were monitored in a postanesthesia care unit and subsequently discharged to a postsurgical ward.

The criteria to diagnose bifascicular block, BBB, hemiblock, and A-V blocks were based on the recommendations of the Criteria Committee of the New York Heart Association[14]:

Bifascicular block: right BBB with left anterior or left posterior fascicular block.

Right BBB: QRS duration $\geq$ 0.12 s and the QRS complex in V_1 has rsR' configuration or is a solitary notched R wave.

LBBB: QRS duration $\geq$ 0.12 s and the QRS complex is notched and splintered and has a QS or rS deflection in lead V_1.

Left anterior fascicular block: Frontal plane QRS axis -45 to $-90°$.

Left posterior fascicular block: Frontal plane axis is $+90$ to $+120°$.

First-degree A-V block: P-R interval >0.21 s and one-to-one A-V conduction.

Second-degree A-V block: presence of sinus rhythm. Some P waves are followed by QRS complexes. Others are not.

Second-degree A-V block, Mobitz Type I: P-R intervals of conducted beats vary according to Wenckebach periodicity.

Second-degree A-V block, Mobitz Type II: P-R intervals of conducted beats are normal or prolonged but constant.

Third-degree A-V block: atrioventricular dissociation and idioventricular rhythm.

Grading of cardiac status was based on the recommendations of the Criteria Committee of the New York Heart Association[14]:

A. No objective evidence of cardiovascular disease.
B. Objective evidence of minimal cardiovascular disease.
C. Objective evidence of moderately severe cardiovascular disease.
D. Objective evidence of severe cardiovascular disease.

A continuous ambulatory magnetic tape recorder (CardioData PaceRecorder, Northboro, MA) was applied to each patient just before induction of anesthesia. Two bipolar leads (CM2 and CM5) were recorded for 24 h. A calibration time of 8 min preceded the start of the monitoring period. The recordings were analyzed on a computer-based Holter analysis system (Cardiodata MK4). Bradycardias (<60 beats/min), pause lengths ≥1.75 R-R interval or >2 s, and rhythm and second- and third-degree A-V block were automatically identified. Tables of bradycardia (minimal, maximal, mean), pauses, rhythm, and supraventricular and ventricular dysrhythmias were obtained. After exclusion of artifacts, bradycardias <60 beats/min, pause lengths ≥1.75 R-R interval or > 2 s, rhythm abnormalities, and second- and third-degree A-V blocks were checked and then printed at 25 mm/s. Afterward, the ECG strips were reviewed again by the investigators. Pauses had to be present in both leads, and pauses after supraventricular or ventricular premature beats were excluded.

The primary endpoint of the study was the occurrence of block progression to second-degree A-V block Mobitz Type II or third-degree A-V block. As a secondary endpoint, bradycardias <40 beats/min (minimum duration ≥10 s) with hemodynamic compromise (systolic blood pressure <90 mmHg) or asystoles >5 s were defined, regardless of conduction intervals.

Fifty percent of the patients had continuous intraarterial blood pressure monitoring. With the other 50% automated noninvasive blood pressure devices (oscillometric principle; 5-min interval during operation and 30-min interval after operation) were used.

During the 24-h period, the ECGs of those patients who stayed in the intensive care unit were also monitored continuously (alarm threshold value for heart rate, 60 beats/min), and the nurses and physicians were advised to record any block progression, heart rate <40 beats/min, or decrease in systolic blood pressure <90 mmHg. If a bradycardia <40 beats/min was recognized during or after operation, the study design stipulated administration of 0.5-1 mg atropine given intravenously as the first-line treatment. For initial treatment of bradycardia <40/min combined with hypotension, 0.5-2 ml Akrinor (Asta Medica, Frankfurt, Germany) (cafedrinhydrochloride 100 mg/ml + theodrenalinehydrochloride 5 mg/ml) was administered immediately after recognition of such an episode.

Each patient was visited by the investigators once a day until discharge from the hospital to record episodes of dizziness, syncope, or hypotension in combination with heart block or bradycardia or further cardiac complications such as myocardial infarction, congestive heart failure, or cardiac arrest.

Statistical Analysis

Discrete demographic and clinical data were analyzed using the chi-squared test. Differences in age between the groups were analyzed using the Mann-Whitney U test. The hypothesis that there are no differences in the incidence of severe bradyarrhythmias between the groups with normal *versus* prolonged P-R interval was tested using the Fisher's exact test. A P value <0.05 was considered significant.

Results

Demographic and clinical data for the 103 patients are presented in table 1. There were no significant differences in age and American Society of Anesthesiologists physical status between the group with and without first-degree A-V block. The patients in the group with prolonged P-R interval suffered more often from moderately severe or severe cardiovascular disease.[14] Table 2 shows the distribution of the conduction abnormalities.

Block Progression

Two cases of second-degree A-V block Mobitz Type I without hemodynamic impairment were observed (in one case during operation, in the other case 2 h after operation); each of these patients had right BBB and left anterior fascicular block with normal P-R interval.

One case of block progression to second-degree A-V block and consecutive cardiac arrest occurred (for details see under case reports).

Severe Bradyarrhythmias with Hemodynamic Depression

Severe bradyarrhythmias with hemodynamic depression during the perioperative period developed in nine patients (9%; table 3): asystoles >5 s occurred in three

Table 1. Demographic and Clinical Data (103 Patients)

	BBB and Normal P-R Interval	BBB and Prolonged P-R Interval
n	56	47
Gender (F/M) (%)	41/59	21/79*
Age (yr)	71 (37–89)	73 (51–95)
ASA Physical Status (%)		
III	95	89
IV	5	11
Hypertension (%)	48	43
Heart disease (%)	20	43*
Congestive heart failure	7	21
CAD	16	28
Valvular heart disease	5	6
Preoperative medication (%)		
Digitalis	27	36
β-Adrenergic blockers	9	4
Calcium channel blockers	4	6
Type of anesthesia (%)		
General anesthesia	66	62
Regional anesthesia†	32	32
General and regional	2	6

Median values (ranges in parentheses).

CAD (coronary artery disease) = typical angina pectoris or previous myocardial infarction or angiographically assessed significant stenosis of coronary arteries.

* Significant difference between the groups ($P < 0.05$).

† Spinal and/or epidural anesthesia.

cases (3%, including the patient with second-degree A-V block and consecutive cardiac arrest), and bradycardias <40 beats/min combined with arterial hypotension occurred in six patients (6%). Therapeutic interventions were necessary in all of these patients. In eight of nine cases, atropine and Akrinor were sufficient to treat these complications, and in one case cardiac massage was necessary. None of the patients died in the perioperative period.

Table 2. Conduction Abnormality Data

	Normal P-R Interval (n = 56)	Prolonged P-R Interval (n = 47)	Total (n = 103)
Bifascicular block			
RBBB + LAFB	23 (41)	20 (43)	43 (42)
RBBB + LPFB	3 (5)	0	3 (3)
LBBB	30 (54)	27 (57)	57 (55)

Values are no. (%).

BBB = bundle branch block; RBBB = right bundle branch block; LBBB = left bundle branch block; LAFB = left anterior fascicular block; LPFB = left posterior fascicular block.

Table 3. Severe Bradyarrhythmias Combined with Hemodynamic Depression in 103 Patients with BBB and Normal or Prolonged P-R Interval

	BBB Normal P-R Interval (n = 56)	BBB Prolonged P-R Interval (n = 47)	Total (n = 103)
Asystoles* >5 s	3	0	3 (3%)
Heart rate <40 beats/min combined with SAP <90 mmHg	2 (i.o.)	4 (i.o.)	6 (6%)
Total	5	4	9 (9%)

BBB = bundle branch block; i.o. = intraoperative; p.o. = postoperative; SAP = systolic blood pressure.

* The asystoles occurred in one case after induction of anesthesia, in a second case intraoperatively, and in a third case on the third postoperative day.

Bradycardias

Bradycardias <40 beats/min occurred in 17% of patients (in 10 cases during operation, in 7 cases afterward [in the 24-h period], and in 1 case during and after operation). During spinal anesthesia, bradycardia <40 beats/min was found in 10 of 25 patients (40%).

Comparison between Patients with Bundle Branch Block with Normal and Prolonged P-R Interval

In one patient only (LBBB without P-R prolongation) progression to CHB occurred.

There was no significant intergroup difference for severe bradyarrhythmias associated with hemodynamic depression ($P = 1.00$; table 3).

Preexisting Cardiovascular Disease and Type of Bundle Branch Block

The three patients with periods of serious asystole >5 s had preexisting LBBB and moderately severe or severe cardiovascular disease.[14]

Patients with Severe Bradyarrhythmias and Hemodynamic Compromise

Tables 4 and 5 present detailed data on the nine patients with severe bradyarrhythmias. Eight of these patients were in sinus rhythm. In seven of them, Holter ECG monitoring showed no perioperative sick sinus syndrome. In one patient only (patient 4, table 5) who had atrial tachyarrhythmia (135 beats/min) 10 h after operation, sick sinus syndrome could not be excluded; however, episodes of sinus arrest could not be recorded in this case.

Table 4. Summary of Severe Bradyarrhythmias Combined with Hemodynamic Depression in Patients with BBB and Normal P-R Interval

Patient No.	Rhythm/Type of BBB	Heart Disease*	Preoperative Medication	Type of Surgery	Type of Anesthesia	Severe Bradyarrhythmias	Therapy
1	SR/RBBB + LAFB	CAD	Nitrate	TURP	SPA	Sinus bradycardia (i.o.)	Atropine
2	Atrial fibrillation/LBBB	CHF	Nitrate Digitoxin	CE	GA	Asystole (30 s) (i.o.)	Atropine/Akrinor
3	SR/LBBB	CAD	Nitrate β blocker Clonidine	Nephrectomy	GA + EDA	A-V block II° → asystole (60 s) (3rd day, p.o.)	Atropine/CPR
4	SR/RBBB + LAFB	—	Propafenone	Herniotomy	SPA + EDA	Bradycardia (i.o.)	Atropine/Akrinor
5	SR/LBBB	CHF	Nifedipine	Abdominal aneurysm	GA	Asystole (10 s) (after induction of anesthesia)	Epinephrine

Akrinor = cafedrinydrochloride + theodrenalinehydrochloride; BBB = bundle branch block; CAD = coronary artery disease; CE = cataract extraction; CHF = congestive heart failure; CPR = cardiopulmonary resuscitation; EDA = epidural anesthesia; GA = general anesthesia; i.o. = intraoperative; LAFB = left anterior fascicular block; LBBB = left bundle branch block; p.o. = postoperative; RBBB = right bundle branch block; SPA = spinal anesthesia; SR = sinus rhythm; TURP = transurethral resection of the prostate.

* Moderately severe or severe cardiovascular disease.[14]

In one patient (patient 3, table 5) taking digoxin the serum digoxin concentration was within the upper range of normal, and the cardiologist recommended withdrawal of digoxin. Three weeks later, the ECG showed no change in the P-R interval, and the patient had surgery.

Case Reports of the Three Most Severe Episodes of Asystole (>5 s)

Patient 2 (table 4), a woman aged 78 yr with moderately reduced left ventricular function and pulmonary arterial hypertension, sustained an asystole during ophthalmic surgery. Because her preoperative heart rate had been 90 beats/min and she had taken 0.07 digitoxin mg/day only, the serum digoxin concentration had not been measured. Pressure on the eyeball had probably induced oculocardiac reflex. Stopping stimulation by the surgeon and injection of atropine and Akrinor were successful. The postoperative course of patient 3 (table 4), a woman aged 69 yr, was more complicated. Nephrectomy was planned because of hypernephroma.

Table 5. Summary of Severe Bradyarrhythmias Combined with Hemodynamic Depression in Patients with BBB and Prolonged P-R Interval

Patient No.	Rhythm/Type of BBB	Heart Disease*	Preoperative Medication	Type of Surgery	Type of Anesthesia	Severe Bradyarrhythmias	Therapy
1	SR/LBBB	CAD	Digitoxin (20 μg/l)† ACEI	CE	GA	Sinus bradycardia (i.o.)/asystole (3 s) (7 h p.o.)	Akrinor
2	SR/LBBB	CAD/MR	Nitrate Digoxin (<0.5 μg/l)† ACEI	CE	GA	Sinus bradycardia (i.o.)	Akrinor
3	SR/LBBB	—	Digoxin (2.0 μg/l)† Nifedipine	Prostatectomy	SPA + EDA	Bradycardia (i.o.)	Atropine/akrinor
4	SR/RBBB + LAFB	—	—	Gastrectomy	GA + EDA	Sinus bradycardia (i.o.)	Akrinor

ACEI = angiotensin-converting enzyme inhibitors; Akrinor = cafedrinhydrochloride + theodrenalinehydrochloride; BBB = bundle branch block; CAD = coronary artery disease; CE = cataract extraction; EDA = epidural anesthesia; GA = general anesthesia; i.o. = intraoperative; LAFB = left anterior fascicular block; LBBB = left bundle branch block; MR = mitral regurgitation; p.o. = postoperative; RBBB = right bundle branch block; SPA = spinal anesthesia; SR = sinus rhythm.

* Moderately severe or severe cardiovascular disease.[14]

† Serum drug concentrations of digitoxin (normal range 7.5–25 μg/l) and digoxin (normal range 0.5–2.2 μg/l) are presented.

When she was admitted, she had LBBB and a first-degree A-V block (P-R interval 0.235 s) and taking nitrate (on demand), clonidine and β-adrenergic blocker (100 mg/day metoprolol) because of hypertension and coronary artery disease. The consulting cardiologist recommended continuous nitrate medication and withdrawal of metoprolol. Four days later, the P-R interval had shortened to 0.17 s, and she was scheduled for operation. Before induction of anesthesia, a sinus tachycardia (113 beats/min) was conspicuous. The course of the anesthesia was stable, and her heart rate decreased to 80-90 beats/min. As a result of sinus tachycardia, clonidine and the β-adrenergic blocker were administered again on the second postoperative day. On the third postoperative day, her heart rate decreased suddenly during an infusion of neostigmine for stimulation of bowel function, a second-degree A-V block appeared on the monitor, and an asystole over 60 s followed, which was treated successfully with atropine, mask ventilation, and cardiac massage. An echocardiographic investigation revealed new wall motion abnormalities (akinesia) at the apex of the left anterior wall. The ECG showed increased S-T segment elevation in V_{4-6}. The maximum values of the enzymes were as follows: serum glutamate oxaloacetate transaminase, 133 U/l; creatine kinase, 810 U/l; and creatine kinase-MB, 40 U/l. These results led to the diagnosis of perioperative myocardial infarction. The asystole was thought to be caused by the combination of drugs with bradycardic effects.

Patient 5 (table 4), a man aged 67 yr with dilated cardiomyopathy, developed asystole after induction of anesthesia, during catheterization of the internal jugular vein and inadvertent puncture of the carotid artery. Carotid sinus stimulation by digital pressure probably caused this complication; the guidewire had not been inserted at this time. Asystole reversed immediately after pressure release and administration of 40 μg epinephrine. The following course was uncomplicated.

Discussion

To our knowledge this is the first prospective study using continuous 24 h (Holter) monitoring to investigate the incidence of block progression and bradyarrhythmias in patients with bifascicular or LBBB. The first major finding of our study is that block progression to CHB is a rare perioperative complication because only one block progression to second-degree A-V block and consecutive cardiac arrest occurred. In a further case with asystole during ophthalmic surgery, the exact cause of the asystole is not clear because the patient presented with LBBB and atrial fibrillation before operation.

Regardless of anesthesia and surgery, the cumulative incidence of CHB in patients with "chronic bifascicular and trifascicular block" at 5 yr is low, at 4.9% and 11.3%, in two prospective studies.[15,16] Anesthesia and surgery might be expected to favor severe dysrhythmias in patients with preexisting BBB: Anesthetics, combinations of antiarrhythmic agents with anesthetics, regional anesthesia, disturbances of blood gases or electrolytes, endotracheal intubation, central venous or pulmonary catheterization, surgical manipulation, hypothermia, or myocardial ischemia or infarction can induce conduction defects and bradyarrhythmias.[4-8] The results of nine studies, however, in 341 patients with asymptomatic bifascicular block or LBBB who received general or regional anesthesia indicated an incidence of serious block progression $<1\%$.[2,3,17-23] Venkataraman *et al.*[3] found one patient with preoperative second-degree A-V block Mobitz Type II in whom "trifascicular block" developed in the recovery room, and Pastore *et al.*[2] observed another one with CHB during intubation. Although many of these patients were investigated retrospectively and none of them was monitored with Holter monitoring over 24 h, our prospective findings confirm these results.

Nonetheless, a report that during a 1-yr observation period perioperative progression of asymptomatic bifascicular block to CHB developed in five patients suggests the potential importance of this complication.[1] In any case, it is necessary to take a detailed medical history to exclude symptomatic BBB because Santini *et al.*[24] observed CHB in 2 of 18 patients with symptomatic LBBB or bifascicular block after succinylcholine. Complete heart block was resistant to 1 mg atropine and only resolved after administration of orciprenaline.[24]

The incidence of bradyarrhythmias with hemodynamic depression in our patients was high. The comparison of our results with previous studies is limited because sinus bradycardia is often not exactly defined and information is missing about whether dysrhythmias led to hemodynamic impairment. Goldman *et al.*[25] described bradyarrhythmias in 10% of 1,001 patients during surgery and early recovery. Carpenter *et al.*[26] prospectively studied 952 patients during spinal anesthesia and identified bradycardia (<50 beats/min) in 13%, a rather low level when compared with the 40% incidence of bradycardia <40/min in our patients receiving

spinal anesthesia. Further, in contrast to our two cases of intraoperative asystole, these authors did not observe any asystole. Even if Holter monitoring increased the detection frequency of bradyarrhythmias, the need for therapy in 9% of our patients emphasizes their importance.

Comparison between Patients with Bifascicular Block or Left Bundle Branch Block with Normal and Prolonged P-R Interval

The second important finding of our trial is the fact that we could not find any difference between patients with normal and prolonged P-R interval regarding the occurrence of block progression or severe bradyarrhythmia. In particular, the most critical bradyarrhythmias (asystoles) developed in the group with normal P-R interval.

In patients with LBBB or bifascicular block, an additional A-V conduction disorder in the last conducting fascicle may be precipitated by anesthesia and surgery and thereby degenerate to CHB. A first-degree A-V block in the surface ECG could indicate an increased risk for this disorder. To determine whether the A-V conduction disturbance is localized in the A-V node (possibly a more benign disorder) or whether it is distal (implying incomplete fascicular block in the last remaining fascicle),[27] it would be necessary to study all patients with BBB and additional first-degree A-V block with invasive His bundle ECG before operation, which is not feasible. Usually, a first-degree A-V block is caused by dysfunction within the A-V node,[28] detectable as a prolongation of the A-H interval.[29] A first-degree heart block in patients with right BBB and left axis deviation, however, suggested abnormality of the H-V interval,[30] and Rosen *et al.*[31] reported that patients with LBBB and P-R interval prolongation frequently had H-Q interval prolongation.

Long-term investigations of patients with BBB showed different results about whether CHB develops more often in patients with additional prolonged H-V interval.[15,28,32,33] For medical patients, the Task Force of the American College of Cardiology/American Heart Association (ACC/AHA) identified P-R and H-V intervals as possible predictors of CHB and sudden death.[34] However, the major determinant of sudden death in these patients was their underlying cardiac disease rather than the BBB.[15]

With respect to general anesthesia in 98 patients with bifascicular block, there was no difference in bradyarrhythmias between patients with normal and prolonged (>75 ms) H-V interval.[20] There was a significantly higher incidence, however, of general cardiac complications in patients with marked H-V-prolongation (>75 ms).[20]

Circumstances that Can Influence the Incidence and Course of Perioperative Bradyarrhythmias

All three patients with severe asystoles >5 s had preexisting moderately severe or severe cardiovascular disease and LBBB. Although the various types of BBB were not predictive for CHB in the study by McAnulty *et al.*,[15] particularly LBBB combined with coronary artery disease, seems to increase the risk of death regardless of the extent of left ventricular dysfunction and coronary artery disease.[35]

With each of the three patients, a second additional factor may have contributed to the prolonged asystole: ophthalmic surgery and probably oculocardiac reflex in patient 2 (table 4), myocardial infarction and combination of specific drugs in patient 3 (table 4), and carotid sinus pressure in patient 5 (table 4). In patient 5, we cannot exclude preexisting hypersensitive carotid sinus syndrome, because ventricular asystole for 3 s or more is an abnormal response after carotid massage. The immediate therapeutic interventions were successful in every case, and no patient showed an adverse outcome.

Thus in patients with underlying conduction abnormalities, the following concomitant circumstances are prone to perioperative bradyarrhythmias: acute myocardial infarction, preexisting severe cardiovascular disease combined with especially LBBB, and pulmonary artery catheterization in patients with LBBB.[36]

Vagal stimulation and combination of bradycardic drugs should be avoided.

Atropine, epinephrine, and isoprenaline should be prepared before induction of anesthesia in any case and temporary pacemaker equipment must be readily available.

Study Limitations

Our study has some limitations. We did not perform His bundle ECG before operation, and we did not follow our patients with serial ECGs beginning after the first 24 h after operation. Adverse episodes could have been missed because 49% of the patients did not go to the intensive care unit. On the other hand, hypotension could have been caused by various reasons unrelated to conduction abnormalities, and thus the incidence could have been overestimated.

In the eight patients with bradyarrhythmias without

block progression, we cannot with certainty attribute these episodes to the presence of BBB *per se*.

Because a clinically significant block progression was detected in one case only (LBBB with normal P-R interval), despite Holter monitoring, this trial was not adequately powered to determine whether first-degree A-V block increases the risk of CHB. Such a study would require a substantially larger sample size. Nonetheless, any effect of a first-degree A-V block seems to be minimal.

In conclusion, serious block progression to CHB occurred in only one patient with asymptomatic chronic bifascicular block or LBBB. This was precipitated by a combination of β-adrenergic blocker and anticholinergic agent. Severe bradyarrhythmias with hemodynamic depression developed in another eight patients. Pharmacotherapy was successful in these latter cases. There was no difference between patients with and without P-R prolongation either in the occurrence of block progression or that of severe bradyarrhythmias. Therefore we believe that administration of a temporary pacemaker is not mandatory in patients with asymptomatic bifascicular block or LBBB, even if first-degree A-V block is present. Temporary pacemaker equipment, however, should be readily available in case antiarrhythmic therapy fails.

The authors thank Lutz Binner, M.D., and Veit Goeller, M.D., of the Department of Medicine II (Cardiology), University of Ulm, for help in interpreting difficult ECG findings, Irmgard Doll for technical assistance with ECG Holter monitoring, and Rainer Muche, Ph.D., Department of Biometry and Documentation, University of Ulm, for suggestions regarding the statistical analysis.

References

1. Boezaart AP, Clinton CW, Stanley A: Pre-operative prophylactic transvenous cardiac pacing for bifascicular heart block. S Afr J Surg 1989; 27:103-5

2. Pastore JO, Yurchak PM, Janis KM, Murphy JD, Zir LM: The risk of advanced heart block in surgical patients with right bundle branch block and left axis deviation. Circulation 1978; 57:677-80

3. Venkataraman K, Madias JE, Hood WB: Indications for prophylactic preoperative insertion of pacemakers in patients with right bundle branch block and left anterior hemiblock. Chest 1975; 68:501-6

4. Atlee JL III, Bosnjak ZJ: Mechanisms for cardiac dysrhythmias during anesthesia. ANESTHESIOLOGY 1990; 72:347-74

5. Doyle DJ, Mark PWS: Reflex bradycardia during surgery. Can J Anaesth 1990; 37:219-22

6. Doyle DJ, Knapp CD: Asystole from unintended myocardial hypothermia. ANESTHESIOLOGY 1994; 80:956

7. Park KW, Lowenstein E: Acute complete heart block during anesthesia in a patient with severe coronary artery disease: Effect of scopolamine versus ischemia of the AV node. ANESTHESIOLOGY 1991; 75:367-70

8. Caplan RA, Ward RJ, Posner K, Cheney FW: Unexpected cardiac arrest during spinal anesthesia: A closed claims analysis of predisposing factors. ANESTHESIOLOGY 1988; 68:5-11

9. Roizen MF: Anesthetic implications of concurrent diseases, Anesthesia, 4th ed. Edited by RD Miller. New York, Churchill Livingstone, 1994, pp 903-1014

10. Zaidan JR: Electrocardiography, Clinical Anesthesia, 2nd ed. Edited by PG Barash, BF Cullen, RK Stoelting. Philadelphia, JB Lippincott, 1992, pp 771-805

11. Atlee JL: Temporary perioperative pacing, Perioperative Management of Pacemaker Patients, 1st ed. Edited by JL Atlee, H Gombotz, KH Tscheliessnigg. Berlin, Springer, 1992, pp 127-37

12. Ford BM, Weich HFH, Coetzee AR: Pre-operative assessment of cardiac patients for non-cardiac surgery. S Afr Med J 1984; 65: 235-9

13. Heinecker R, Gonska B-D: EKG in Klinik und Praxis, 13th ed. Stuttgart, Thieme, 1992, pp 153-60

14. The Criteria Committee of the New York Heart Association: Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels, 9th ed. Boston. Little, Brown and Company, 1994, pp 174-255

15. McAnulty JH, Rahimtoola SH, Murphy E, DeMots H, Ritzmann L, Kanarek PE, Kauffman S: Natural history of "high-risk" bundle-branch block. Final report of a prospective study. N Engl J Med 1982; 307:137-43

16. Dhingra RC, Wyndham C, Amat-y-Leon F, Denes P, Wu D, Sridhar S, Bustin AG, Rosen KM: Incidence and site of atrioventricular block in patients with chronic bifascicular block. Circulation 1979; 59:238-46

17. Berg GR, Kotler MN: The significance of bilateral bundle branch block in the preoperative patient. Chest 1971; 59:62-7

18. Kunstadt D, Punja M, Cagin N, Fernandez P, Levitt B, Yuceoglu YZ: Bifascicular block: A clinical and electrophysiologic study. Am Heart J 1973; 86:173-81

19. Rooney S-M, Goldiner PL, Muss E: Relationship of right bundle-branch block and marked left axis deviation to complete heart block during general anesthesia. ANESTHESIOLOGY 1976; 44:64-6

20. Bellocci F, Santarelli P, DiGennaro M, Ansalone G, Fenici R: The risk of cardiac complications in surgical patients with bifascicular block. Chest 1980; 77:343-8

21. Mikell FL, Weir EK, Chesler E: Perioperative risk of complete heart block in patients with bifascicular block and prolonged PR interval. Thorax 1981; 36:14-7

22. Coriat P, Harari A, Ducardonet A, Tarot J-P, Viars P: Risk of advanced heart block during extradural anaesthesia in patients with right bundle branch block and left anterior hemiblock. Br J Anaesth 1981; 53:545-8

23. Eledjam JJ, de la Coussaye JE, Colson P, Viel E, Bassoul B, Bertinchant JP, D'Athis F: Is epidural anaesthesia using bupivacaine safe in patients with atrio-ventricular conduction defects? Acta Anaesthesiol Scand 1989; 33:402-4

24. Santini M, Carrara P, Benhar M, Piovano G, Rocchi M, Di Mascolo R, Masini V: Possible risks of general anesthesia in patients with intraventricular conduction disturbances. Pace 1980; 3:130-7

25. Goldman L, Caldera DL, Southwick FS, Nussbaum SR, Murray B, O'Malley TA, Goroll AH, Caplan CH, Nolan J, Burke DS, Krogstad

D, Carabello B, Slater EE: Cardiac risk factors and complications in non-cardiac surgery. Medicine 1978; 57:357–70

26. Carpenter RL, Caplan RA, Brown DL, Stephenson C, Wu R: Incidence and risk factors for side effects of spinal anesthesia. ANESTHESIOLOGY 1992; 76:906–16

27. Vera Z, Mason DT, Fletcher RD, Awan NA, Massumi RA: Prolonged His-Q interval in chronic bifascicular block. Relation to impending complete heart block. Circulation 1976; 53:46–55

28. Scheinman MM, Peters RW, Modin G, Brennan M, Mies C, O'Young J: Prognostic value of infranodal conduction time in patients with chronic bundle branch block. Circulation 1977; 56:240–4

29. Dhingra RC, Wyndham C, Amat-y-Leon F, Wu D, Denes P, Towne WD, Rosen KM: Significance of A–H interval in patients with chronic bundle branch block. Am J Cardiol 1976; 37:231–6

30. Levites R, Haft JI: Significance of first degree heart block (prolonged P–R interval) in bifascicular block. Am J Cardiol 1974; 34:259–64

31. Rosen KM, Rahimtoola SH, Chuquimia R, Loeb HS, Gunnar RM: Electrophysiological significance of first degree atrioventricular block with intraventricular conduction disturbance. Circulation 1971; 43:491–502

32. Flowers NC: Left bundle branch block: A continuously evolving concept. J Am Coll Cardiol 1987; 9:684–97

33. Scheinman MM, Peters RW, Morady F, Sauvé MJ, Malone P, Modin G: Electrophysiologic studies in patients with bundle branch block. Pace 1983; 6:1157–65

34. Dreifus LS, Fisch C, Griffin JC, Gillette PC, Mason JW, Parsonnet V: Guidelines for implantation of cardiac pacemakers and antiarrhythmia devices. J Am Coll Cardiol 1991; 18:1–13

35. Freedman RA, Alderman EL, Sheffield LT, Saporito M, Fisher LD: Bundle branch block in patients with chronic coronary artery disease: Angiographic correlates and prognostic significance. J Am Coll Cardiol 1987; 10:73–80

36. Risk SC, Brandon D, D'Ambra MN, Koski EG, Hoffman WJ, Philbin DM: Indications for the use of pacing pulmonary artery catheters in cardiac surgery. J Cardiothorac Vasc Anesth 1992; 6:275–9