Cardiac response and complications during endovascular repair of abdominal aortic aneurysms: A concurrent comparison with open surgery

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Purpose: The purpose of this study was to assess and to compare perioperative changes in left ventricular function and the incidence of adverse cardiac events in two groups of patients with abdominal aortic aneurysms, one during endovascular aneurysm repair (EAR) and the other during open aneurysm repair (OAR).
Methods: One hundred twenty consecutive patients who underwent EAR (49 patients) or OAR (71 patients) were prospectively studied. During the operation, the left ventricular function was assessed by the recording of the left ventricle stroke work index (SWI) and the cardiac index (CI) with a pulmonary artery catheter. Measurements were performed before, during, and after stent-graft deployment or aortic cross-clamping. Both maneuvers were defined as aortic occlusion (AO). Transesophageal echocardiography was performed to identify signs of wall motion abnormalities of the left ventricular wall, which indicated myocardial ischemia. Six-lead electrocardiograph monitoring was maintained until discharge from the intensive care unit. Postoperative cardiac complications were diagnosed by clinical observation, 12-lead ECG analysis at 1, 3, and 7 days after the operation, transthoracic echocardiography at 1 month, and measurement of cardiac enzymes.
Results: The two study groups were comparable with regard to most clinical aspects. The baseline myocardial performance was worse in patients who underwent EAR compared with patients who underwent OAR, as indicated by a reduced SWI (33.1 and 37.4, respectively; *P* = .03). During AO there was a comparable increase of the CI in both groups. However, after AO the rise in CI was higher in patients who underwent OAR compared with patients who underwent EAR (0.7 and 0.2, respectively; *P* < .01), representing a more pronounced hyperdynamic state. In addition, the SWI demonstrated a decrease in patients who underwent OAR compared with an increase in patients who underwent EAR during AO (−1.4 and +1.9, respectively; *P* = .04) and after AO (−0.9 and +2.6, respectively; *P* = .01). These findings represent more severe myocardial stress in patients who underwent OAR. The incidence of postoperative clinical cardiac adverse events was comparable in the two study groups. However, myocardial ischemia, as indicated by electrocardiography and transesophageal echocardiography, had a higher incidence in patients who underwent open surgery as compared with patients whose condition was managed endovascularly (57% and 33%, respectively; *P* = .01).
Conclusion: Hemodynamic alterations during endovascular repair were not as severe as those in patients with open surgery and indicated less myocardial stress in the former category. These findings may explain a lower incidence of myocardial ischemia that was observed during endovascular repair. A lower frequency of clinical perioperative cardiac events in patients undergoing endovascular treatment may ultimately be expected. (J Vasc Surg 2001;33:353-60.)

Endovascular repair of abdominal aortic aneurysms (AAAs) is currently being evaluated as an alternative to open aneurysm repair (OAR). Several reports have established the feasibility and potentially low operative risks of this less invasive technique.[1-4] Indeed, one of the principal challenges for stent-graft treatment will be to demonstrate that it is associated with a significantly lower operative mor-

tality rate. OAR is associated with a mortality rate of approximately 4% in institutional series and 6% to 10% in regional or national studies.[5-8] Coronary artery disease (CAD), the most prominent risk factor, is responsible for up to 60% of perioperative deaths after OAR.[6,7,9] Adverse cardiac events have been attributed to excessive stress on the myocardium caused by the combined effect of anesthetic induction, aortic clamping and declamping, operative blood loss, and associated hemodynamic and metabolic changes.[10,11] It is likely that hemodynamic alterations associated with endovascular aneurysm repair (EAR) are less severe. If this assumption is true, myocardial stress will be reduced, and the risk of cardiac complications will be minimized. At the present time, no comprehensive studies are available on hemodynamics and left ventricular response during endovascular AAA treatment. Neither have proper comparisons with open AAA repair been performed for clinical cardiac outcome events and findings at hemodynamic and cardiac examinations. Apart from electrocardiographic analysis, left ventricular wall imaging has

Table I. Patient characteristics in the two study groups

| | Study group | | | |
| | EAR* (n = 49) | | OAR† (n = 71) | |
Characteristic	n	%	n	%
Sex (F/M)	2/47	—	10/61	—
ASA classification[16]				
1	7	14	10	14
2	20	41	42	59
3	15	31	14	20
4	7	14	5	7
History of myocardial infarction	15	31	21	30
Previous PTCA	7	14	14	20
Previous coronary artery bypass graft	11	22	19	27
History of CAD	22	45	36	51
Preoperative b-blocker	16	33	29	41
Hypertension‡	23	47	40	56
Smoking‡	21	43	27	38
Diabetes‡	3	6	5	7
COPD‡	17	35	17	24

PTCA, Percutaneous transluminal coronary angioplasty; *COPD*, chronic obstructive pulmonary disease.
*Average age, 70 years (range, 66-74 years); *P* < .01; maximum AAA diameter, 55 mm (range, 50-60 mm).
†Average age, 73 years (range, 69-77 years); *P* < .01; maximum AAA diameter, 53 mm (range, 50-64 mm).
‡Patients with a Society of Vascular Surgery/International Society for Cardiovascular Surgery risk score of more than 0.[17]

been shown to provide accurate predictors of myocardial ischemia and cardiac complications.[12-14] These findings may be considered as surrogate parameters for adverse events that enable meaningful conclusions on the basis of considerably smaller numbers of patients than would be required if only clinical outcome is studied.

At the present time, there is hardly any objective justification to recommend either EAR or OAR to most patients as the preferred treatment. Therefore, the bearings of a comprehensive comparison of cardiac events that are associated with the different treatments may be considerable, because answers to fundamental questions may come at hand. The aim of the present prospective study is to assess and to compare the perioperative changes in left ventricular function and the incidence of adverse cardiac events in two groups of patients with AAA, one group during EAR and the other group during OAR. This study was not randomized, and the medical condition of the patient was a consideration in treatment selection. Therefore, as a second objective, the influence of any differences in preexisting patient characteristics between the study groups, possibly because of patient selection, was assessed.

PATIENTS AND METHODS

Between May 1996 and August 1998, 135 consecutive patients with AAA underwent repair. Fifteen patients were ineligible for investigation because the hemodynamic measurements were unreliable due to pulmonary artery catheter malposition (6 of these patients underwent OAR; 9 of these patients underwent EAR). Thus 120 patients were available for analysis, of whom 71 patients underwent OAR and 49 patients underwent EAR. Patient selection was based on the following considerations: OAR was performed in 58 patients who had an adverse anatomy for endograft treatment[15] and in two patients with a contrast allergy. In addition, 11 patients with a suitable anatomy for EAR were included in a substudy that required randomization between OAR and EAR and were allocated to OAR. In the OAR group, 56 patients received a tube graft, and 15 patients received a bifurcated graft; in the EAR group, all patients received a bifurcated device. In the EAR group, the Stentor system (Mintec, Inc, La Ciotat, France) was used in three patients, the Vanguard system (Boston Scientific, Oakland, NJ) was used in 28 patients, and the AneuRx system (Medtronic, Inc, Sunnyvale, Calif) was used in 18 patients. Patient characteristics in the two treatment groups are summarized in Table I. The study groups were comparable with regard to most, but not all, clinical aspects. Most notably, patients who underwent OAR were older than those who underwent EAR. A substantial number of patients in both groups had had clinically evident manifestations of CAD. Three patients in the EAR group were considered unfit for OAR.

Before the operation, all patients underwent a comprehensive examination by the cardiologists involved in this study (M.G., C.P.). This examination was according to a fixed protocol and consisted of a physical examination, a standard 12-lead electrocardiogram, and a dobutamine (Dobutrex) stress-echocardiogram (for this analysis, a wall motion score was calculated). The left ventricle was divided into 13 segments, and the segmental wall motion was graded on a 5-point scale, varying from –1 for hyperkinesia, to 0 for normokinesia, 1 for hypokinesia, 2 for akinesia, and 3 for dyskinesia.[18] The wall motion score was the sum of the segmental wall motion scores. The baseline wall motion score of the left ventricle (ie, before dobutamine

stress) was comparable between the two groups (median, 2 [EAR] vs 0 [OAR]; *P* = .5). The occurrence of wall motion abnormalities (WMAs) at the time of dobutamine stress was also comparable (16% [EAR] vs 8.5% [OAR]; *P* = .3).

In this study clinicians were not blinded for the results of the testing. The preoperative tests resulted in therapeutic measures in several instances, including a change of preoperative medication in 25 patients and supplementary intraoperative medication in 47 patients. Overall, 59 patients had a change of medication, with an uneven distribution between the two study groups (20 patients [41%] with EAR vs 39 patients [55%] with OAR; *P* = .03). A coronary angiogram was performed when severe CAD was suspected, which resulted in a coronary angioplasty in three patients and a coronary bypass graft in four patients before aneurysm repair. All seven patients with coronary interventions underwent OAR. The study protocol was approved by the Hospital Review Committee.

Operative procedures. Anesthesia was induced with etomidate, pancuronium bromide (Pavulon), and fentanyl. Anesthesia was maintained with nitrous oxide and oxygen gas mixtures and fentanyl and pancuronium as needed. General endotracheal anesthesia was used in all patients. The EAR procedures were performed according to previously described guidelines.[15]

Operative and postoperative measurements. Standard intraoperative monitoring included a radial artery cannula to continuously record blood pressure (BP) and heart rate, and a I-II-V5/6 ST-segment electrocardiogram analysis. Monitoring was continued throughout the period that the patient remained in the intensive care unit. A balloon-tipped pulmonary artery catheter was inserted in the pulmonary artery by standard techniques[19] to record pulmonary wedge pressure (PWP), systemic vascular resistance (SVR), stroke volume (SV), cardiac index (CI), and the left ventricle stroke work index (SWI). All intraoperative measurements were performed at three points of time: before (baseline), during, and after aortic occlusion. During EAR, inflation of the latex balloon within the aorta (Stentor or Vanguard system) or deployment of the aortic part of the device until the point that the aorta was occluded (AneuRx system) was considered as aortic occlusion. In addition, during the same three consecutive periods, myocardial performance was assessed by transesophageal echocardiography (TEE) to record any signs of WMAs of the left ventricular wall. TEE-images were stored on a videotape and reviewed off-line by a second cardiologist, who was blinded for the type of operation performed.

All patients were admitted at the intensive care unit for continued monitoring at least until the next day. A 12-lead electrocardiogram-analysis was obtained at 1 hour, 1 day, and 1 week after operation. Blood samples for measuring cardiac enzymes (creatinine kinase, creatinine kinase-MB, lactate dehydrogenase, and transaminase) were obtained 24 hours after operation. One month after the procedure, a transthoracic echocardiogram was performed and compared with preoperative imaging.

Table II. Procedural data in the two study groups

	Study group*	
Procedural data	*EAR (n = 49)*	*OAR (n = 71)*
Duration of operation (min)	180 (150-240)	180 (150-210)
Packed cells (n)	0 (0-0)	2 (1-3)†
Duration of intubation (h)	8 (3-13)	12 (8-15.5)†
ICU stay (h)	20 (18-22)	22 (20-23)†
Clamping time (min)	<1	50 (44-60)†
Hospital stay (d)	5 (3.5-8)	11 (9-15)†

ICU, Intensive care unit.
*Range is given in parentheses.
†*P* .01.

Outcome events. Risk factors (of which the association with adverse outcome events were assessed) were the type of operation, American Society of Anesthesiology (ASA) classification, history of CAD, age of patient, and use of β-blockers. The following outcome events were distinguished: The first group of clinical events included death of all causes, cardiac death, myocardial infarction, congestive heart failure, and a combination of the clinical cardiac events 2 through 4; the second group of outcome events consisted of electrocardiogram- or TEE-identified abnormalities (ie, ST abnormalities, WMAs of the left ventricular wall, and a combination of events 6 through 7 that indicated myocardial ischemia).

An ST abnormality was defined as ST-segment depression of 0.5 mm or more or ST-segment elevation of 2 mm or more in the precordial leads and 1 mm or more in all other leads, compared with the baseline reading and lasting for at least 1 minute. Regional WMAs, as identified on intraoperative TEE, were defined as a change of 2 or greater in wall motion score in one or more of the 13 segments of the left ventricle compared with previous echocardiography. The definition of myocardial infarction was based on the existence of at least one of the following criteria: ST abnormality and elevation of cardiac enzymes greater than twice their normal level or persistant WMAs on the 1-month postoperative transthoracic echocardiogram. Congestive heart failure required the symptoms or signs of pulmonary edema, chest radiographs (which showed vascular redistribution, interstitial and alveolar edema), signs of new heart failure (cardiomegaly, S3 gallop, jugular venous distension, peripheral edema), and a change in medication.

Statistical analysis. Data were expressed as medians and interquartile ranges. Differences in paired data sets (Δ-values) were expressed as mean values. The comparison of data between groups was performed by chi-squared analysis for categoric variables. The *t* tests were used for continuous variables with approximate normal distribution. The Mann-Whitney nonparametric test was used to compare other continuous or ordinal variables. Wilcoxon's signed-rank test was used for the analysis of paired data. Statistical significance was reached when the probability value (two-sided) was less than .05. Multivariate logistic regression analyses were used to investigate correlations

Table III. Hemodynamic parameters relative to aortic occlusion (*AO*) in patients who underwent EAR and who underwent OAR

| | *Study group* | | | | | |
| | *EAR (n = 49)* | | | *OAR (n = 71)* | | |
Parameter	*Before AO*	*During AO*	*After AO*	*Before AO*	*During AO*	*After AO*
Heart rate (beats/min)	60	62	60	61	58	65
Mean BP (mm Hg)	74*	74	76†	84*	75	71†
SVR (dynes/cm⁵)	1182	1030	1050‡	1328	1153	820‡
PWP (mm Hg)	13	14	14	14	13	16
CI (L/min/m²)	2.3	2.8	2.6§	2.4	2.4	3.1§
SWI (g·m/m²)	33.1∥	35.3	37.3	37.4∥	35.7	33.7
SV (mL)	76	83	82	76	81	90

*$P < .01$.
†$P = .03$.
‡$P < .01$.
§$P < .01$.
∥$P = .03$.

between a number of clinical variables, the clinical outcome events, and electrocardiogram-TEE–derived outcome events. The analysis of data was performed with the statistical software SPSS (SPSS, Inc, Chicago, Ill) for Windows 7.0 (Microsoft, Inc, Redmond, Wash).

RESULTS

Procedural data. The duration of tracheal intubation, stay in the intensive care unit, and duration of hospital admission were significantly shorter in patients who underwent EAR (Table II). In addition, the volume of required packed red cells was less in the EAR group. The duration of the operative procedure was similar in the two study groups.

Intraoperative cardiac responses. Hemodynamic parameters, including CI, SWI, SV, PWP, heart rate, BP, and SVR measured before aortic occlusion (baseline measurements), were all in the normal range in both study groups. Besides a higher BP in patients who underwent OAR, there were no significant intergroup differences (Table III). However, the baseline physiologic myocardial performance was less in patients who underwent EAR, which was indicated by a significantly lower preaortic occlusion value of the SWI of 33.1 gm/m² (range, 26.8-39 gm/m²) in the EAR group compared with 37.4 gm/m² (range, 31.7-46.5 gm/m²) in the OAR group ($P = .03$). A graphic illustration of the procedural evolution of CI and the SWI is presented in Fig 1. During aortic occlusion, there was a comparable increase of the CI in both groups. After aortic occlusion, however, the CI remained stable in patients who underwent EAR, although it continued to increase in patients who underwent OAR (mean rise from preaortic occlusion to postaortic occlusion, 0.2 and 0.7 in the two groups, respectively; $P < .01$). The changes in terms of percentage of postaortic occlusion CI were +27% in OAR versus +8% in patients who underwent EAR. The SWI during the procedures represented a distinctly different cardiac response in patients who underwent EAR and patients who underwent OAR, with an increase of 1.9 in the former group and a decrease of –1.4 in the latter ($P = .04$). This inverse change in SWI continued after aortic occlusion, with an increase of 2.58 and a decrease of –0.91, respectively ($P = .01$). The changes in terms of percentage of postaortic occlusion SWI were –2% in patients who underwent OAR versus +7% in patients who underwent EAR. Details of changes between groups are illustrated in Table IV. In addition to this intergroup comparison, a paired statistical analysis was performed of changes within the same groups. The SWI in patients who underwent OAR decreased (37.4 before aortic occlusion and 33.7 after aortic occlusion; $P = .04$), although no significant change was found in patients who underwent EAR (33.1 before aortic occlusion and 37.3 after aortic occlusion; $P = .16$).

Outcome events. The 30-day overall mortality rate in this patient series consisted of five patients (4%). In three of these patients (2.5%), the cause of death was of cardiac origin (two patients in the OAR group and one patient in the EAR group). One patient died of pulmonary insufficiency (EAR group), and one patient died of multi-organ failure (OAR group). Two of the perioperative deaths had an ASA-4 classification, two of the perioperative deaths had an ASA-3 classification, and one of the perioperative deaths had an ASA-2 classification. There was no difference in the overall or cardiac mortality rate between the two study groups (Table V).

Myocardial infarction occurred in four patients, resulting in one death (25%). Congestive heart failure was encountered in nine patients, three of whom died (30%). The incidence of combined clinical cardiac complications (including cardiac death, myocardial infarction, or congestive heart failure) was 6% and 11% in the EAR group and the OAR group, respectively; the difference was not significant. Clinical complications occurred at a mean interval of 4.6 days after operation.

In the perioperative period, ST elevation or depression occurred in 39 patients (33% of observations). These abnormalities were observed immediately after aortic occlusion in 11 patients and on the first 24-hour electrocardiogram analysis in 33 patients. WMAs of the left ven-
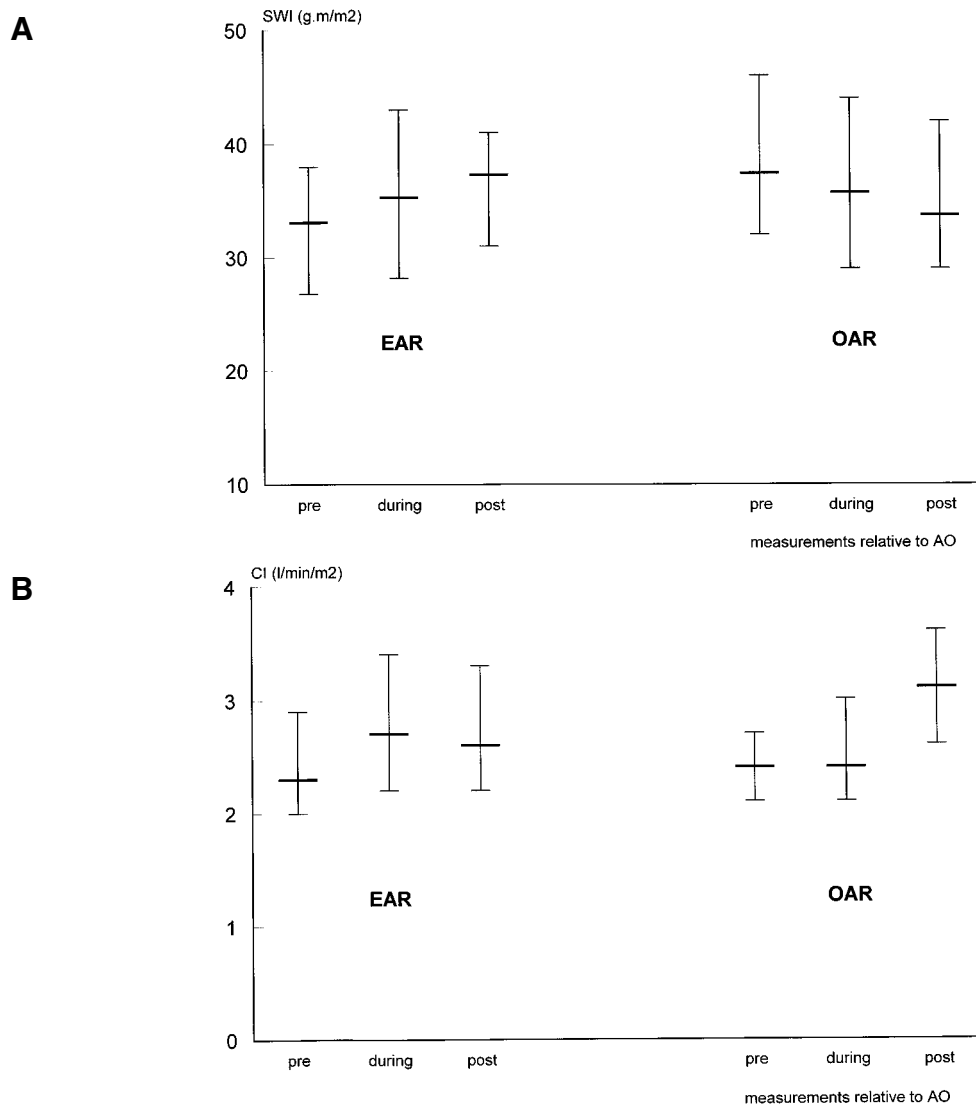
A

B

Fig 1. A, Comparison of SWI between the two study groups at three points of time relative to aortic occlusion. The value before aortic occlusion is significantly worse in patients who underwent EAR ($P = .03$). In patients who underwent EAR, the SWI improves during each successive step. In patients who underwent OAR, there is a gradual worsening of SWI. **B**, Comparison of CI between the two study groups. During EAR, the CI is relatively stable. After clamp release (after aortic occlusion), there is a significant increase of the measured value in the patients who underwent OAR.

tricular wall was observed during TEE in 22 patients (22% of recorded examinations). Nine patients with WMAs on TEE had no ST abnormalities. Signs of myocardial ischemia, as observed on electrocardiogram or TEE (either ST abnormalities or WMAs), were present in 50 patients (47% of observations), with a significantly lower incidence in patients who underwent EAR (33% vs 57% in patients who underwent OAR; Table V). Occurrence of myocardial ischemia was clinically silent in 84% of the patients.

Multivariate analysis. Five pertinent clinical risk factors (ASA classification, patient's age, history of CAD, type of operation, and the use of β-blockers during the procedure) were entered into a multivariate logistic model for correlation with two outcome events: combined clinical cardiac complications and electrocardiogram/TEE-identified myocardial ischemia (Table VI). The ASA classification appeared to be the only independent significant risk factor with a positive association with combined clinical cardiac complications. OAR, as the type of operation, was the single risk factor that significantly correlated with electrocardiogram/TEE-identified myocardial ischemia.

These findings were thought to be particularly relevant because the OAR group consisted of older patients. This analysis indicated that age correlated neither with the

Table IV. Comparison of the changes of hemodynamic parameters during and after aortic occlusions within the two study groups

| | Changes during aortic occlusion[*] (± SEM) | | Changes after aortic occlusion[†] (± SEM) | |
	OAR	EAR	OAR	EAR
PWP	−1.74‡ ± 0.61	1.48‡ ± 0.59	0.98 ± 0.89	1.51 ± 0.83
CI	0.13 ± 0.10	0.22 ± 0.12	0.68§ ± 0.11	0.21§ ± 0.12
SWI	−1.4| ± 2.43	1.86 ± 1.78	−0.91¶ ± 2.7	2.58¶ ± 1.8
SV	8.48 ± 4.42	0.68 ± 3.10	13.1 ± 3.14	4.23 ± 3.41

*Parameter value during aortic occlusion minus during aortic occlusion.
†Parameter value after aortic occlusion minus before aortic occlusion.
‡P < .01.
§P < .01.
|P = .04.
¶P = .01.

Table V. Outcome events in the two study groups

| | Study group | | | |
| | EAR (n = 49) | | OAR (n = 71) | |
Outcome event	n	%	n	%
Death (all)	2/49	4	3/71	4
Death (cardiac)	1/49	2	2/71	3
Myocardial infarction	2/47	4	2/65	3
Congestive heart failure	3/49	6	6/71	8
Combined clinical cardiac outcome events[*]	3/47	6.4	7/66	11
ST abnormality	13/49	27	26/71	37
WMA on TEE[†]	7/44	16	15/58	26
Electrocardiogram/TEE-detected myocardial ischemia	15/45	33‡	35/61	57‡

*Including the occurrence of cardiac death, myocardial infarction, and heart failure.
†TEE data for 18 patients were missing because of the inability of probe introduction (patients) or logistic reasons (13 patients).
‡P = .01.

occurrence of clinical cardiac complications nor with electrocardiogram/TEE-detected ischemia.

DISCUSSION

Since the introduction of open repair of AAAs, the mortality rate has decreased from approximately 10% in its early days to below 5% in contemporary institutional series.[6,7,20,21] On the other hand, in regional and national surveys, the reported mortality rate is still between 6% and 10%.[5] Approximately 40% to 70% of perioperative deaths are due to cardiac causes.[6,7,9,22] The frequent occurrence of cardiac complications has been attributed to a high incidence of preexisting CAD in this particular patient category.[23,24] Equally important is the impact of the procedure itself, which imposes a considerable stress on the myocardium. This stress is the combined result of anesthetic induction, aortic clamping and declamping, blood loss that is inherent to open aortic surgery, and the associated hemodynamic and metabolic consequences.[10,11] In contrast, endovascular repair is believed not to produce such profound hemodynamic alterations. Therefore, EAR may be expected to impose less stress on the myocardium and to cause fewer cardiac complications. In a recent study by the Eurostar registry[2] that included 899 patients with an endovascular AAA repair, the overall mortality rate was 3.2%,[5] which is lower than the mortality rate in most series with open surgery. This low figure was achieved despite the fact that 9% of the patients were at high operative risk, because their conditions had an ASA class 4.[2] The few studies that have compared conventional and endovascular repair found no difference in mortality rates between the two treatment methods.[1,3,4] However, these studies suggested that EAR was associated with less systemic complications, reduced blood loss, and shorter intensive care and hospital stays. We were able to confirm these findings in the present study. This may suggest that the endovascular operation is better tolerated by the patients than conventional repair.

The primary objective of our study was to compare the cardiac implications of the procedures and, in particular, the cardiac complications. For this purpose, we performed a prospective study in two patient groups that were comparable for most clinical variables. The patients' ages were

Table VI. Results of multivariate analysis that correlated risk factors to the probability of adverse events

	Coefficients	*SE*	*Odds ratio*	P *value*
Combined clinical cardiac complications*				
ASA (class 4)	1.93	0.94	6.9	.04
Age (yr)	.03	0.06	1.0	.63
History of CAD	.39	0.80	1.5	.63
Type of operation (OAR)	.70	0.82	2.0	.39
β-blockade	.35	0.85	1.4	.68
Myocardial ischemia†				
ASA (class 4)	1.22	0.81	3.8	.13
Age (yr)	−.3	0.04	0.9	.42
History of CAD	.83	0.46	2.3	.07
Type of operation (OAR)	1.31	0.47	3.7	<.01
β-blockade	−.7	0.47	0.9	.89

*Includes the occurrence of cardiac death, myocardial infarction, or heart failure.
†As detected by electrocardiography and TEE.

higher in those who underwent OAR. However, the regression model that was part of our analysis showed that age had no significant effect on the study end points. As shown in Table V, we found no difference between the two treatment groups with regard to myocardial infarction, congestive heart failure, overall deaths, cardiac death, and all clinical cardiac complications combined.

The detection of adverse cardiac events is highly dependent on the sensitivity of the perioperative surveillance because it is recognized that clinical adverse events only constitute a small proportion of all perioperative ischemic events. Not only do many ischemic events occur several days after surgery, but most such events (up to 90%) are silent (ie, without any clinical sign).[25-27] For this reason, we used an extensive protocol of intraoperative and postoperative monitoring to detect subclinical episodes of myocardial ischemia to accurately compare the two treatment modalities with regard to their cardiac effects. In addition to electrocardiogram analysis (as the most common method of detecting myocardial ischemia), echocardiographic monitoring was included in our protocol. In previous studies, regional WMAs of the myocardium, as detected by TEE, have been demonstrated a sensitive indicator of myocardial ischemia and a predictor of cardiac complications.[12-14] WMAs may occur before or even without electrocardiographic or clinical manifestations. In this series, nine cases of myocardial ischemia were identified by TEE alone. In 47% of our patients, we found signs of myocardial ischemia, of which 84% were clinically silent.

The important finding in this study was that, in comparison with OAR, EAR carries a lower risk for perioperative myocardial ischemia (33% vs 57%; *P* = .01). This observation may be related to the relative minor cardiac function changes associated with EAR that were found with the use of hemodynamic measurements. In accordance with two recent studies, we demonstrated that hemodynamic alterations were significantly less severe in patients who undergo EAR.[28,29] Patients who underwent OAR, in our study, experienced an important hyperdynamic circulatory state after the aorta was unclamped, as demonstrated by a rise in CI (Table IV; Fig 1, *A*). The rise in CI was significantly more important in the

patients who underwent OAR than in the patients who underwent EAR. It is of note that the CI reflects the general hemodynamic state, which is highly dependent on the existing preload and afterload. The increase of CI after cross-clamp release is caused by a decrease of SVR combined with sufficient volume load. For these reasons, the SWI was calculated, as a more direct reflection of the left ventricular myocardial contractility.[30] During aortic occlusion, SWI significantly decreased in the patients who underwent OAR, as opposed to a nonsignificant increase in the patients who underwent EAR. The changes were statistically significant between the two study groups and indicated a depression of myocardial contractility in the patients who underwent OAR as opposed to the patients who underwent EAR. It may be assumed that the increased CI causes a higher workload for the myocardium and a higher oxygen consumption.[31] This may be detrimental for patients with CAD and may precipitate myocardial ischemia by altering the relation between the myocardial demand for oxygen and the supply. The simultaneous occurrence of hemodynamic changes and myocardial ischemia appears to confirm a causal relationship between the two events.

Myocardial ischaemia was detected more frequently in patients who underwent OAR. However, this was not associated with an increase in early cardiac death or other adverse cardiac events in the perioperative period. This finding may be due to the relatively small number of clinical events in both treatment groups. Additionally, it is of note that, on the basis of the preoperative cardiac screening and intraoperative monitoring, more coronary interventions and medication changes were performed in patients who underwent OAR than in patients who underwent EAR. It seems reasonable to assume that this affected our results by having improved the cardiac status of the OAR group. Consequently, relatively more adverse cardiac events may have been prevented in the OAR group than in the EAR group. Another interesting speculation in this respect may be derived from the findings of Mangano and colleagues,[32] who demonstrated that perioperative myocardial ischemia increases the risk for serious cardiovascular outcomes by a

factor of 2 to 20 over a period of 2 years after noncardiac surgery. These considerations make it likely that EAR will be associated with fewer adverse cardiac events in large study groups, with completely comparable patient characteristics and an extended follow-up time.

One may ask what practical consequences the observed lower degree of cardiac stress may have on the extent of the preoperative cardiac workup algorithm. In our institution, screening by a cardiologist is the routine schedule, with dobutamine stress echocardiography performed in patients with a clinical suspicion of significant CAD (ie, angina pectoris, previous myocardial infarction, and/or congestive heart failure). In case important left ventricular WMAs are observed, a coronary angiogram is performed. One could argue that such a stringent protocol is no longer appropriate for a procedure that is significantly less stressful than open aortic surgery. Advanced pretreatment cardiac diagnostic procedures may become unnecessary in the large majority of patients with AAA.

In conclusion, important differences in cardiac response were demonstrated between endovascular and open AAA repair. Our findings indicate less myocardial stress during endovascular repair. A lower frequency of clinical adverse cardiac events in patients undergoing endovascular repair may ultimately be expected.

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