production. In fact, normoxemic reperfusion of a distal ischemic organ (eg, the intestine) has been shown to cause myocardial injury that was prevented with progressive oxygen re-entry during reperfusion.⁴

Second, in the above studies, similarly to the current one, with the exception of MDA measurement, the favorable effect of hypoxemic reperfusion was not substantiated by direct indices of oxidative stress alterations, such as ROS measurement. ROS and cytokines were recently assessed in a model of hypoxemic resuscitation of hemorrhagic shock in rabbits and found to be particularly diminished compared with those seen in the standard resuscitation mode of clinical practice.⁵ The weakening effect on cytokine production is an additional important achievement of hypoxemic resuscitation because these mediators lead to post-ischemia-reperfusion inflammatory injury.

Third, the authors have repetitively used erroneously the term “hypoxic reoxygenation” instead of “hyperoxic reoxygenation” to describe Pao₂ values of 250 to 350 mm Hg, which is usually applied in the clinical setting. Furthermore, we consider even more accurate the term “hyperoxemic” because it describes higher arterial oxygen content largely and easily modifiable by fraction of inspired oxygen changes; in contrast, the term “hyperoxic” describes tissue oxygen concentration dependent on addition to hemodynamic parameters. Correspondingly, the other treatment branch is represented by gradual reoxygenation by means of an initial “hypoxemic” instead of “hypoxic” period.

Our published data support the findings of Abdel-Rahman and colleagues¹ regarding the principal idea of the strategy for the prevention of reperfusion injury,²⁻⁴ the restoration of hemodynamic parameters,^{4,5} and the attenuation of oxidative stress.²⁻⁵ Additionally, these data provide a further insight into the potentially protective role of this strategy through the atten-

uation of the inflammatory response during reperfusion.⁵

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HYPOURICEMIC EFFECT OF STATINS: ANOTHER PLEIOTROPIC BENEFIT?

To the Editor:

We read with interest the recent trial by Hillis and associates,¹ demonstrating that hyperuricemia was associated with poorer survival after coronary artery bypass grafting (CABG), independently of other risk factors, including the European System for Cardiac Operative Risk Evaluation (EuroSCORE). Also, this increased risk was particularly evident among patients with levels in the upper quartile.

It has been showed that treatment with statins² can reduce serum uric

acid (SUA) levels; thus they would have an additional beneficial effect apart from that derived for lowering low-density lipoprotein cholesterol (LDL-c) levels. However, little information is available about the effects of high doses of statins on the SUA levels. The present study focuses on the possible effect of using a high dose of atorvastatin on SUA levels. We therefore hypothesized that the use of the highest dose of atorvastatin (80 mg/day) in high-risk patients under treatment with atorvastatin 40 mg/day would reduce SUA levels.

We studied 29 “high-risk” patients (inclusion criteria: severe triple vessel disease but rejected for bypass for extensive coronary disease, severe effort angina after coronary artery bypass, and premature coronary disease with ≥ 3 risk factors) with an abnormal lipid profile despite atorvastatin 40 mg/day at baseline and at 3 months after increasing the dose to 80 mg/day. Baseline results in patients were compared with those of 39 healthy controls. The lipid profile, C-reactive protein levels (CRP), SUA levels of patients and controls, and the changes after increasing doses of atorvastatin are shown in Table 1, A and B. At baseline, patients had higher levels of SUA ($P = .036$) compared with controls. Follow-up was completed in a total of 26 patients (2 patients withdrew voluntarily from the study and 1 patient had no samples at 3 months). After the dose of atorvastatin had been increased, a significant decrease in CRP ($P = .004$) and SUA ($P = .018$) levels were detected among treated patients. Also, these reductions did not show correlation with the changes of lipid profile (cholesterol, triglycerides, LDL-c, and high-density lipoprotein cholesterol). No adverse effects were observed in any patients.

The present study shows that increasing doses of atorvastatin at 80 mg reduces SUA levels compared with the 40-mg dose in high-risk patients with ischemic heart disease. Previous studies have shown that

TABLE 1. A. Test values of patients and controls. B. Test values at baseline (atorvastatin 40 mg/day) and 3 months of study (atorvastatin 80 mg/day).

Part A	Patients (n = 29)	Controls (n = 39)	P value	Part B	Baseline (n = 26)	Postintervention (n = 26)	P value
SUA (mg/dL)	6.0 (4.9–6.9)	5.2 (4.0–6.1)	.036		6.0 (4.9–6.8)	5.8 (4.8–6.4)	.018
Total cholesterol (mg/dL)	221 (228–252)	230 (204–249)	.862		229 (201–258)	192 (173–217)	.001
HDL-c (mg/dL)	42 (37–52)	50 (43–56)	.011		42 (36–50)	42 (38–48)	.517
LDL-c (mg/dL)	145 (130–174)	155 (130–177)	.820		146 (131–175)	122 (95–138)	.001
Triglycerides (mg/dL)	163 (97–252)	110 (77–146)	.008		166 (87–250)	132 (103–194)	.201
CRP (mg/dL)	0.30 (0.16–0.64)	0.15 (0.07–0.37)	.019		0.32 (0.17–0.69)	0.14 (0.09–0.34)	.004

n, Sample size; SUA, serum uric acid levels; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol, CRP, C-reactive protein.

preoperative statin therapy can reduce cardiac mortality after CABG by means of mechanisms that are independent of their cholesterol-lowering properties.³ We suggest that one of these mechanisms may be the hypouricemic effect of statins. The underlying mechanisms of this effect of statins are not clear-cut. It has been speculated that statins increase renal blood flow and, thus, increase renal urate excretion and lower serum creatinine levels.⁴ According to the results shown by Hillis and associates¹ and given that measurement of SUA is a routine procedure with repeatable results in clinical laboratories, we propose the clinical importance of monitoring and intervention on the basis of increased SUA levels and re-emphasize the positive effect of using high doses of statins.⁵

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EXTENDING THE SUITABILITY OF ENDOVASCULAR THERAPIES DURING TYPE A ACUTE AORTIC DISSECTION REPAIR

To the Editor:

We read with interest the article of Sun and coworkers¹ on the use of the stented elephant trunk for type A acute aortic dissection (TAAD). The aim of this hybrid approach is to stabilize the residual dissected aorta and thus prevent late thoracoabdominal aneurysm formation and the need for reoperation. Even though the idea is extremely attractive, we are doubtful that stenting the descending aorta is really needed during TAAD repair. Indeed, a literature review reveals that expansion of the residual dissected aorta after surgical repair is a slow and linear process that makes distal reoperations relatively uncommon. In an article published on the same issue of this *Journal*, Stevens and colleagues,²

reporting the 25-year experience of the Massachusetts General Hospital with 195 patients treated for TAAD, concluded: "Aortic reoperations were infrequent during follow-up." In another recent paper, Geirsson and colleagues³ reported that only 12 of 221 patients required late reoperation for thoracoabdominal aneurysm. Similarly, Dobrilovic and Elefteriades⁴ reported just 4 distal reoperation in a series of 200 patients operated on for TAAD.³ Taking these data into consideration, we believe that adjunctive descending endovascular stent grafting during emergency surgical repair might add supplementary risk to an already complex procedure with a high mortality rate, stabilizing something that has a low probability to cause a surgical problem in the future.

In our opinion, a solution to improve long-term results of TAAD is represented by combining a radical surgical resection with an extensive arch debranching to create a safe and long proximal landing zone for subsequent endovascular procedures on the descending aorta, if needed.

Our technique consists in replacing the ascending aorta and the arch and relocating the origin of the supra-aortic vessels very proximally just above the sinotubular junction, using a specially designed quadrifurcated vascular prosthesis (The Plexus 4-Branch; Vascutek Terumo Inc, Scotland, United Kingdom). If the patient will be one of the few who needs a second procedure on the descending aorta, this preventive debranching will allow a safe and effective endovascular treatment in both the short and the long term.