

Discussion: Session 4— Descending/Thoracoabdominal Aorta

Moderator: Joseph S. Coselli, MD

Panelists: Michael Jacobs, MD, Robert Dion, MD, Matthias Karck, MD, Charles Dong, MD, John A. Elefteriades, MD, FACS, Nicholas T. Kouchoukos, MD, and Inderjit S. Gill, MD

DR JOSEPH S. COSELLI (Houston, TX): I would like to start out just with a side note. I found of interest Dr Gill and associates' experience with traumatic aortic aneurysms and their concept that placing these patients on pump or left heart bypass really did not impact the incidence of paraplegia. Interestingly, we went back and looked at a fairly comparable group of our own descending thoracic aortic aneurysms, and these were proximal third, middle third, distal third, and combinations thereof. We also found that left heart bypass in the descending thoracic aorta really has not impacted our incidence of paraplegia or mortality.

I would, however, like to start off by asking at least one question myself. Dr Jacobs, you heard Dr Dion's presentation that false negatives and false positives with somatosensory-evoked potential monitoring just simply were not an issue in their experience. I noticed in your presentation that along with motor-evoked potential monitoring, you are simultaneously also looking at somatosensory-evoked potential monitoring. I would hope that you would comment.

DR MICHAEL JACOBS (Maastricht, The Netherlands): During the last 10 years, we simultaneously recorded the SSEPs and the MEPs, and without exception, we had a 100% reliable outcome with the use of the MEPs and a consistent false-negative outcome of between 20% and 25% with SSEPs. We are not the only ones. In Holland, we have another group using exactly the same technique with a huge experience. In Nieuwegein, they have exactly the same experience with false negatives, and in the literature, we find exactly the same outcome. It is logical that MEPs should correlate better with paraplegia because with MEPs we are measuring the anterior horn, which is the part where the motor neurons are located; that is the area we are interested in, and they are much more vulnerable to ischemia.

And the other drawback, in our experience at least, is that the SEP information has a delay of at least 10 to 30 minutes, and we do not have the time to wait for that information. So that is one of the reasons we are not relying on SEP anymore. But maybe Dr Dion has a much better machine than ours.

DR ROBERT DION (Leiden, The Netherlands): I have played the devil's advocate with my colleagues, the

neurophysiologists, in this type of discussion on many occasions. In fact, everybody is right: the only problem in reconciling our data is that the way in which we measure SSEPs is different from what is done by many others.

It is true that MEP is faster than SSEP, and with SSEP you sometimes have to wait as long as 15 minutes. We have no cases where the SSEP was positive after 15 minutes. I can not explain why we do not have that and others may. But the maximum time for positivity of the SSEP has been 15 minutes. That is why we wait for 15 minutes with the aorta being clamped segmentally.

The second point is that it is important to measure multilevel and bilateral SSEPs, because if you are not measuring multiple levels and bilaterally, you can indeed have false-positive and false-negative results. This was already published by Crawford, I believe, in 1988, and it has been discussed in previous meetings by my neurophysiological colleagues. I believe that if I look at the way my two colleagues are measuring SSEPs, I would expect that they would have false positives and false negatives. I believe it is very important to have a neurophysiologist in the operating room, and to do SSEPs bilaterally and at multiple levels, and also to vent, of course, the segmental part of the aorta between the two clamps. When we have looked back at our results, and have discussed them with the team, not a single patient had a false negative or a false positive. Of course, we had 4 of 82 patients who developed paraplegia, but we knew it. It was not a false negative or a false positive. I was not able during the operation to correct the cause.

There is also the advantage with SSEPs of preoperative and postoperative measurements. We have seen how painful MEP can be; it is obvious when you look at the patient that he is being shocked by this electrical stimulation. There is definitely an advantage to being able to follow SSEPs before and after the operation.

So my conclusion is that one is not better than the other. I think we should simply use both. And the assumption that you only measure the posterior column with the posterior SSEP is not really true. If you look at my slide showing the lumbar enlargement, you can see that the point N24, which is in the gray matter, is 1 cm away from the alpha motor neuron. So it must be very rare indeed that you have ischemia of the anterior wall gray matter and not of the posterior wall gray matter.

This is all I have to say. I am not a neurophysiologist,

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but this is the consequence of multiple discussions of this problem.

DR COSELLI: I would submit that the monitoring devices and the techniques, the unilateral versus bilateral recordings, may indeed impact the false positives. But clinically, at least intuitively, we see patients who would fall into the category of a false negative. I have patients who wake up with motor dysfunction (in effect, paraplegia from a motor standpoint) but have their sensory function intact; in effect, they have a partial anterior cord infarct with preservation posteriorly.

I would like to move on to a couple of other issues, and I would like to poll the audience on a number of things. I would like to go through some of the monitoring techniques and then some of the protective adjuvants. How many in the audience are using motor-evoked potential monitoring in their descending and thoracoabdominal work? It is a sprinkling but it does not seem to be very popular.

How about somatosensory-evoked potential monitoring, is there a greater usage of that? There is some use, but again, it is not exactly a consensus.

And finally, with regard to preoperative evaluation, spinal angiography, anyone in the audience in institutions that are using that particular approach routinely? One, two, three; not too many.

Let us move on to protective adjuvants then. How about cerebrospinal fluid drainage? It looks as though that has moved into a majority. I think that would be probably a bit different than a decade or so ago.

Finally, how about use of left heart bypass in descending aorta operations? Again, quite a number. This seems a little less popular than cerebrospinal fluid drainage, but probably is used by a majority.

What I would like to do is ask the panel to discuss a problem that I think we all wonder about. Again, it is probably a multifactorial issue. It has not really been addressed yet, and so I would like the panel to tell us just briefly about their experience with delayed paraplegia and what they do about it when they see it.

DR JOHN A. ELEFTERIADES (New Haven, CT): We do see it, and what we do is hop on it immediately. We raise the blood pressure and we expand the volume of the patient, and we often replace our spinal drain, if it is out. And I do not have exact numbers, but we have a good number of successes at restoring normal neurologic function.

DR JACOBS: If they are not on CSF drainage anymore, we immediately put a drain in, if possible. If the paraplegia disappears on the needle, so to speak, then that is it. The two delayed paraplegic patients we had were patients who had selective grafts to intercostal arteries, and emergency angiography showed that they had clotted off. So we brought the patient to the OR and took the clot out. One patient profited from that, and the other one did not.

DR MATTHIAS KARCK (Hannover, Germany): We have limited experience, with two cases in the last year who presented with delayed paraplegia following acute aortic dissection repair. They woke up the next morning

and could not move their legs. One patient we took to the interventional radiologist; he did a fenestration, and there was slight improvement of motor function so that he could at least move his toes. And over a couple of weeks or so, he recovered slightly but he did not go back to normal. And in the other case we did two things: we did CSF drainage and also fenestration, but we did not see any improvement.

So this is a very limited experience. But if one becomes aware of this problem, first of all, one has to stress that it should be treated as early as possible. Usually the problem is not detected until the next morning, but in dissection cases, at least, one should nevertheless consider fenestration. It may be worthwhile, although it did not work out so very nicely in our two cases.

DR DION: Well, we believe that the prevention starts in the operating theater. I think it is important not only to rely on neuromonitoring, but if it is technically feasible (and without the pressure of time) to reimplant more intercostal and lumbar arteries. I believe one of the major advantages of the technique of Dr Kouchoukos is that he has the time to reimplant everything he wants, and I think that is probably the reason why he has so few delayed paraplegias.

If cerebrospinal dysfunction happens postoperatively, I believe that the first thing we should do is to reintubate and sedate the patient. I think we should decrease absolutely the oxygen consumption and reestablish the equilibrium that we had in the operating theater. Of course, we would also utilize CSF drainage; we would improve PO_2 ; we would increase hemoglobin. And we have had 2 patients in whom we could treat delayed paraplegia and allow recovery. But I believe it requires a completely multifactorial management.

DR NICHOLAS T. KOUCHOUKOS (St. Louis, MO): With the technique that I mentioned, one of the patients developed delayed paraplegia. The patient had a ruptured a type II aneurysm and had had a previous colon resection. She had absolutely normal neurologic function for 10 days, and then she perforated her colon, which had previously been treated surgically. And after that procedure, with a hypotensive episode, she became paraplegic.

I would tend to agree with what Dr Dion said. We have not seen any other cases of delayed paraplegia, and I think a lot of it may relate to the hemodynamic instability early postoperatively. And this is only an impression, but it seems to me that as the techniques for protecting the spinal cord have been modified and improved (and you heard a number of them presented today), the prevalence of delayed paraplegia has increased. Would you agree with that? The ratio of initial to delayed paraplegia has changed some, if you look at the earlier series. Maybe the delayed paraplegias were missed because the patients may have been sedated or intubated longer. But I have a feeling that it has changed. And I think that relates, to some degree, to what Dr Dion has said. I think that if the protection is optimal intraoperatively, then we will see less paraplegia later. And we have situations that are marginal when the patient comes out of the operating

room, and these patients are vulnerable to changes not only in cerebrospinal fluid pressure but also changes in arterial pressure. And in looking at some of the series that have addressed this issue, it is hard to sort out whether it is the CSF drainage or whether it is the maintenance of very high arterial pressures that are responsible for the reversal in some situations.

DR COSELLI: At least in our work, we have not seen an increase in delayed paraplegia. I agree that the improvement in the preservation of the spinal cord has very likely had an impact, but I think that it is balanced out by our improved postoperative care and recognition of possible etiologies for hypotension. An increase in our reattachment of numbers of intercostal arteries, I think, also may leave us with a far less precarious collateral blood supply to the cord than we had in the past. I think with those two balances, we really have not seen much of an increase, but we still see it from time to time, and it is always very discouraging.

DR KOUCHOUKOS: Do you make a point of waking the patient up immediately after the operation to assess their neurologic function? I mean, if you are going to look for it, you have to look for it early.

DR COSELLI: I was actually going to ask you a little bit about that, because we do try to wake the patients up within 2 or 3 hours postoperatively; therefore, not quite immediately. But in order to have a fairly smooth hemodynamic course, we do not reverse them in the operating room. But with your particular approach, how long in your patients after cardiopulmonary bypass and hypothermic circulatory arrest are you able to assess them postoperatively for spinal cord problems?

DR KOUCHOUKOS: Probably not for at least 12 hours

DR RANDALL B. GRIEPP (New York, NY) (editorial comment): With postoperative SSEP monitoring, it is possible to allow patients to awaken slowly or remain sedated and still assure that the spinal cord is intact. Thus, the benefits of both hemodynamic stability and hour-to-hour assessment of cord integrity are not mutually exclusive.

DR JACOBS: In Europe, we do not fight with the lawyers as much as you do over here, but there is one group of surgeons who are fighting a lot with the lawyers, with paraplegia as the main subject, and those are the orthopedic surgeons. If a patient is paraplegic in Europe, or in The Netherlands—my country—at least following scoliosis correction (pulling up the spine), and the surgeon did not use motor-evoked potentials, he is in big trouble. So it is amazing to see when you posed the question here that nobody is using this very, very simple technique, which is one hundred percent reliable. So it is just an interesting phenomenon to see, in a country where liability is so important, that you get away without monitoring.

DR KOUCHOUKOS: Can I ask you a question in that regard? Don't you have to have a technician or somebody who is experienced in doing this kind of work? Also fairly sophisticated equipment? We have had experience with somatosensory monitoring, and it is not technically simple to do. Certainly, if you make it a consistent part of

your practice, it works wonderfully. But cost is an issue I guess that comes into consideration in our country for that kind of monitoring.

DR JACOBS: You are absolutely right, but I am just amazed by the difference in the fact that orthopedic surgeons are almost obliged to do it. We are at liberty to put it in our protocol or not.

DR COSELLI: I can not speak to orthopedic surgery as a whole, but I can tell you that when they do have a paraplegic patient postoperatively, they are pretty much guaranteed a lawsuit. At least in our institution, many of the orthopedic surgeons are not using motor-evoked potential monitoring, but they do use some somatosensory-evoked potential monitoring in cases that they believe are at risk.

For descending and thoracoabdominal aneurysms, for spinal cord protection and visceral protection (not for patients who are anatomically unsuitable for a cross-clamp), how many are using cardiopulmonary bypass and hypothermic circulatory arrest, similar to what Dr Kouchoukos does? It is probably about 25% or so, maybe 25% or 30%. That is quite a good following.

With regard to that, something that comes up from time to time is the question of the additional heparinization with full cardiopulmonary bypass and the use of cerebrospinal fluid drainage. Could you comment?

DR KOUCHOUKOS: We have not used cerebrospinal fluid drainage. When we looked at this technique in the primate, we actually found out that during the course of using cardiopulmonary bypass and hypothermic circulatory arrest in a baboon, the cerebrospinal fluid pressure went up during the time of the operative procedure and then went back down. If we had been concerned about it, we would have instituted some other measures, but the fact is, none of those animals developed spinal cord ischemic injury. So we do not use it at all. Since we have not seen any delayed paraplegia except in the case that I mentioned, we do not use it postoperatively. I suspect that if we saw a patient develop paraplegia postoperatively, we would use CSF drainage. But because of heparinization, I think there is an additional risk, and we do not use it intraoperatively.

DR COSELLI: For the rest of the panel that uses CSF drainage, one of the complications reported in the anesthetic literature is subdural hematomas in a few of these patients. Has anyone on the panel seen that? (No response.) Has anyone in the audience seen it? So, there are a few.

DR DION: We have had an infection. It was a really particular situation. Sepsis from the drainage line occurred 3 days after the operation. We had maintained the catheter for 3 days because we were still unsure, early in our experience. But we have had no hematomas.

DR COSELLI: The point is that CSF drainage is not without some potential complications. It is not exactly a free ride.

DR SCOTT BUCHANAN (Portland, ME): I wonder if the panel could address two specific issues: the use of aprotinin in these patients, and the use of epidural catheters for pain control postoperatively.

DR ELEFTERIADES: We do use aprotinin and we interrupt it during circulatory arrest on our ascending operations and arches, and we have not had a problem with it.

DR KOUCHOUKOS: We had an experience with aprotinin early on and had some catastrophic complications with intravascular thrombosis, and there are a lot of reasons for why that happened that are probably not relevant any longer: the techniques for monitoring ACTs and so forth were different. We do not use it in patients in whom we use hypothermic circulatory arrest until after the procedure, and we use it there only if there is significant bleeding. We do not use it routinely.

Epidural catheters we will occasionally put in during the postoperative period for control of pain, but not in everybody.

DR DION: I would use aprotinin for every single patient with a thoracoabdominal aneurysm. Of course, there is always the point that if you have to reoperate on the patient later on for something else, then you have to use aprotinin perhaps again, but I have never seen complications from aprotinin in this setup. We have seen complications in coronary artery surgery, and in valvular surgery, I saw a catastrophic anaphylactic reaction with total vasoplegia of a patient coming back for a tricuspid valve repair. But I am feeling stronger if aprotinin is given; just a feeling.

DR COSELLI: Dr Gill, you had a lot of bleeding in those traumatic cases. Did you use aprotinin?

DR INDERJIT S. GILL (Cleveland, OH): We never use aprotinin, and we normally use a catheter the next day postop.

I would just like to make a comment about your delayed paraplegia. One of the things we do in our practice is that we do not extubate the patients right off the bat. We usually keep them intubated for 24 hours, and the instructions the nurses have are to keep the PO₂ more than 100, keep the hemoglobin more than 10, and keep the pressure more than 100 to prevent delayed onset of paraplegia.

DR COSELLI: Hemodynamic stability, blood pressure, oxygenation, volume.

DR KARCK: We also use aprotinin, and we have not yet experienced serious complications that may be attributed to the fact that aprotinin was used. Epidural catheters we do not use.

DR COSELLI: We use aprotinin in about half of our patients, and we use hypothermic circulatory arrest through the left chest. So I have used it about 25 times. I have not had the same catastrophic events that Dr Kouchoukos mentions, but we have had enough problems with those patients that it may have actually been hidden among some of the other difficulties encountered and it was probably not isolated. So it is a little hard to speak to. But when you get bleeding in that particular set of circumstances, it is awfully tempting to use aprotinin, at least after you have exhausted some other efforts and end up with a coagulopathy type of picture.

DR MEONG GUN SONG (Seoul, Korea): I have a couple of questions for Dr Kouchoukos. My first question

is why don't you perfuse the femoral artery during the proximal anastomosis instead of using total circulatory arrest? I think it might contribute to reduced ischemic time.

And my second question is, why don't you perfuse the brain and the heart after proximal anastomosis through a graft sidearm? I think it would decrease the brain ischemic time significantly, and also, you would have plenty of time and there would be no hurry after the proximal anastomosis.

And my third question is, your pulmonary complications are very high compared with my cases. How do you protect the lung during cardiopulmonary bypass? I think touching the lung during cardiopulmonary bypass will create pulmonary edema or pulmonary hemorrhage. It will induce a high incidence of pulmonary complications.

And I have the same question for Dr Gill. Why don't you use total circulatory arrest or hypothermic cardiopulmonary bypass instead of partial bypass or a simple cross-clamping technique? I have done 24 cases of the same kind of disease but with no mortality and no paraplegia.

DR GILL: As far as doing the traumatic cases the way you described, I think you can do them any way you want. There are papers that will support every point of view. This is one method we have chosen to use and we have done it successfully, and so we stick by it. But there is nothing wrong with what you do. If you do it well, that is fine.

DR KOUCHOUKOS: Let me just address the lung issue. We utilize femoral cannulation. If there is any concern about the femoral artery, one can use other methods. We have used the axillary artery; you can use the subclavian artery; you can use other areas of access to the aorta. I do, however, believe, and this I think is in consonance with what Dr Jacobs said, that a substantial amount of the collaterals supplied to the spinal cord come from the lower portion of the body, from the lumbar and from the iliacs and hypogastrics. So whenever possible, after completing the proximal anastomosis and working on the intercostals, the lumbar, the renals, et cetera, and doing distal anastomoses near the bifurcation of the aorta, we continue to perfuse through the lower femoral arterial cannula, because that provides substantial flow and hypothermic protection through the hypogastrics and the lower body. We may not use it initially to establish cooling, but we always put the femoral cannula in and perfuse that area for the type I and type II aneurysms.

Now, the issue of the lungs is an important one, and obviously with a fully heparinized patient one runs the risk of intraparenchymal hemorrhage of the left lung. Dr Crawford, I think, reported a very large complication rate early on, and he was discouraged from using the technique because of the significant prevalence of pulmonary complications.

I think it is important to keep the lung totally collapsed and not to try to inflate it, if at all possible, until the procedure is completed and the heparin has been reversed. We do see occasional intraparenchymal hemor-

rhage. It has never, in our experience, been a cause of death or serious morbidity. There are pulmonary complications, but in my review of the literature and what is available from other series, our pulmonary complication rate does not exceed that for any of the other techniques. But one has to be careful how one treats the lung when the patient is fully heparinized.

DR STEWART FINNEY (Baltimore, MD): How long do you leave your epidural spinal drainage catheters in, on the average? And does your threshold for operating on an aneurysm change when you have, for instance, a woman of smaller stature, if that woman is 120 pounds with a bicuspid valve and a 4.5-cm ascending aorta and a 2-cm descending aorta?

DR COSELLI: Let us let Dr Elefteriades look at the latter part of that question. The aortic sizes in your database, as I understand it, are direct aortic diameters at the maximum dilatation but not really indexed to the size of the patient. Could you comment?

DR ELEFTERIADES: That is a very important observation, and we take it into account in an informal way. If we have a small woman who has a 5-cm aorta, we consider that about the same as a 6-cm aorta in a small woman, tall man. So we take that very much into account, but we have not formalized it.

DR COSELLI: It comes to one of those last lines in one of your slides: individualization is extremely important.

For the panel, how long do they keep the epidural catheters in?

DR JACOBS: Seventy-two hours; 3 days

DR COSELLI: Forty-eight hours.

DR DION: Two days, and a little bit more if we have a patient with adult respiratory distress syndrome. If you are in a situation where the patient is not yet completely awake or still agitated, there is low hemoglobin, low PO₂, atelectasis, or something like that, then a third day would be better.

DR DAVID SPIELVOGEL (New York, NY): I want to make two comments regarding the MEPs in the United States, and I have a question. Motor-evoked potentials are not FDA approved, so any institution that is interested in adopting this method of monitoring has to go through the IRB. We have done that at Mount Sinai, and they are embarking on a program, but it is not easy. So just keep that in mind.

The other thing I wanted to mention is that we have had two instances of meningitis related to CSF catheters. So that it is not a benign procedure, although we keep the catheter in as long as we think it is necessary, usually about 48 hours, depending upon the number of intercostals sacrificed, and as long as 72 hours if we are really concerned about our patient.

As I am sure you are familiar, we use a technique that Dr Griep developed, where we sacrifice intercostals in groups while sequentially monitoring SSEPs, looking for changes, and we almost never reimplant intercostals. So I am a little curious about the issue of delayed paraplegia in the surgeons that reimplant intercostals. Do you think it is possible that these grafts or these patches of intercostals are causing delayed paraplegia by thrombosis

and that this actually might be a setup for that scenario? The spinal cord is at a stage where the supply is balanced between those intercostals that are reimplanted and the supply from above and below. If the reimplanted intercostals thrombose in an acute setting, then the cord may become ischemic. In our technique, we sacrifice the intercostals and allow accommodation to take place slowly before resection. And although we have seen some delayed paraplegia, it usually is responsive to CSF drainage and to super-normal increases in the blood pressure.

So I just wanted your comments about the implantation of these intercostals.

DR GRIEPP: Dr Jacobs, I think you said something once about it or twice about seeing this. Does it worry you to have a single intercostal hanging on a graft like this?

DR JACOBS: I guess it is a very complex discussion, because, first of all, if I can bounce the question back. To me, one and one makes two. If you disconnect blood supply to an organ, you have to reattach or reconstruct it to make the organ live again. So I have never understood the beautiful results achieved, for example, by Dr Acher and yourself, where you sacrifice all the intercostals and still have a living spinal cord. That is a question for you.

But, in general, I am convinced that if you revascularize the spinal cord in an optimal way, the chance of delayed paraplegia is very, very limited. If you only have one or two intercostals reattached and one of these two clot off or are blocked, yes, of course, you have delayed paraplegia. But if you have a liberal reattachment, you have backup systems proximally and distally, and the system has a reserve blood supply, so to speak, even if one or two occlude. So I feel that it is a very logical, functional approach as long as you reimplant liberally.

DR ELEFTERIADES: When I was in The Netherlands, Dr Jacobs and I were talking, and we use exactly the same technique. We implant the intercostals with a sidearm graft, which we call a cobra head graft, off the main graft, and it has been very effective for us. We have done several dozen of these. We use multiple techniques for intercostal reimplantation. There has not been a paraplegia in a patient who has had one of these sidearm grafts. But whether they stay patent or not, I do not know, and it may be that if they stay patent for a few days, that is all that you need.

DR GRIEPP: Dr Kouchoukos, do you want to say anything? Do you advocate attaching everything? You did not tell us exactly what you do. What is your policy for intercostal attachment?

DR KOUCHOUKOS: I mentioned it briefly. For the patients who had extensive thoracic or thoracoabdominal aneurysms, we attempted to reimplant in every case, and we were able to reimplant the vessels in about 62% or 63% of the group. So we do attach intercostals.

DR GRIEPP: But how do you decide how many to do?

DR KOUCHOUKOS: Since we do not have monitoring, we generally try to attach all the patent vessels below the level of T5 or T6. Now, with dissections, it is easier because one can make a large pedicle. With the athero-

sclerotic grafts, beveling sometimes will preserve some of the vessels without the need for separate attachments. We use either buttons or the graft technique that the others have mentioned of attaching them separately. And it is not unusual for us to attach one or two with a separate 10-mm or 12-mm graft. We have seen CT studies of some of these patients, and they remain open; not all do, but certainly more than half.

DR MANUEL IRARRAZAVAL (Santiago, Chile): I would like Dr Elefteriades to elaborate a bit more on the value of the pathology report on the prognosis of patients with aortic aneurysms. Does it make any difference whether the pathologist tells you that you have cystic necrosis, for instance? Is it useful to have pathology reports any longer?

DR ELEFTERIADES: That is really a pet peeve at our institution. Because you do this big operation, you send down the aorta, and you expect a meaningful reply. And the reply is "aorta." We are so concerned about the lack of attention to the information in that very valuable specimen that we now freeze all of our aortas. We freeze every aorta that we take out at minus 60°C so that we have a tissue bank that we can call on in the future to look at MMPs or whatever other parameter we may wish to examine. But we find the routine pathology report totally useless.

DR GRIEPP: I might just add to that (and we are not sure it is that beneficial), but with pathology reports, one will find a small but real incidence of giant cell aortitis and on occasion other forms of aortic pathology that were unsuspected clinically. We are not sure what you do about giant cell aortitis, but some cases that show progressive dilatation of unresected aorta are treated with corticosteroids. So there is some value to pathology, I think.

DR DOMENICO SCALIA (Padova, Italy): To Dr Gill, a very short question. Do you have any experience with endovascular stent grafts in traumatic rupture of the aorta?

DR GILL: We are in the process of starting a study on that, but we have not done any yet.

DR GRIEPP: Does anybody want to say anything about that? I think there have been attempts to do that.

DR GILL: There have been attempts to do that, and some have been done successfully. There have been some reports from South America. I think Dr Buffolo was talking about some yesterday. But we have not done any in our institution yet.

DR GRIEPP: While Dr. Gill is speaking, I would just like to editorialize a little bit with regard to traumatic rupture, and I do this speaking from ignorance because we rarely see patients in New York City that get going fast enough to have traumatic ruptures. But I worry about a group of cardiac or cardiothoracic surgeons coming away with the message that clamping and sewing is as good as using distal bypass in a setting in which distal bypass is readily available. One can not argue with your data. The two cases of paraplegia occurred in the bypass patients, and there was none in the clamp and sew group. But I do not know how to explain that, because this is the

one model in which reattachment of intercostals and so on all should be moot, where one is dealing with a part of the aorta that does not generally supply the spinal cord. I am uncomfortable with the inference that use of distal bypass might increase the incidence of paraplegia, if that is what you meant to imply.

DR GILL: I think it is a little too early to make any firm conclusions from these data. I think our study is fairly small, and I pointed that out repeatedly during my talk. So I think it is too soon to say whether this inference will bear out down the road. But you see what we reported. I do not want to send a message out either that clamp and sew is the safe way to do it, but that is something we have seen.

I can tell you that we have had problems with patients who have been put on partial bypass during a trauma setting; we have seen dissections. I am actually involved in a case, not with a trauma patient but in a reoperation, where distal femoral bypass started a dissection and it ended up causing a compromise of the subclavian and IMA graft. So these are not techniques without problems or complications. And I think if somebody can do something that is fairly simple and straightforward in 20 or 30 minutes, I do not think you need to add any adjunct therapies to do it. But if you cannot, then I think partial bypass is certainly helpful.

DR KOUCHOUKOS: Can I make a comment about that? An excellent meta-analysis was done by one of the South African surgeons, van Oppell. That study indicated that the average number of procedures done was something like 1.3 per year per center, and that would include the major trauma centers and also the other community hospitals where these lesions are encountered. That reason alone would tend to make me think that one needs all the support one can get. I do not know about the other surgeons on this panel, but one of the things I am still asked most often to review in terms of legal issues relates to paraplegia that occurs after a traumatic dissection. And if you look at some of those cases, obviously a straightforward transection can be repaired very quickly. But if one encounters a tear that extends underneath the subclavian artery, or there is the necessity to establish some sort of circulatory support, you have lost it because you have already exceeded the safe limits of aortic cross-clamping. So the best method is not clearly established, and there is no doubt that experienced centers can do these procedures in a variety of ways. The Houston group (the trauma center there) has been advocating for a long time that they be done simply. But I think the surgeon who only does one of these occasionally and who does not have extensive experience in aortic surgery should use some sort of distal support.

DR GILL: I would agree with that. I think that idea should be passed on: that distal support should be used by most trauma surgeons who do not see more than one or two cases a year. But a surgeon who is a trained cardiac surgeon and is going to take whatever time it takes to do his anastomosis, if he can do it less than 30 minutes, it does not really matter whether he does it on-clamp or off-clamp. But the point is well taken. I think

the average surgeon should be using some kind of distal perfusion.

DR ERNESTO LARRAIN (Santiago, Chile): I would like the panel to comment on the methods of diagnosis in traumatic injuries of the aorta. Do you think one should do aortography (which is time-consuming, which is not always available) versus TEE, which is almost everywhere?

DR GILL: At our center, a first test is a chest roentgenogram, and that usually is done in the emergency department. We have a CT scanner in our ED. So the patients usually go from one room to the next for the CT scan. And then the next thing is an aortogram, and then to surgery, if need be.

There have been papers published that suggest that

the specificity and sensitivity of TEEs and CT scans is in the range of 75% to 80%. So the gold standard still is an aortogram. So if you are going to take somebody to the OR, I think at the end of the day you will have to have a firm diagnosis. There are centers that are becoming adept at doing TEEs, and that is fine, but the gold standard still in our practice and in our minds is an aortogram.

DR GRIEPP: I would like to thank very much the panelists, the faculty members, and all of you who contributed your abstracts and videos to this meeting. It looks as though there is enough interest in aortic disease that we should do this again in 2 years. Until then, thank you very much.