Panel discussion

J. E. ECKENHOFF
M.D., D.Sc., F.F.A.R.C.S.

Northwestern University, Chicago

G. E. HALE ENDERBY
F.F.A.R.C.S.

Queen Victoria Hospital, E. Grinstead

I. R. VERNER
M.B., Ch.B., F.F.A.R.C.S., D.A.

Middlesex Hospital, London

C. PRYS-ROBERTS
F.F.A.R.C.S.

Radcliffe Infirmary, Oxford

J. B. HESTER
F.F.A.R.C.S.

Queen Victoria Hospital, E. Grinstead

PROFESSOR ECKENHOFF: Dr Enderby's change of title from 'Ganglionic Blockade' to 'Pharmacological Blockade' was a very appropriate one. Too many of us have figured that deliberate hypotension and ganglionic blockade were simultaneous expressions of exactly the same thing; that the connotation was that if you had deliberate hypotension you had to have ganglionic blockade. I would point out that you are not trying to use a technique for the sake of the technique; you are using it to produce certain operative conditions and you can do it by a variety of techniques. You may use one or another or you may have to use all of them, but the idea that deliberate hypotension implies the presence of ganglionic blockade to the exclusion of all else is wrong.

I would add one final comment before going to the questions, and perhaps Dr Enderby would like to respond to this. The use of deliberate hypotension depends upon the surgeons with whom you work. What you do in one hospital and with one surgeon may not be applicable to another institution and another surgeon. When Dr Enderby makes the statement that you never use halothane before you use a ganglionic blocker this is dependent upon the speed with which anaesthesia can be administered and the surgery started. If I were to use a ganglionic blockade in the same way as Dr Enderby I would have hypotensive anaesthesia for 45 min, with the surgeons still not cutting, and I have to do something to keep the patient asleep, and for this reason I go ahead with halothane and cut down on the ganglionic blockade.

DR ENDERBY: I feel very strongly that deliberate hypotension has two sides, anaesthetic and surgical, and unless the two are in balance it should not be attempted. Indeed I now say that unless a surgeon can make effective use of what I honestly and truthfully think are the best operative conditions that we can presently provide, then I will not go ahead and do it. It is indeed a marriage between anaesthesia and surgery. We are both on each other's ground. We are assisting their surgery and they in their turn must be able to make use of what we are doing. I have had one or two experiences, some in this

country and some in yours, Mr Chairman, which have led me to the conclusion that you have got to keep the balance between these two arts.

With regard to the time spent before the commencement of surgery this can, of course, bring difficulty for the anaesthetist. If you must have 45 min of scrub-up as some surgeons insist on, before you get down to surgery well, in my opinion you have not got to use halothane for that period. You may perhaps think of using a relaxant such as decamethonium although there are, of course, many other agents to help over such a period.

Still, these are but small facets of this broad picture and the fundamental issue remains; if the patient's sensitivity is altered with halothane it may well be the reason for considerable regret when a powerful ganglion blocking drug is used.

PROFESSOR ECKENHOFF: This assumes you use the same dose in both instances, but I use halothane and then cut down on the dose of the ganglionic blocker.

DR KARL ECKHARDT: What pathophysiological conditions are considered contra-indications to deliberate hypotension?

DR VERNER: When using sodium nitroprusside liver failure or renal failure are serious contra-indications. These are the only absolute ones I can think of. As regards relative ones, I think patients with pre-existing neurological disease need special care, although when one is doing neurosurgery many patients have neurological disease and they usually respond favourably.

As regards cardiovascular disease, this is seldom either an absolute or relative contra-indication, unless, of course, the patient is at death's door. Many people have used sodium nitroprusside in treating acute coronary thrombosis.

So, in brief, therefore, liver or renal failure are definite contra-indications where the reserves of these organs are already restricted and any further trespass might prove disastrous.

PROFESSOR ECKENHOFF: If my memory serves me

correctly Dr Enderby considers bronchial asthma a contra-indication. Does he still believe this?

DR ENDERBY: There are two conditions which I think contra-indicate the use of ganglion blockade for deliberate hypotension—asthma and diabetes. I do not know what relationship exists between them and the use of sodium nitroprusside.

PROFESSOR ECKENHOFF: Let me ask a further question in relation to asthma. Have you had any problem with an asthmatic with deliberate hypotension since you ceased to use trimetaphan? What I am getting at is that trimetaphan releases histamine, depending on the dose of the drug used, and I have often wondered if the problems you described in your early reports in 1961 were due to this.

DR ENDERBY: I have never made much use of trimetaphan at any time. I have always preferred a long-acting ganglion-blocker and it is with these drugs—almost exclusively pentolinium and some hexamethonium—that my work has been done. I did in the early years encounter quite a few attacks of bronchial spasm or asthma during deliberate hypotension, so much so that I was of the opinion that the use of a ganglion blocking agent makes the asthmatic more liable to an attack. I do not know what the relative position is with regard to trimetaphan vis-a-vis pentolinium or hexamethonium.

DR VERNER: It may be of interest that about 3 weeks ago I had a chronic bronchitic who when intubated went into severe bronchospasm. I was in a hospital where there was no sodium nitroprusside and during the course of about a 2-hr operation we tried unsuccessfully with everything we could think of including halothane and ether. I then sent for some sodium nitroprusside in the hope that its smooth muscle relaxant action would relieve the bronchospasm. When the systolic pressure was taken down to 60 mmHg there was a great increase in the compliance of the lungs, but it was apparent that the cardiovascular effects of the nitroprusside outweighed the bronchial ones. Possibly a combination of salbutamol and nitroprusside might be more effective.

PROFESSOR ECKENHOFF: I have deliberately used the technique in several asthmatics, trying to obtain some information on this, and I find this is an occasion where giving a little more halothane and a little less ganglionic blocker has proved worthwhile.

DR PRYS-ROBERTS: One has to be particularly careful about patients with ischaemic heart disease and those with hypertension, not only because of the hypertension but because many hypertensives have ischaemic heart disease which may not be recognized. The problem is not that we do not use these drugs to bring down blood pressures when they are very high, but that we do not bring them down to the sort of levels that are being used during hypotensive anaesthesia because the anaesthetic *per se* is already depressing myocardial function. Hypotensive drugs, used on their own in conscious patients to regulate blood pressure, cannot have that effect. The problem with both groups of patients is that they are intolerant to both hypotension and hypertension. They do not tolerate hypertension because this increases myocardial work at a time when their coronary blood supply is insufficient to meet the demands of the increased work. The problem at the other extreme of

hypotension is that even though the myocardial work is reduced, if the perfusion of those impaired sections of the myocardium is dropped below a certain level, one can easily recognize it by ECG evidence of ischaemia, if one drops blood pressure beyond that stage then one gets into difficulties. A certain number of them retain ischaemic ECG signs afterwards and one has to say that they have had a silent infarct, which might otherwise have passed unrecognized. This is not to say these patients should not be subjected to hypotension, but if they are they should be carefully assessed beforehand and carefully monitored throughout operation and this is the one situation where the ECG really is valuable. A lot of people say ECG during anaesthesia is a useless measurement. I agree that in normal healthy people it does not tell you very much, but in patients with ischaemic heart disease, be it from simple ischaemia and atheroma, with or without hypertension, the ECG is the first and best early-warning signal of disaster.

PROFESSOR ECKENHOFF: Now, in relation to what you have just been talking about, if you have a patient who does have signs of myocardial ischaemia and a blood pressure of 160/110, and you drop his pressure to 130/90, which produces the conditions that you want, do you consider you have indulged in deliberate hypotension?

DR PRYS-ROBERTS: No, because in all probability that pressure would have been achieved whatever you did.

PROFESSOR ECKENHOFF: Well, let us say you could not get it with whatever you did and you have instead to use some of the techniques that we have been talking about?

DR PRYS-ROBERTS: On the contrary I think you could. You do not have to use either ganglion block or any specific pharmacological technique in these patients. If you anaesthetize them and tilt ever so slightly their blood pressure will go down.

PROFESSOR ECKENHOFF: Would you not consider that deliberate hypotension?

DR PRYS-ROBERTS: Yes, I would. It is deliberate in that one is trying to produce something extra, over and above the pharmacological effects of straightforward anaesthesia. But in most people of that type, blood pressure comes down whatever you do.

PROFESSOR ECKENHOFF: I would agree. My interpretation, however, has been that if I deliberately do something to lower blood pressure, that is deliberate hypotension; and, again, I do not like to equate deliberate hypotension with a systolic blood pressure of 60 or 50 mmHg.

DR ENDERBY: It is purely a matter of degree, is it not? I may well run my patients lower than some and there may be others that do them lower than myself. It is very difficult to say 'Thus far and no further' from the point of view of figures; you can only do this in relationship to what is being performed, that means to the requirements of surgery. I speak for myself and say I seldom get what I want with a pressure higher than 80 mmHg. On the other hand, I seldom need go lower than 60 mmHg. So there is a band here within which I usually find I am working, and within that band I have found little to contra-indicate either the fit young patient or those with ischaemic heart disease such as Dr Prys-Roberts is talking about. In this latter group there must, of course be a careful and preferably slow reduction of pressure

and the ECG is a valuable safeguard which should always be used.

DR MIDDLETON: Has nitroprusside been passed by the Committee for Safety in Medicines or is it too old to need their approval?

DR HESTER: As far as I know it is too old to need approval.

DR VERNER: I have been told by a member of the Scowen Committee that it is too old.

DR ENDERBY: Was the question directed at the age of the drug or of the Committee?

PROFESSOR ECKENHOFF: I had a hunch it might be both. I would suspect that if this drug were to be considered in the United States by the FDA, even though it were an old drug, it is being subjected to a new use, and it would have to be equated, and it would never get through. We have many good drugs lying around and we send most of them to other countries to test because the law prohibits us from testing them ourselves, except under pretty rigid conditions.

QUESTIONER: What do the speakers think of the combination of halothane and β -blockers in view of the myocardial depressant effect of halothane?

DR PRYS-ROBERTS: It is a complicated question because one cannot take all β -blockers as a generality. They have different effects. If we take the two most commonly used, propranolol and practolol, which are quite distinctly different, we have studied them firstly in man and animals already receiving halothane, where we have found remarkably little effect of the β -blocker *per se*, except in those situations where there has been a very fast heart rate beforehand, and I would stress that we almost invariably give atropine at the same time as the β -blocker. Secondly, we have studied the effects of halothane anaesthesia in patients who have been pre-treated with these β -blockers, and as far as we can see, if one considers such measurements as blood pressure and cardiac output, the patients under β -blockers are more normal than those without. We cannot find any evidence to support the statement, widely made in the United States, that β -blockers and anaesthesia are bad news. If anything, they are good news. We can find no evidence that β -blockade *per se* causes any depression of the myocardium other than that which would be expected from withdrawal of pre-existing sympathetic activity. The question has been fouled up, I think, by misunderstanding in the past of statements made about propranolol and some other agents, stated to have direct negative inotropic effects. Now it is true that propranolol in very high doses has what is called a membrane-stabilizing or quinidine-like effect. The point is that the doses needed to produce these effects are enormous compared with those used clinically. In the clinically-used doses of propranolol, I was talking of anything up to 20, 25 mg intravenously of propranolol, in man, and of practolol in doses up to 100 mg intravenously (with practolol there is no membrane-stabilizing activity) we cannot find any evidence in either man or animals, of direct depression of the myocardium. So one is really dealing with the synergistic effect or additive effect of what is already there in terms of halothane anaesthesia. If the halothane anaesthesia is associated with tachycardia (because it is too light and there is a response to

surgery) then if you give a β -blocker you will get a fall in cardiac output and fall in blood pressure, because you are removing a pre-existing sympathetic activity. Similarly the same will occur if you use it with halothane in states of hypercapnia and under the response of carbon dioxide. But if the patient has not got any obvious evidence of sympathetic over-activity, giving practolol or propranolol (and I presume any of the other β -blockers) will have very little direct effect *per se*, except that it will block that patient's response to either endogenously or exogenously induced catecholamines.

PROFESSOR ECKENHOFF: What size doses of propranolol were you speaking of in your use of this drug?

DR ENDERBY: In the age group up to 30 I am talking in terms of up to 2 mg of propranolol and up to 8 mg of practolol—the one being about four times the dose of the other. The other category which I mentioned is the older patient where tachycardia interferes with circulatory control and then relatively very small doses, 0.25–0.5 mg of propranolol or up to 1 mg of practolol, are quite adequate.

PROFESSOR ECKENHOFF: I think this is a dose-related phenomenon. I look upon the use of the β -blocker and halothane in the same way as I do the ganglionic blockade with halothane, or of d-tubocurarine and ether. There are additive effects with these drugs, and if you have one aboard then you had better use the second cautiously or think the matter out. I believe the criticisms that have been offered against the use of propranolol (we do not have practolol available to us) in relation to halothane has usually been when halothane has been administered to individuals who have been on propranolol, and one always suspects that the anaesthetist goes out and anaesthetizes that patient with halothane just as he would a normal patient, using a high concentration, and gets into serious trouble. I believe this is where the problem lies.

DR PRYS-ROBERTS: The question is, what sort of troubles does he get into, and why does he get into trouble? Does he get into trouble because of bradycardia? Because if so he should be giving atropine before it. If not, where does the trouble arise? Excessive hypotension?

PROFESSOR ECKENHOFF: It is excessive hypotension with myocardial depression and it is difficult to get out of it in a hurry. This is the way I look at it.

DR ENDERBY: May I be so bold as to ask whether myocardial depression *per se* is necessarily either troublesome or contra-indicated?

DR PRYS-ROBERTS: I do not think it is either troublesome or contra-indicated, unless by inducing pharmacological impairment of ventricular contractility you impair the ability of that heart to respond to a load. This is the important thing about the heart. The normally functioning heart, that is neither physiologically nor pharmacologically depressed, can cope with enormous changes of load and still maintain an output. It is only when you subject the physiologically or pharmacologically depressed heart (and the former, of course, is the patient with the infarct) to an increase in load that it leads to left ventricular failure. Of course this has been the problem with propranolol that has been given to patients who are in incipient heart failure who are reliant on a very high

degree of sympathetic control in order to maintain a normal cardiac action. As soon as it is removed there is trouble and the patient goes into left ventricular failure. It is a question of load. If the patient's heart is not asked to work against a high load, then he is all right; but once the failing heart is asked to work against a load it just fails. It cannot do it. It fails to produce an output. The left ventricular pressure and end-diastolic pressure rise, and you get the classical picture of failure.

DR VERNER: The only trouble I ever got through using β -blockers and halothane together was that I was always left in the theatre looking after the patient when the others had coffee! If you have to use these drugs to produce a bradycardia, in order to get the blood pressure down, their effects will not wear off quickly; and if you wish the pressure to rise quickly, having given a β -blocker, this may be difficult. You are sometimes left with a patient whose blood pressure remains around the 90–100 mmHg systolic for 2 or 3 hr.

DR ENDERBY: If you use sodium nitroprusside it comes back without any hesitation.

DR PRYS-ROBERTS: With sodium nitroprusside and β -blockade the blood pressure will come straight back up, but this is not so with halothane.

DR DAVID ENDERBY: Have you measured cyanide levels in any of your patients that have had sodium nitroprusside?

DR HESTER: No, we have not.

DR DUNCAN FERGUSON: Would you please comment on use (or not use) of atropine before administration of propranolol during general anaesthesia?

DR HALE ENDERBY: I started by using atropine in the premedication, but I now omit it because I consider I am using propranolol with the intention of slowing the pulse rate and it seems a contradiction to combine it with another which does just the reverse. My feeling at this moment is that the use of atropine is unnecessary.

DR PRYS-ROBERTS: I would like to add a rider to that. I think it is unnecessary if the purpose of giving the β -blocker is to reduce an already elevated heart rate; but if the heart rate is normal and you are giving a β -blocker for another reason as for instance to prevent the sympathetic response to surgery or to catecholamines, then I think it is important to give atropine, because if you do not you may get a marked bradycardia. If you are giving small doses of β -blocker to reduce an already elevated heart rate, and if you take the intrinsic rate in man as between 95 and 105, if the rate is above that level there is certainly no need to give atropine, but with heart rates very much slower then it should be given.

DR ENDERBY: I agree in theory but I just wonder in practice what this means? I am using propranolol with the deliberate intention of slowing the heart. You are saying that if you are going to use it for any other reason—and there are many reasons why you might want to use it—then you should not slow the heart. In my opinion the use of atropine appears to be unimportant.

DR PRYS-ROBERTS: Well it may matter in ischaemia. I am talking particularly about patients with ischaemic heart disease, where anything that is going to slow the heart is going to reduce output, and this is not well tolerated. But in the normal healthy patient it does not matter a great deal.

DR ENDERBY: On that ground I agree with you.

DR PRYS-ROBERTS: And in any case the sort of doses you are using are very small. I am talking now of using very large prophylactic doses of β -blockers, to prevent a known response to a sympathetic stimulus.

DR ENDERBY: We have heard today of doses as large as 150 mg of practolol which are indeed enormous, because for me about 8 mg, or perhaps a little more, is usually a maximum. This sort of dose is usually quite adequate, whilst in older people there is often an amazingly good response out of minute amounts of this drug. I am not claiming to be a homeopathist but I do feel we can easily make a mistake in thinking that the dosages we anaesthetists use are unimportant. I think they are terribly important. They are small, but they can be very effective.

PROFESSOR ECKENHOFF: Now we come back to something we were talking about this morning—that is, you have got to be very careful when you extrapolate things done in non-anaesthetized patients with those who are anaesthetized, for you are talking about dose levels under two different conditions.

DR ENDERBY: For instance if you consider the treatment of hypertension with propranolol you will find in the literature doses of 1000 mg administered 4-hourly I believe.

DR PRYS-ROBERTS: Daily.

DR ENDERBY: Surely more than daily—at least four times a day. In any case it is fantastically big in comparison with the 0.5 to 1 mg I am using. I agree we must be very careful in making comparisons.

DR PRYS-ROBERTS: The point here is that in your situation you are trying to take the top off a pre-existing response, and I agree that minute doses are adequate. In tetanus patients, to reduce heart rate from 180/200 down to 120, we need less than 1 mg of propranolol. But if you are deliberately going to produce—we cannot talk about a total β -blockade, because there is no such thing, but an effective ED 50 for β -blocking, if for instance you want to protect a patient against the infusion of adrenaline, then you must use very much larger doses. In your case you are not talking about β -blockade, you are talking about the very bottom end of the dose response curve to the particular stimulus.

PROFESSOR ECKENHOFF: In controlling some of the tachycardias that occurred during halothane anaesthesia (this is unrelated to deliberate hypotension) it is interesting what three-tenths or four-tenths of a milligramme of propranolol will do, and it behoves anyone to use the smaller dose under these circumstances before going on to the larger dose.

DR FERGUSON: You state that nitroprusside gives a well perfused patient with a low arterial pressure. Is this not contradictory to good surgical visibility and/or reduced blood loss? Would you not agree that reduced perfusion is the essential factor?

DR FERGUSON: Is the term deliberate hypotension not euphemistic and are we not really seeking something other than hypotension *per se*?

DR VERNER: It was claimed in a recent article that good cardiac output with a reduced peripheral resistance will lead to hypotension yet flow can be maintained and it does not lead to a bloody operative field, if you apply

a tipping posture as well; that if you get good venous drainage with good flow the tissues will be perfused, but it is pressure which is responsible for bleeding. I agree with this. We should not aim for deliberate underperfusion. But the practical thing is that with hypotension you can get good operative fields. In my opinion you should always employ posture wherever possible and from the evidence of two recent publications cardiac output may be unchanged or is even raised.

PROFESSOR ECKENHOFF: What was the body position of this group of patients when their cardiac output was measured?

DR VERNER: Offhand I cannot tell you.

DR PRYS-ROBERTS: Flat, I expect. Most of the studies done on sodium nitroprusside have been done with the patients flat. If you tilt them it does not matter whether you are using nitroprusside or a ganglion blockade, you get a fall in cardiac output.

PROFESSOR ECKENHOFF: My own recent experience has been that if you lower blood pressure and do not elevate the field above the level of the heart, even though you have a low blood pressure you will not reduce blood loss or produce a good field; and most of the studies on drugs that produce vasodilation show that as long as the patient stays in the supine position the cardiac output is elevated, but as soon as the body is tilted blood is pooled and cardiac output reduced.

DR ENDERBY: Dr Pallister's recent Hunterian Lecture at the Royal College of Surgeons provided interesting evidence in support of this. He talked in terms of what he calls 'rheostasis', a means of reducing bleeding without sustained hypotension and he showed in a series of excellently documented cases that they all exhibited one thing of importance, namely that the cardiac output was considerably reduced. In my opinion if you are going to get a good operative field it is very difficult to combine it with a normal, and certainly not with a raised, cardiac output. It is reasonable for me to assume that in my own patients the output is almost certainly reduced.

PROFESSOR ECKENHOFF: I would agree with that; and I think that appropriate operative conditions parallel lowered cardiac output better than they do lowered blood pressure. When you tilt the ordinary patient, and there is no significant change in blood pressure, the reason why you get the operative conditions you seek is because cardiac output goes down. This is my assessment. I find it very difficult to believe that cardiac output can be elevated and still produce the conditions desired.

DR PELMORE: H. J. V. Morton found drip counting intriguing and when he studied the size of drip from several similar drip chambers the volume varied by over 100%. Is this your finding, too? Have you ever checked the size of your drips?

DR HESTER: No, we have not. I think our need is just a case of juggling with the rate and the effect of this drug. The problem to me is the lag which always occurs after turning off the drip. Blood pressure continues to fall, and this is the major problem.

PROFESSOR ECKENHOFF: Drip size is, of course, a well known phenomenon illustrated by the old story of adding adrenaline to a local anaesthetic solution. One assistant uses 10 drops and the next also uses 10 drops, but the concentration may be 1 in 50,000 in one case and 1 in

400,000 in the next, simply because of needle size. Does Dr Verner have any information about this?

DR VERNER: No, I have not. I think the ideal thing if you want to study this problem is an infusion syringe, but it is an expensive piece of apparatus and not easily available within the operating theatre.

There is another observation I would like to make concerning sodium nitroprusside. My concept of hypotension has altered from use of this drug. We have today spent a lot of time talking about the β -blockers and halothane and of their prolonged effects, some of which are unacceptable. With sodium nitroprusside it is now possible to split the technique into two parts. For anaesthesia there is a wide choice of drugs to suit the patient and to this is added hypotension of whatever degree is necessary and it can be maintained for as long as desired. Furthermore, the hypotension can be slowly induced and there is seldom any 'after-fall'—this occurs mainly after a large dose, or if the drip has been running very fast when there may be sufficient left in the veins to cause a continued fall of pressure after it has been stopped. Its use can be tailored to whatever the surgeon and the patient needs. If your need is for pure hypotension, this is the drug to use. The size of the drip is largely immaterial unless of course you want to calibrate or measure it. It is necessary to give undivided attention to the drip rate and pressure readings, and the anaesthesia must be sound enough to look after itself.

PROFESSOR ECKENHOFF: I have to be careful, in most Anaesthetic Societies when I say I distrust drips, because of my long association with Dripps and Vandam. Nonetheless by choice I will not use a drip, and the reason is because I have had to appear too often in malpractice suits because of them.

DR VERNER: Could I ask you, then, how you would use sodium nitroprusside?

PROFESSOR ECKENHOFF: I would give 10 mg of pentolinium!

DR ENDERBY: We have been using intermittent doses of sodium nitroprusside in a dextrose/saline drip.

PROFESSOR ECKENHOFF: This is reminiscent of the use of Arfonad. So many people think the only way it can be used is by an intravenous drip, but in fact it can be used with satisfaction by intermittent doses.

DR VERNER: I cannot envisage being able to use sodium nitroprusside by intermittent doses. The action of this drug is so evanescent, within 15 sec there is usually clear evidence of a change in blood pressure, and it would seem impossible to obtain a steady control in this way.

DR EDRIDGE: I started by using sodium nitroprusside in a drip, and followed this with intermittent doses. In my experience this often gave a very jumpy, difficult to control pressure which did not encourage me to persist in using it in this way, nor for that matter was my interest maintained in this drug until it came into use again recently. However, I agree it is not an agent to be used by an intermittent dose technique.

PROFESSOR ECKENHOFF: Its action sounds very evanescent.

DR EDRIDGE: It is indeed.

DR VERNER: With experience of the drip it is possible to control blood pressure so that it does not fall too

quickly nor too far, and for the hypotension to be maintained satisfactorily. It is prudent to allow plenty of time to achieve the required blood pressure before surgery is commenced.

DR PELMORE: How do you suggest that closed circuit halothane, as used by Dr Beddard of Bath, showed such success—no fatalities in 10,000 patients deliberately hypotensed by this drug and method?

DR PRYS-ROBERTS: I do not know of this publication. My comment is that fatality is a very crude assessment of success. I would want to know a great deal more of the assessment of those patients in the immediate post-operative period, in the immediate week after anaesthesia and surgery. There are some very good studies being done, in Sweden especially, on old people and their responses to anaesthesia and surgery, and most of us would be pretty shocked by the results. I think we have a tendency to say that because the patient did not die on the table and that all appeared well at the end of anaesthesia then everything is satisfactory. This is a fallacy. In my opinion many old people get silent infarcts immediately after operation and very often this is not attributed to either anaesthesia or surgery, but simply to the fact that the patient was old and was in the 'coronary belt'. The results from Sweden show that in some of these patients the quality of life has been reduced, and the way they detected it was to look for evidence of infarction post-operatively and for evidence of ischaemic changes post-operatively. The average British patient is not assessed post-operatively, so how can we say that we are being successful? From the evidence of five papers reporting well conducted trials, from three different continents, 30% of patients with either hypertension or ischaemic heart disease show incidence of worsening of previous ECG changes, and in just under 10% of them there is evidence of frank infarction. Therefore, when you say there have been 10,000 without a fatality that does not necessarily imply anything. It is necessary to know what is the quality of life and what are the morbid effects of anaesthesia and surgery rather than what is the fatality rate.

PROFESSOR ECKENHOFF: In like manner I think this point of view is applicable to the National Halothane Study which involved 800,000 patients and used as its end point severe hepatic necrosis confirmed at death. It reached the conclusion that halothane was not involved in the production of hepatitis and I think most people would agree today that that was fallacious. I think that is a good example of the point that is being made.

DR DUNCAN FERGUSON: Do you use a standard premedication with your technique? If so, what?

DR ENDERBY: For many years the premedication used at the Queen Victoria Hospital has been levorphan and phenergan with atropine or scopolamine. Further to this I frequently use pethidine with atropine or scopolamine. I feel that the actual drugs used are never so important as the quality of their effects. If you want good hypotension easily controlled and satisfactory in the broad sense of the word, you must commence with a quiet and tranquil patient. One of the worst mistakes is for the premedication to be given late, then to drag the patient through cold, draughty corridors, before being left in an anaesthetic room where all the sounds and noises of the

theatre are easily heard. This sort of treatment results in veins getting tighter and tighter—a fact easily confirmed by observation of the hands and feet, and as it is the veins which, in large part, control the circulation they must instead be encouraged to dilate. Thus if it is possible to pick the patient out of bed after induction of anaesthesia many of these problems are avoided. Therefore, it is the quality of the premedication which is important; it determines how easy it is to perform deliberate hypotension thereafter.

DR PELMORE: Have you seen any post-operative haematomata as a result of the quick rise in post-operative pressure with sodium nitroprusside?

DR HESTER: In our series it occurred once following an operation on the leg for the removal of an extensive lesion.

DR VERNER: I have not experienced it with sodium nitroprusside but I have certainly had haematomata with other drugs and techniques. This does not mean of course that sodium nitroprusside is blameless in this respect. Post-operative haemostasis is influenced considerably by operation site and the ease with which counter-pressure can be applied. My experience with this drug in plastic surgery is limited but I doubt whether its effect on haemostasis is different from any other drug or technique.

PROFESSOR ECKENHOFF: I have always thought that one of the advantages of pentolinium was that pressure did not rise abruptly immediately after the procedure. Admittedly, it puts a greater strain on recovery-room personnel but on the other hand I had the distinct impression that it lessened the incidence of bleeding post-operatively.

DR ENDERBY: I would agree with those remarks. There is no doubt that if blood pressure rises too fast, there is a strong possibility of bleeding. There are many factors responsible for maintaining haemostasis during and after operation and pressure is only one of them, but should it swing violently then bleeding is likely to occur. These swings are small and certainly not violent if there is prolonged ganglion blockade. Indeed under pentolinium, at the end of an operation, when most pressor stimuli are removed the blood pressure, far from coming up too soon, may then tend to run too low. This, of course, is one of its drawbacks. These patients always need careful attention post-operatively. Nevertheless, it is my opinion that a long acting ganglion blocker has very definite advantages, particularly when dealing with skin surgery on the head and neck, where it is often difficult to apply any very effective counter-pressure to maintain haemostasis.

PROFESSOR ECKENHOFF: Before closing this panel discussion I would like to comment on the observation that during deliberate hypotension there is a widening in the A-V oxygen difference in the jugular bulb. Does this suggest cerebral hypoxia? Many years ago, certainly within a year or two after I returned from this institution to Philadelphia, we were able to complete satisfactorily four cerebral blood flow estimates out of seven. Some of these were in elderly individuals. We found that with a mean blood pressure around 40 mmHg cerebral blood flow was reduced on average by approximately two-thirds but, owing to a widening of the A-V oxygen difference, there was no evidence of a reduced cerebral

metabolic rate for oxygen nor was there any evidence of change in lactate, pyruvate or the lactate-pyruvate ratio, all of which suggested there was no sign of cerebral damage. However, the important question remains as to whether under abnormal conditions like these the jugular bulb is a valid place from which to draw samples?

A final question to Dr Prys-Roberts. It is very common in patients after halothane to have them shiver, sometimes rather violently. What happened in your series of patients in whom the pressure was reduced quite low with halothane alone? Did they shiver more than the usual patients?

DR PRYS-ROBERTS: No, they did not; in fact not a

single one shivered, at least not overtly, and we know from the measurements previously done on shivering patients that if oxygen uptake is within $\pm 20-30\%$ of basal oxygen uptake predicted for that patient it is almost certain that there is no shivering because otherwise there would be a big increase in oxygen consumption. The patients whom we studied who did shiver were not all on halothane. A fair number of them had had no halothane at all. The worst shiverer we saw did not have any halothane, and the concept that shivering is entirely due to halothane is fallacious.

PROFESSOR ECKENHOFF: The worst shivering I have ever seen occurred in those patients who were cold.