Clinico-pathological Conference

TRAUMATIC ISCHAEMIA

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PROFESSOR DONALD: Ladies and gentlemen, today we are going to deal with a research project which opens up an enormous field of therapeutic possibilities, particularly in traumatic surgery. There are, of course, many uses other than those to be discussed today.* We in our own department have benefited from the treatment of the respiratory distress syndrome, hyaline-membrane atelectasis, of the newborn, while the treatment with the pressure chamber of carbon monoxide poisoning is well known.

Today Mr. Smith is going to talk about traumatic ischaemia and it therefore gives me the greatest pleasure to introduce him.

MR. G. SMITH: Traumatic surgery is becoming more and more important as the incidence of road casualties rises. In many instances we feel that oxygen therapy has something to offer in the management of these cases. Let us consider, for example, the question of diminution of the blood supply to a limb. Normally the resting limb tissues themselves do not have a very high metabolic rate and they can get on with very much less blood flowing through them than ordinarily is the case. However, the margin between viability and non-viability of a limb, differing as it does between its various components, may be improved by extra oxygen. The basis of treatment in a pressure chamber is now fairly well known. The extracellular fluid is the end reservoir for the availability of oxygen to the individual cells. Under normal circumstances, while breathing air at atmospheric pressure the level of this extracellular fluid oxygen is about 0.25 volume %. By breathing oxygen at two atmospheres pressure we can push this up to about 4.2 volumes % regardless of what the haemoglobin is carrying. This, you will appreciate, is a very marked increase, which goes far to com-

pensate for a diminished arterial input. Furthermore, this increase of oxygen in solution should be regarded as occurring in the whole aqueous phase of the body so that we may in point of fact put oxygen directly into skin floating on a plasma exudate and thereby keep it alive.

So far we have treated eight cases of traumatic ischaemia in a pressure chamber, including two cases from the Southern General Hospital, one from Dundee and one from the Vale of Leven Hospital, Dumbarton. First, we have two cases of compartmental compressions in a limb, one in an upper limb and one in a lower limb. Here the genesis of the ischaemia has been trauma causing muscle damage and swelling within rigid osteofascial compartments with compression of the nerves and blood vessels therein, producing distal ischaemia. Secondly, there is a case of near avulsion of the foot which was sewn back, the posterior tibial artery reconstituted, and much of the foot kept alive. Thirdly, we have an example of a degloving injury about which Mr. Sillar will speak. Fourthly, we have a case of cold injury referred from Mr. A. B. Kerr's ward with which Dr. Ledingham will deal. Fifthly, apart from the ischaemic aspect of trauma we have an example of the hazard from clostridial infection which occurs particularly in road accidents. Sixthly, there is the treatment of shock, and we have an example of a girl with multiple injuries in the lower limbs and pelvis and a rupture of the left kidney who needed massive transfusions. She was transferred from the Vale of Leven Hospital with her blood pressure still down about 80 mm. Hg, and she responded very rapidly to treatment in the chamber. And, finally, we have a man who was run over by a railway truck and had a traumatic amputation of the foot. Mr. Brown did a Syme's amputation. The heel flap was very dubiously viable and he was treated in the chamber with the retention of that flap.

* See 'Treatment of Arterial Occlusion under Oxygen at Two-Atmospheres Pressure' by Sir Charles Illingworth, *Brit. med. J.* (1962), ii, 1271.

Compartmental Compression

The first case I wish personally to present is that of a boy aged 26 who sustained a tibio-fibular diastasis with fracture of the fibula at a rugby game in Dundee on November 4, 1961. The fracture was reduced and cross-screwed and at that time there was no suspicion that the blood flow to the foot was anything other than it should be. But two days later and more especially on the third day it was quite obvious to both Mr. Smillie and Professor Douglas in Dundee that the blood supply to the foot was severely impaired. He was sent here and was seen by Professor Barnes and myself who concurred with the opinion that the leg was not viable. At that time it was grey, cold and insensitive and there were no peripheral pulses.

Immediately after arrival he was put in the chamber and was kept there for four days, the pressure being allowed down for an hour every 12 hours. Compression was in compressed air while the patient breathed oxygen through a B.L.B. mask. The foot gradually became pinker overnight and the interesting thing which has been common to all these ischæmic cases is that the pain disappeared. With restoration of the circulation, bullæ appeared over the foot and over the anterior fascial compartment which was tense and exquisitely tender (Fig. 1). Tension was also building up in the posterior compartment. Now, of course, at this stage, one would be tempted to open the compartments but this almost certainly would have resulted in muscle pouring out and so we continued with the hyperbaric oxygen treatment. By the end of the next week when he was taken out of plaster for check X-ray it was quite obvious that there was still residual tension along both the anterior and the posterior osteofascial compartments. Further, on testing, he had no function at all in his anterior or posterior tibial nerve components but did have functioning cutaneous components, both saphenous and musculo-cutaneous nerves. The small muscles of the sole of the foot were probably also partly infarcted. Fig. 2 shows the leg at the end of the second week and you can see that all the œdema is gone but that there is a small area of full thickness loss on the dorsum of the ankle. The nerve function remained fairly stable.

On January 18th I put on a little skin graft and did an arteriogram at the same time which showed a completely normal pattern right down past the area of injury to the ultimate ramifications. This confirmed the clinical impression that this man had suffered ischæmia from the building up of tension in the osteofascial compartments through which the nerves and vessels were running and that this had placed the foot in jeopardy. He also

went through a phase where the small bones of the foot rarified patchily but they have now recalcified satisfactorily, again suggesting an ischæmic episode which has been adequately

FIG. 1.—Ischæmic foot one day after treatment in the chamber with bullæ over the dorsum of the foot and along the anterior fascial compartment.

FIG. 2.—The same foot as in Fig. 1 at the end of the second week showing full-thickness skin loss on the dorsum of the ankle but no œdema.

compensated for. There is still no function in the anterior tibial nerve component so that it may conceivably have suffered actual physical trauma as well as ischæmic damage at the time of injury. The posterior tibial components are migrating

downwards and have reached the heads of the metatarsals. The saphenous, sural and musculocutaneous nerves have stayed intact throughout. He is now walking, and indeed walked down the aisle about four weeks ago.

Near Avulsion of the Foot

MR. J. STEVENS: The second patient we wish to present this morning is a 16-year-old youth who was admitted to the Casualty Department in March last year within an hour of being involved in a motor-cycle accident. He had sustained a severe compound fracture dislocation of the left ankle with anterior displacement of the foot. When we first saw him, the foot was completely white and cold with total loss of sensation except for a small area of skin about 1.5 cm. in diameter over the lateral aspect of the base of the 5th metatarsal which was probably subserved by a small cutaneous branch remaining in a persistent skin bridge which I shall mention in a moment. The soft tissue injury was of an almost circumferential bursting type at and just beyond the level of the ankle joint. The only intact skin bridge between leg and foot was about 4 cm. wide and situated antero-laterally just beyond the level of the ankle joint. Immediately proximal to this was one of several extensive friction burns and there was also a 5-cm. laceration over the lower third of the anterior compartment of the leg through which torn muscle had herniated (Figs. 3a and 3b). All the tendons in the ankle region were intact but the medial collateral ligament of the ankle joint itself had been completely torn across and the lateral collateral ligament was partially divided. Continuity of the posterior tibial nerve remained but extensive haematoma formation within the sheath suggested that it had been stretched to some degree. The anterior and posterior tibial and peroneal vessels were completely divided with retraction of their torn ends.

The initial treatment consisted of the reduction of the dislocation but this produced no improvement in the vascular condition of the foot after 15 minutes. A formal operation was then embarked upon. The small leg wound was excised and the skin loosely sutured so achieving a satisfactory decompression of the anterior compartment. Inspection of the circumferential wound showed that only the posterior tibial artery was suitable for repair and so an end-to-end anastomosis was performed by Mr. Smith. This gave a good pulsatile flow of blood into the vessels and resulted in the oozing of blood from the distal end of some of the torn veins. After a very limited wound toilet and *debridement* the wound edges of this circumferential wound were loosely approximated and the foot was splinted

FIGS. 3a and 3b.—Medial and lateral aspects of foot in a case of near avulsion showing the extent of skin laceration and the presence of friction burns.

at right angles on a well-padded piece of Kramer wire. The use of anticoagulants was obviously precluded but the venous outflow from the foot was allowed to drain into the dressings, the estimated blood loss being replaced by transfusion.

Four hours after injury the patient was transferred to the pressure chamber where he breathed oxygen at 2 atmospheres pressure for 12 hours. For the next 80 hours similar treatment was given for progressively shorter periods at increasing time intervals. Skin temperatures were taken and showed that though the injured foot did not reach the temperature of the normal foot it was nevertheless considerably higher than the temperature of the chamber.

After the patient had been in the chamber for

FIG. 4.—Five days after near-avulsion of the foot with gangrene confined to the tips of the toes.

about 3 hours the foot became pink and remained so for about 40 hours afterwards. The change in colour was quite dramatic and there was a sluggish skin circulation. During periods of decompression, however, when normal air was being breathed, the condition of the foot deteriorated and it became cyanotic again. Forty-three hours after the transfer of the patient to the chamber the ooze into the dressings stopped and the toes became dusky and congested in spite of further intermittent pressurization and high-pressure oxygen. This suggested that the torn veins had now thrombosed. A venous obstruction type of gangrene followed which was confined to the toes. Fig. 4 shows the condition of the foot five days after injury with gangrene largely confined to the tips of the toes although the toes as a whole were somewhat ischaemic. A formal amputation was carried out through the tarso-metatarsal joints at which free bleeding was noted at the line of section. Healing after this amputation was satisfactory.

Today, over a year later, the patient is here for you to see. He has some ankle stiffness which is more prominent in the mornings and this is not surprising with this type of fracture dislocation. He also has a little residual hyperaesthesia of the skin of the stump but manages to wear a boot, the forefoot of which is padded with cotton wool. He is left with end-weight-bearing, earth feeling, normal sensation to the heel and a good heel pad, and this is better than the below-knee amputation that he would have had without the services of the pressure chamber. Furthermore he is at work. *At this point the patient was presented.*

FIG. 5.—Extensive degloving injury of leg in a girl of 13. The skin over the sole of the foot was also mobile apart from a tenuous attachment of the heel pad.

Extensive Degloving Injury

MR. W. SILLAR: This is the story of a 13-year-old schoolgirl who ran out of school at 12 noon on November 29, 1961, and was knocked down and run over by a lorry. She had a degloving injury of one forearm and both legs but only on the right foot was the circulation impaired. On this side the skin was degloved almost completely from midcalf to toes so that a hand could be passed freely between the skin and deeper structures all the way round the calf and the ankle. The skin on the heel and the sole of the foot was also mobile though the heel pad had a tenuous attachment and an instrument could be passed between the skin of the sole and the muscles of the foot which were also grossly damaged (Fig. 5). The toes were white and there was no sensation in response to pin prick or light touch on either the toes or the foot. She required resuscitation with 1½ pt. of plasma which were followed by 5 pt. of blood. When the anaesthetic was started about 3 p.m. her toes became grey in colour having been quite white until then. The skin was largely defatted, replaced and sutured so that there was a nearly linear suture line from the calf to near the big toe. The dead space under the skin was then emptied by gentle compression with wool and plaster and the patient returned to the ward.

At 6.30 p.m. the toes were still quite cold and grey and without any apparent circulation and so contact was made with Mr. Smith and the child admitted to the pressure chamber in the evening. The toes became patchily pink in the chamber but, in the morning, 10 to 15 minutes after decom-

FIG. 6.—Feet of an elderly woman three hours after being admitted as a result of prolonged exposure to cold. The oedema and skin discoloration are clearly seen.

FIG. 7.—Residual area of skin necrosis three weeks after cold injury, which healed spontaneously.

pression, became quite blue, quite cold and quite without sensation. Periods of pressurization were repeated and with this it was noticed that the periods of patchy pinkness became longer. Sensation returned about the second day. By the fifth day it was possible to leave the child out of the chamber, the foot remaining a dull dusky pink rather than the previous blue colour. There was also a little warmth in the toes.

She returned to the Southern General Hospital about this time with extensive skin loss which required skin grafting. She has now left hospital and returned to school. She walks fairly well, though curiously enough the foot which shows most swelling at the moment is the left one. At

about 10 days there was shedding of a complete cast of skin of the toes as happens sometimes after scarlet fever. Underneath was supple, pink, warm skin. *At this point the patient was presented.*

Cold Injury

DR. I. LEDINGHAM: This is the history of a 44-year-old woman of a vagrant disposition who spent one of the coldest nights last December sleeping on a bench in Kelvingrove Park. Her left fifth toe had been amputated in the Royal Infirmary following exposure in the winter of 1960. On this occasion she was admitted to Mr. A. B. Kerr's unit and found to have a cold, blue, insensitive left forefoot with bluish-black mottling of the skin. The right forefoot was also affected but to a lesser degree and all the pulses were palpable. On the left side only the femoral and popliteal pulses were palpable but the pedal pulses on this side may have been obscured by oedema. Fig. 6 shows the condition of both feet two to three hours after admission showing the area of skin discoloration on the left foot, a similar area of skin demarcation over the right foot and on the sole.

Six-hourly heparin was instituted immediately and the patient transferred to the pressure chamber. Within two hours only the tip of the fifth toe of the right foot remained cyanosed and some sensation had returned to the left forefoot. At the end of the next hour pinkness appeared over the lateral border of the left forefoot and later on that same evening only the left third and fourth toes remained doubtfully viable. Later some blistering appeared over these two toes. Pressure-chamber treatment continued throughout that night and during the next two days with continuing steady improvement until at the end of the third day treatment had to be terminated prematurely to allow another patient into the chamber. At this stage there was an area of skin necrosis on the lateral plantar aspect of the left forefoot about the size of a half-penny and some skin blistering over the left third and fourth toes. Fig. 7, taken three weeks later, shows this area of skin necrosis, the rest of the toes and distal part of the foot having become practically normal. Thereafter, the patient was returned to the ward and, apart from two short periods of pressurization, was treated with anticoagulants and local dressings. The oedema settled quickly and the left dorsalis pedis pulse became palpable shortly thereafter. The patient has attended outpatients several times and when seen two weeks ago had a tiny healing skin lesion as the only residuum of the original cold injury. This has given her no trouble at all and no grafting was required.

I would like to draw your attention to two points

of interest, firstly the rapidity of improvement in the pressure chamber and, secondly, the fact that we have been able to avoid any form of amputation or skin grafting.

Discussion

PROFESSOR DONALD: Dr. Richards, you have had a lot of experience of this sort of case, would you care to say anything?

DR. R. L. RICHARDS: Yes, thank you. I think that this question of cold injury is extremely interesting. There is probably less evidence here than in the other cases, however, that the results have been due to the pressure chamber. It is natural in cold injuries for the patients to go through a phase of extreme hyperæmia once they are in the recovery phase and I would be doubtful whether what Dr. Ledingham has shown us is any different from what would be expected during the hyperæmic phase of a cold injury of this degree.

It is extraordinary how little we really know about the pathogenesis of cold injury to peripheral tissues. There have always been two schools of thought; one, that it is the cold itself that produces the injurious effect, and, the other, that this is entirely a vascular reaction. The evidence that the injuries are due to cold itself is becoming less and less as time goes on. It is now pretty evident that you can chill not only tissues but whole living organisms down to extremely low levels and revive them without any evidence of tissue damage whatsoever. The general impression now is that cold injury is a vascular reaction associated with chilling or extreme cold in part of an organism the rest of which is alive and in which the circulation is active. The exact role of anoxia in this is again in doubt. During World War II, and since, the Americans found that anoxia certainly predisposes people to severe cold injury. At one stage in the war they ran into a very bad phase of cold injuries in their high-altitude pilots, and they found that this improved considerably when they provided additional oxygenation for these people without any other alteration in the environment in which the injuries occurred. There is, therefore, no doubt that anoxia is an important factor, and therefore any attempt to get additional oxygen to the tissues is to be commended. I think that the problem often is that, although the oxygen is there all right, the tissues cannot use it because they are chilled down to a level below which oxygen utilization fails to occur, so that, even if you provide additional oxygen in the blood and in the extracellular fluid, it is sometimes ineffective. Nevertheless the provision of oxygen to the extracellular fluid is very important because cold injury is undoubtedly associated with much exudation of

fluid from the damaged small vessels, consisting not only of ordinary extracellular fluid but also of serum and even some blood cells. If you can get more oxygen into this fluid which is actually bathing the tissues then at least it has an opportunity to produce a beneficial effect. One would like to have the opportunity in a patient who has two limbs affected of putting one foot in an individual pressure chamber and use the other as a control, but this is not a possible experiment as the oxygen would get into all the tissues and you would be unable to separate one foot from the other.

PROFESSOR DONALD: Thank you very much, Dr. Richards. Another application of the pressure chamber which Mr. Smith mentioned is its influence on anærobic bacteria. Have you any views on this, Dr. Scott?

DR. A. C. SCOTT: Increasing the oxygen potential in the tissues is obviously important in the treatment of anærobic infection. Even when the anærobic organisms have gained a foothold in the tissues their ability to flourish will be cut down, though I think it is important to remember that low oxygen tension is not the only factor which allows anærobic infection to occur in tissues. It is very important that these organisms which are very active biochemically and produce a large number of very potent exotoxins should not be given a chance to break down devitalized and necrotic muscle and to release their toxins. Low oxygen tension, of course, is not the whole answer and the use of the chamber in anærobic clostridial infections is only an extension of the other methods of increasing oxygen tension which have been advocated in treatment, such as local application of oxidizing agents or even the direct injection of oxygen into the tissues at the site of the infection. It is probably a very important supportive method of treatment but cannot and obviously should not be expected to take the place of excision of muscle, removing dead tissue which provides the ideal nidus for the infection, and the use of antitoxins and antibiotics.

DR. S. FLETCHER: We very often tend to forget that there are two sides to infectious disease, first the ability of the organism to grow, and secondly the ability of the defences of the body to deal with the infection. It may well be that increased oxygen tension in the tissue causes the phagocytes to go about their activities with a little more gusto. This idea would be amenable to experimental observation and I wonder if any work has been done on this—perhaps Mr. Smith could tell us.

MR. SMITH: No, I know of none. Most of the work on clostridial infections has been done with Brummelkamp working in Boerema's laboratory. I think that it has been done mainly by the assessment of the survival of guinea pigs to a standard

dose of *Clostridium welchii* and has not been related to the phagocytic activity.

DR. S. FLETCHER: I think it would be something well worth looking into. It would be relatively simple to do *in vitro*.

MR. SMITH: I quite agree.

PROFESSOR DONALD: Are you suggesting that if you had a case of overwhelming clostridial infection that this treatment alone might help to assist phagocytosis? It seems to me to be an entirely reasonable suggestion because operative intervention at this stage is completely out of the question.

DR. SCOTT: I think this question might possibly be more easily solved than one might think. It is said to be characteristic of the effluent from a typical case of gas gangrene that there are very few polymorphs in it, because they are rapidly killed by the toxins produced by the clostridia. If a patient with typical gas gangrene, clinically and bacteriologically, was treated with oxygen and found to have a very purulent exudate this would be evidence that increased oxygenation was helping phagocytosis and that the phagocytes are able to resist the clostridial toxins a little more readily.

PROFESSOR DONALD: I think that both in traumatic surgery and in septic abortion there might be a future in this. It should certainly be borne in mind. I remember from my experiences during the last war the prophylactic amputations we used to undertake for infected wounds in the absence of penicillin or other antibiotics. If this kind of treatment could be made sufficiently available on the field, it could make quite a lot of difference in the conservation of both limb and life. I would be rather interested to hear what the orthopaedic surgeons have to say about the future of this in conservative surgery.

MR. STEVENS: If I may say a word as a representative of the Orthopaedic Department. You have heard of the application of oxygen under pressure in the treatment of injuries of various types and we have experience of many other cases. I think it has an even wider application than has already been said because we have got to bear in mind that we are dealing not only with viability of skin and muscle and nerve but also with the viability of bone which is a very tenuous thing in many respects, particularly in the adult. The bone cell itself is in a precarious position buried in a piece of solid bone tissue and if it happens to be more than one-tenth of a millimetre away from a useful capillary then it is likely to die. So it is quite clear that extensive anoxia of bone could cause extensive death of bone and in this regard it may be that in the future we will see a more extensive application of this technique in, for example, the investigation of avascular necrosis of

bone such as follows fracture of the neck of a femur.

Another important application is in Volkmann's ischaemic contracture which is not very common nowadays but is obviously an outstanding candidate for this sort of treatment. Yet another one which is terribly important and does have some relationship to hyaline membrane disease in the premature newborn is fat embolism. This complication occurring 48 hours after injury can be of the pneumonic or of the systemic type. Quite clearly both of these types of fat embolism are very strong candidates for treatment by oxygen at a pressure of 2 atmospheres and we are just waiting for an opportunity to try it.

MR. A. D. ROY: What measurements, if any, have been made of tissue oxygen in these cases?

MR. SMITH: As far as available instruments in this area are concerned it is possible but by no means easy to do tissue oxygen. We haven't done this—I only wish there was a more easily available cheap apparatus for doing it.

PROFESSOR DONALD: No one has mentioned the question of burns yet. I would like to ask Mr. Smith if this kind of treatment might in any way assist in the treatment of major burns?

MR. SMITH: We have not had the opportunity to treat any of these cases. Possibly in certain cases of marginal viability of skin, some skin dies unnecessarily from the action of hyperaemic exudation fluid. One could imagine that the chamber could have some effect on this but we have not had any opportunity to test this. It would be a reasonable proposition.

MR. G. H. PARKER: Does the prolonged increase in oxygen tension have any adverse effect on brain tissue or on the cerebral circulation such as producing oxygen convulsions and so on?

MR. SMITH: We have almost two years' experience of this chamber now without any evidence of oxygen toxicity but then we are using 2 atmospheres pressure and this is probably well within the safety limits. Mr. Jacobson, a neurosurgeon, is here and he might be able to elaborate on this. We have not seen it.

MR. I. JACOBSON: If you do cerebral blood flows with the nitrous oxide method or with any of the more accurate radio-isotope methods, then by getting the patient to breathe pure oxygen and using the simple relationship between blood pressure, blood flow and peripheral resistance you will find a 10 to 15% increase in cerebro-vascular resistance—this has no effect on the patient. Now when this is done under a pressure of $3\frac{1}{2}$ atmospheres the cerebro-vascular resistance goes up by about 35% but again the significance of this is doubtful. However, volunteers breathing pure oxygen at $3\frac{1}{2}$ atmospheres did encounter difficulties

and did have oxygen toxicity. It is interesting that even at that high pressure level the arterio-venous oxygen difference between samples from the jugular bulb and the internal carotid artery increases—the explanation for this is difficult.

DR. A. FAIRLEY: Mr. Chairman, I would like to ask Mr. Smith if he is of the opinion that it is worth while putting patients on oxygen with a B.L.B. mask as a temporary measure in the absence of a pressure chamber. Are there any comparative figures which he could perhaps give us?

MR. SMITH: I think that if you admit the reasonableness of the pressure chamber then the next best thing is to go on to pure oxygen at 1 atmosphere. I think the answer is distinctly 'yes'.

MR. PARKER: Would there be any place for intravenous dyes to show if there is circulation in the affected extremity before putting the patient in the pressure chamber? If there is no communication at all then it would presumably be a waste of time putting the patient in the chamber.

MR. SMITH: Yes, that is true. We have thought about that but the answer becomes self-evident in a matter of 24 hours or thereby because the skin does not become pink at all. Obviously this treatment is useless unless there is a trickle of blood going in and, most important, the same amount coming out. An ischaemic leg with a venous block in addition to the arterial disease just will not improve.

PROFESSOR DONALD: Another obvious field of application here is already being explored in St. Thomas's Hospital. All malignant tumours are relatively hypoxic and become more radio-sensitive if you can increase their oxygen saturation. In St. Thomas's Hospital there is a different sort of pressure caisson in which the patient is put in at 4 atmospheres and exposed to the cobalt bomb. The oxygen saturation of the malignant tumour is increased from about 60 to 97% and this greatly increases its radio-sensitivity relative to the surrounding tissues. However, they have a lot of trouble from cartilage which is an anoxic structure.

MR. H. I. TANKE: Mr. Smith, do you have any views on the treatment of non-traumatic ischaemia? Is the chamber of any value in the ordinary ischaemic foot?

MR. SMITH: Well, I think that this is a difficult thing to judge. As far as one can say the vast majority of patients with ischaemic neuritis do improve in the tank. This is one observation which I think is significant. We now have several patients in whom treatment was continued for over a month and in whom the ischaemic neuropathy has subsided enough to allow the patient to carry on his everyday existence with the limb still intact,

where I would have thought previously that these particular cases would require a major amputation. We have also tried some five or six patients with indolent thromboangiitic ulcers of the foot of up to a year's duration and have healed all these by prolonged treatment in the chamber. Again, we think this is a positive indication and that some good is being done. We have the impression that some cases who would have required major amputations have got off with minor amputations. We also have some patients with arterial emboli where treatment in the tank has saved a major part or all of the limb. All this, as you will appreciate, is a very difficult thing to assess.

DR. A. PRINGLE: You say, Mr. Smith, the oxygen is dissolved in the plasma. When you say that a limb becomes pink does it mean that the oxygen is being transferred to the haemoglobin?

MR. SMITH: I don't know exactly what the state of the capillary bed and the small arterioles has been, but obviously to get a pink skin you must have enough oxidized haemoglobin in the capillary layer, apart from the skin plasma. I am merely saying that the increased oxygen saturation of the plasma provides a better tension gradient for diffusing out into the cells at jeopardy from anoxia. I don't know how much flow you need through a limb to give a pink limb. When you are cold, the answer is very little indeed, and one must not equate pinkness with flow. Pinkness in the absence of extreme cooling indicates that there is flow.

PROFESSOR DONALD: But you can also get the oxygen into the cells without the medium of haemoglobin.

MR. SMITH: If you get a foot staying pink whereas before it was dusky black or blue-black then there must be transport of oxygen by the plasma element alone to keep the haemoglobin from reducing.

PROFESSOR DONALD: But obviously there is cartilage at stake too which has survived and this could not have been done so through the medium of haemoglobin, could it?

DR. RICHARDS: I think that part of the point in the management of these cases of trauma and of cold injury is that the toes are blue because there are red cells which have given up their oxygen trapped in the small cutaneous vessels. In experimental cold injuries certainly at this stage there is still a flow of plasma going on. If you get oxygen into this plasma, it flows into the tissue and the red cells become oxygenated *in situ* as it were and this is why they become pink. As the tissue oxygenation improves the major circulation may restart.

MR. PARKER: Are there any contra-indications to pressure therapy, for example, a patient who has chronic carbon dioxide retention?

DR. FAIRLEY: I think one should take measures to ensure that the carbon dioxide retention was corrected by the use of buffers and so on and perhaps by the use of adequate respiratory measures.

PROFESSOR DONALD: I should have thought that adequate respiratory ventilation under such conditions would have blown off the carbon dioxide. Would this factor of supersaturation with oxygen worry you?

DR. FAIRLEY: Well, it would perhaps produce apnoea in the presence of carbon dioxide retention and one would perhaps have to think of forced ventilation.

MR. SMITH: I think what you have in mind is the type of patient who has chronic emphysema and you interfere with his carbon dioxide drive. If, however, you are faced with such a patient requiring this sort of treatment in an emergency I think you might well carry such a patient through successfully, if you have adequate biochemical control in the chamber as, of course, we have.

PROFESSOR DONALD: Little has been said this morning about biochemical control in the chamber but I can say that in the cases of the respiratory distress syndrome in infants, there is biochemical control throughout, coupled with the pressurized oxygen treatment and one could not have the one without the other.

MR. TANKEL: I think it is worth while pointing

out that if you happen to be dealing with a patient in whom there is a waterseal drain into the chest you must clip it off as near the chest as possible otherwise the fluid in the waterseal drain will be pushed into the chest.

MR. SMITH: Yes. There is a lag in stabilization between the intra- and extra-pulmonary pressures but not much and now we can quite easily compensate for it by putting on suction via a small pump on the chest drain, providing the airway is not blocked.

PROFESSOR DONALD: Well, we've had a most exciting morning and there are many angles of this we can think about for days to come. Would you care to say anything in conclusion, Mr. Smith?

MR. SMITH: Thank you. You have heard the gamut of cases we wish to deal with and now perhaps just to add a final note to this discussion it is obvious that one should think about a service with pressurized ambulances if this treatment should become more universally adopted. Glasgow now has its first pressurized ambulance with a portable pressure chamber which can be taken out and left in a hospital for continuing treatment while a spare is loaded into the same ambulance which can then go back on the beat again. This should be of value in the treatment of carbon monoxide poisoning but its use for transport of patients suffering from various forms of anoxia is likely to follow.