clotting. You can still activate clotting without platelets. The only protein in the clotting chain that is sensitive to surfaces is Hageman factor. It is sensitive to the lipid monolayer formed around the bubble. The centre of some of the lipo-proteins in the blood become denatured and adhere to the bubble and change the polarity so that there is a lipid monolayer around the bubble surface. That may be the link that gets Hageman factor activated. The fact is that you will find intrinsic clotting activated by bubbles and as far as we know the only component of the clotting system that is sensitive to bubble is Hageman factor. It may not be the surface per se which causes it, it may be the lipid layer that forms from the lipoproteins around the bubble.

These briefly described processes can be altered by agents which interfere with one or more of the components of the inflammatory reaction which has been triggered by bubbles in the blood or by tissue injury caused by gas expansion or by local ischaemia.

THE TREATMENT OF DECOMPRESSION SICKNESS

AA Bove

The treatment of the diver or aviator with decompression sickness (DCS) should always be based on recompression therapy. Recompression is the most important modality, but for the best results it should be combined with oxygen, adequate fluid therapy, corticosteroids, other anti-inflammatory drugs and, in selected cases, anticoagulants. When no chamber is readily available, the other measures used for therapy, even when successful, must be considered as inferior or palliative. Recompression treatment should always be provided even if a significant delay is anticipated prior to recompression.

RECOMPRESSION

Standard recompression therapy can still be based on the somewhat artificial, but practical division of DCS into minor and major types. Minor DCS involving joints is treated with a US Navy table 5 or equivalent [60 ft (18m) intermittent air/O_2] lasting two to two and a half hours. Major DCS is treated with a US Navy table 6 or equivalent [60 ft (18m) intermittent air/O_2] lasting about five hours. Other useful tables such as the Comex 30 [100 ft (3Om) intermittent air/O_2 and air/50% Nitrox] should be made available when a deeper treatment depth is required, and saturation is impractical. Special adaptations of the 60 foot oxygen table can be made for a monoplace chamber where pure oxygen is used to compress the chamber. In-water recompression can used with extreme caution and careful planning, when nothing else is available and the diver's life is threatened.

OXYGEN

The next most important treatment is oxygen. The use of 100% oxygen at 60 ft, 50% oxygen at 1OO ft and 100% oxygen at 1 ATA have all been established as useful therapy for DCS. Two benefits accrue from oxygen: ischaemic tissues may be provided with enough O2 to prevent cell death, and inert gas will be removed from bubbles and replaced by oxygen which will be metabolized. Although administration of oxygen appears simple, several precautions should be mentioned. The oxygen must be supplied with a tight fitting mask to ensure that the necessary concentration is provided. An overboard dump must be used in multiplace chambers to prevent oxygen build up in the chamber atmosphere. The hazard of fire with high pressure oxygen must be borne in mind. Oxygen should be provided to divers with DCS, while at one ATA awaiting transportation and during transit to a treatment chamber. Oxygen toxicity will not be a problem at one ATA unless oxygen breathing is prolonged. (eg. four or more hours continuously). At 60 ft on 100% oxygen however, signs of CNS oxygen toxicity must be watched for diligently.

FLUIDS

Fluid therapy is the next treatment consideration. The diver with DCS is likely to be hypovolaemic because of plasma loss into tissues, and may be dehydrated because of inadequate fluid intake, excess fluid loss or vomiting. The resulting hypovolaemia and haemoconcentration can aggravate the microcirculatory perfusion defect associated with DCS, and produce permanent tissue injury. Thus an important goal of therapy in DCS is to prevent haemoconcentration and hypovolaemia. The best replacement fluid is a crystalloid solution such as normal saline. The use of plasma or dextran does not appear to provide additional benefit. Also these solutions can result in fluid overload which is not readily corrected. Conversely, an overload of crystalloid is easily dealt with by giving a diuretic. Enough fluids should be given to maintain urine output at 100 cc/hour. Calculations based on the standard estimator for fluid deficit can be used successfully. Be sure the patient has normal lower urinary tract function, since bladder paralysis from cord injury may result in urinary retention and an improperly estimated fluid need. One should start an intravenous line in divers with DCS whenever practical and infuse normal saline to reverse or prevent hypovolaemia. One should monitor the urine output and blood pressure. It is essential that the state of bladder function is known.

CORTICOSTEROIDS

Corticosteroids should also be used in treating serious DCS. The glucocorticoids interfere with the inflammatory process. In DCS where the inflammatory reaction is inappropriate and detrimental to the diver, aborting

inflammation is a useful therapeutic goal. Steroids block the action of vasoactive mediators on post capillary venules, they prevent prostaglandin synthesis, stabilize the leucocyte lysosomal membranes, and have other actions which interfere with inflammation. The diver with serious DCS, such a spinal cord injury, or with brain injury from a pulmonary overpressure accident should be given high dose steroids for several days along with other therapy. Dexamethasone 10 mg IV initially followed by four to six mg every six hours for three days will provide optimum benefit. There will be minimal steroid side effects from this regimen.

OTHER ANTI-INFLAMMATORY DRUGS

Non steroidal anti-inflammatory agents such as aspirin, indomethacin, isobrufen also may be useful in DCS treatment. The important actions of these drugs involve platelet inhibition, and blockade prostaglandin synthesis. Prevention of platelet aggregation will reduce the possibility of intravascular clotting and reduction in thromboxane release will prevent local vasoconstriction and subsequent ischaemia. Excess aspirin can block vasodilator prostaglandins. Thus doses of aspirin in excess of 600 to 900 mg/day may have detrimental effects. A reasonable dose is 600 ml/day for several days after the DCS injury.

ANTI-COAGULANTS

Anti-coagulants should not be used routinely but reserved for severe DCS cases. Heparin in full anti-coagulant doses can be used when intravascular coagulation is a major component of the illness. Normally, this type of anti-coagulation is not needed in DCS therapy, and anti-coagulation with heparin is reserved for complex, life threatening cases of severe DCS. Low dose heparin (5000 units every eight hours) should be considered in DCS cases with spinal cord injury because of the risk of venous thrombosis and pulmonary embolism from prolonged immobility. Late anti-coagulation (4-6 days following injury) with anti-vitamin K agents (warfarin) may also be useful in preventing pulmonary embolism in severe, prolonged DCS cases. Nevertheless the occurrence of haemorrhagic areas in the spinal cord in DCS makes one very chary of using anti-coagulants during the acute phase of DCS.

FUTURE DRUGS

Other agents may emerge which will be useful adjuncts to pressure and oxygen therapy of DCS. These include antihistamines, anti-kinin agents and specific prostaglandin inhibitors. These drugs will be developed for use in inflammation occurring for other reasons. As the process is the same regardless of the trigger, they can be used for treating DCS.

TREATMENT OF DCS

The full battery of treatment which includes pressure, oxygen, fluids, steroids and aspirin should be provided for all divers with CNS involvement from DCS. Anti-coagulants and other anti-inflammatory drugs should also be considered in difficult or prolonged cases.

When a chamber is not readily available, all therapy except recompression should be provided. Even then delayed recompression therapy should be carried out to prevent permanent residual injury.

Question:

Why do you say that 500 ml of colloid may be too much? That is only 10% of the blood volume.

Dr Fred Bove

We are not talking about one unit of dextran of 500 ml . If you are going to make a commitment to treating a diver with dextrans, you may use five or six units or more, in the same way that you would use serum. You have an injured diver somewhere at a remote site and you tell the EMTD, Emergency Medical Technician (diver), to start an IV and to run in fluids until the patient is stable. You may now have a diver at a remote site, loaded with dextran, who may be in pulmonary oedema, and there is nothing you can tell the EMTD to do to relieve the pulmonary oedema. You could tell him to do a phlebotomy but that really complicates the issue. Whereas if you have some IV lasix in the kit, and if he has been given too much saline, then all you have to do is tell the EMTD to give him 20 mg of lasix and watch him. Saline is cheaper, it is usually more available and it is easier to administer from the point of view of worrying about allergy and things like that. I would recommend saline, because you get the same basic effect. You get the haemodilution effect with saline. It does not seem to warrant the use of the more expensive and more complicated fluid if you can get the same response from saline. Most of us will start an IV with saline and run it. If the patient is getting better, fine. If he is not getting better, then we go back and reassess. Then we decide whether we want to use a colloid or not. The first response should be to get a needle in the vein and start a drip. The drip ought to be a crystalloid solution rather than a dextran solution.

Question:

Is there a late resorptive phase to decompression sickness as in other illnesses with increased vascular permeability?

Dr Fred Bove

I do not know. You would expect that there would be

because the process is the same. It is a breakdown of the normal vascular integrity, which allows protein to leak. As the fluid gets pulled back into the vascular system you should, in decompression sickness, find that phase. A problem is that we do not have enough of a population of those kind of patients to start doing good clinical trials where we can observe them. There are some efforts in the States to get some of the centres together to try to decide on a couple of protocols so that we can make some sense out of the anecdotal information that everybody has. There have been no clinical trials and there are probably only six or seven a year of the really complicated cases where you can gain some experience. I think you are right, theoretically, because the process is the same. We ought to see a resorptive phase, and yet that has not been described so far. I think only because there have not been enough patients to make the observation.

Question:

Is there a possibility that the aspirin and steroids could cause haemorrhage?

Dr Fred Bove

There is a chance that you can get excess bleeding if you use aspirin and steroids. But that seems to be less of a problem than if you use heparin. In the States most of us would rather hold heparin in reserve and use it for a complicated, difficult case, rather than give it as an initial bolus for the first round of treatment.

Question:

Should not pressure always be part of the treatment of decompression sickness? After all pressure will reduce the size of the bubbles and presumably reduce the surface area activating clotting.

Dr Fred Bove

The point that you made about pressure reducing bubble size and then reducing the surface that activates things is valid, I think that pressure still ought to be on the top of the list. Every effort should be made to use pressure. In the States we insist on not bypassing the hyperbaric chamber for the sake of all the other things. You can use the other things on the way to the hyperbaric chamber. The chamber has to be on your list of things to use. It is still important. It is very important when you have somebody from a deep depth. If you have a blowup from two or three hundred feet, there is no way that you can save them unless you put them back under pressure. You can not stop bubble formation any other way. These people have such a large gas load that they will soon be like a Swiss cheese without pressure. You have got to get them into a chamber and back to pressure within minutes to get them to survive. Pressure is always important and should always be on top of the list.

THE EDMONDS UNDERWATER OXYGEN TREATMENT FOR DCS

John Knight

If you follow the tables closely, as do the USN, serious decompression sickness has an incidence of about 7% of the cases (Table 1). The USN actually normally dive more conservatively than the tables as supervisors add on an extra increment of both depth and time.

TABLE 1

PRESENTING SYMPTOM OF DECOMPRESSION SICKNESS. US NAVY
From Rivera (1963) 900 cases

Cerebral (including inner ear)	6.4%
Spinal	0.2%
Cardiorespiratory	0.4%
Pain only	82.7%
Other	10.3%

However, if you treat sports divers who have deep water to dive in, the picture is very different. (Table 2). Edmonds worked in Sydney and Erde in Hawaii. They treated 100 people and over 50% had serious decompression sickness.

TABLE 2

PRESENTING SYMPTOMS OF DECOMPRESSION SICKNESS. SPORTS DIVERS
From Erde & Edmonds (1975)

	100 cases
Cerebral (including inner ear	33%
Spinal	13%
Both Spinal and cerebral	5%
Cardiorespiratory	1%
Pain only	33%
Other	15%

TABLE 3

HOW TO AVOID DECOMPRESSION SICKNESS

Always do “no-stop” dives
Stay well within the tables
Know your maximum depth
Have an accurate depth gauge
Watch your time
Ascend at 18m a minute or slower
Always do a stop at 5m

If you must do a decompression dive decompress for the next depth and time

Use a shot rope
Have extra air on the shot rope

Do not fly or cross mountains for at least 12 hours