was more a consequence of the small sizes of the air emboli, which produced marked local reactions but little CNS destruction, than of the non-specific initial treatment. The delay in contacting any recompression unit might have had a less happy result if it had occurred in another case. In Case 2 the delay in seeking treatment could have been serious had the surgical emphysema increased more rapidly and severely at home. It is hoped that trained divers will be alert to the possibility that the diving troubles they learn about may actually occur in themselves or their buddies. They should INSIST on others taking notice of any possible diving relationship to their, or their buddy's, troubles and NEVER EXPECT A NON-DIVER TO UNDERSTAND DIVING-RELATED DISABILITIES!

SPUMS 1981 SCIENTIFIC MEETING

THE DECOMPRESSION DISEASES
PART ONE

David Elliot

I think it is important to include both types of decompression illness together, because there are many occasions when the differential diagnosis between the two is not only difficult but really, from a practical point of view, rather unimportant. So, instead of breaking it up into pulmonary barotrauma and decompression sickness, I am going to lump them together, call them decompression illnesses and then deal today with pathogenesis and presentation, leaving treatment for tomorrow.

I am aware that the group here has probably had quite a lot of this in past years. I am also aware that some of you are not so familiar with diving accidents. So as a matter of policy I will go for some fairly elementary stuff, even though this will be repetition for some of you. Repetition does not do any great harm and helps to reinforce existing knowledge. Also, coming from North-west Europe, I may well have a different slant on decompression illnesses to some of the previous speakers who have been to SPUMS meetings. In a number of treatment seminars over the past few years, there have been many arguments not only between the Anglo and the American groups, but particularly between the Anglo-American and the French groups of diving doctors. I think that it is very important to air differences, because they emphasise the fact that we certainly do not know everything about the subject. Indeed if you meet anyone who says that he knows all about the subject, the only thing that you know about him is that he does not know anything about

The dysbaric illnesses comprise two illnesses, pulmonary barotrauma and decompression sickness. Pulmonary barotrauma is the illness due to the expansion of gas inside the chest, whereas by definition decompression sickness is the illness which arises from bubbles from gases that have been dissolved in the tissues. The first essential point of today is that as diving emergencies both comprise one syndrome, the decompression disorder and to subdivide these into minor and major, or serious, varieties of the disorder is useful in retrospect, but in practicality I would suggest we avoid it, because every single such instance must be regarded as an emergency, until you have got it sorted out.

PULMONARY BAROTRAUMA

Two reminders about Boyles Law, which I trust everybody knows inside out. The compression of the air in an inverted bell jar in water is exponential. The rate of expansion is greatest in the last few feet.

When a men does a buoyant ascent from a submarine he comes up very fast. The stole of his survival suit is inflated and venting. The enormous trail of bubbles gives some idea of the rate of gas expansion that can occur during a rapid ascent. He comes up between five and eight feet per second. The slide is of a submarine escape instructor coming up from a submarine at 600 feet. That incidentally is a compressed air dive, a twenty second compression to 600 feet, 4 seconds bottom time and then a minute and a half back to the surface. The slide gives a really remarkable illustration of the amount of gas which is vented. The picture was taken about 40 or 50 feet below the surface.

Causes of Pulmonary Barotrauma

What are the causes of pulmonary barotrauma? Bear in mind that an overpressure of a mere 80 mm of mercury is quite sufficient to blow a set of lungs. The first and most common cause, failure to exhale during rapid ascent, is something which we should be able to avoid by good training of both divers and submarine escape trainees.

The second most common cause is some form of local pulmonary pathology, that causes a retention of gas in the lung. The various causes include tuberculous glands, cysts, some sort of lung pathology which on the whole can be detected by X-ray. One of the things which therefore will come out when we are discussing fitness for people for sports diving, is whether or not an annual chest X-ray should be mandatory. It certainly is for professional divers. But the point about the chest X-ray is that of course it is a very gross test and there are many reports of people who have had serious pulmonary barotrauma who have breathed out properly during their ascent, and who have had perfectly normal chest X-rays within a very short period before that ascent. There was the Australian report of increased compliance of the lungs of such people, which may have been post-hoc rather than propter-hoc.

The third case in the normal individual making a normal ascent, is airway collapse. You can demonstrate that with fast rates of flow the airways will naturally collapse at the equal pressure point, and therefore dam up the peripheral gas. This is something which one can not train the diver to avoid. One can not detect it with X-rays. But for it to happen one has to come up extremely fast. However,

even in an emergency, making a buoyant ascent, you still have quite a lot of control over your rate of ascent. All you have to do is to come up with your legs astride and your arms out instead of keeping yourself streamlined. It is remarkable how much drag that will cause to slow you down. If you think you are coming up too fast, or are out of control, or even if you are breathing out properly, stick your arms and legs out and it will really slow you down.

Pathophysiology

The pathophysiology of decompression barotrauma we can deal with fairly quickly. Pneumothorax obviously, possibly tension pneumothorax, which is very occasionally bilateral. On the whole, pneumothorax on its own is unusual. Much more commonly the gas goes along the peri-vascular sheaths, causing mediastinal emphysema, pneumopericardium and retro-peritoneal gas. I have one slide which is a very nice view of the upper pole of the right kidney neatly outlined with alveolar air. The gas can spread. The diagnostic point is gas subcutaneously in the anterior triangle of the neck. If it is not there, he may still have burst a lung. But if it is there, then we can say quite positively that there has been a episode of pulmonary barotrauma.

However, the most important thing is the arterial air embolism. I prefer to use arterial gas embolism, because I am not dealing just with compressed air divers. It has been shown quite reasonably well scientifically that the gas tends to enter the pulmonary capillaries as the diver takes his first breath on reaching the surface. The gas is dammed up in the lungs until that particular moment. The clinical picture which follows is just a few seconds of circulation time from the lungs to the brain before the guy goes unconscious. The gas enters the pulmonary capillaries and will distribute by buoyancy, which also has been well demonstrated experimentally, to the carotid arteries and to the vertebrobasilar arteries. I do not think that particular point has been fully appreciated. It has been shown experimentally that vertebrobasilar embolism will cause cardiac irregularity as a reflex. There is of course the possibility of gas embolism to the coronary arteries. It is not surprising that one of the presentations of pulmonary barotrauma is sudden death. It is a cardiac arrest. Work is now going on at the Submarine Escape Training Tank at HMS DOLPHIN to see whether or not one can get a defibrillator that will work at pressure with everybody in that chamber soaking wet.

Now what I have said has been dramatic so far. There are two reports that are both worth attention. In 1964, or thereabouts, the US Navy did some routine chest X-rays of people who made normal buoyant ascents in the Submarine Escape Training Tank. They found that in about 1% of those cases there was evidence of mediastinal emphysema, in perfectly normal people after perfectly normal ascents. The Swedish Navy did some work in their Submarine Escape Tank which showed a 3% incidence of abnormality in otherwise normal ascents. This study is not quite as acceptable because the abnormality is an electro-encephalographic record, but it was well presented. It is clear that it is not an all or none phenomena. It is a condition in which there will be varying grades of cerebral embolism.

In 200,000 man ascents made in the RN Submarine Escape Training Tank over some 20 years, there have been about 88 cases of pulmonary barotrauma, including five fatalities. That is making ascents of from 30, 60 and 100 feet and gives a prevalence of about 1 incident to 2,200 ascents. Those were all buoyant ascents of people who were breathing out correctly and who had normal chest X-rays.

Manifestations of Cerebral Gas Embolism

The manifestations of cerebral gas embolism can happen within a second or so of reaching the surface. The first point is no significant latency. However there can be delayed onset. There was one famous incident at HMS DOLPHIN where a submarine rating finishing his day’s training, actually went on the Gosport Ferry before he found that he was getting giddy, feeling pretty ropey and losing power. Luckily the skipper of the Gosport Ferry was an ex-submariner and knew what was happening. He turned the ferry around and took it straight to HMS DOLPHIN, where the man was immediately recompressed successfully. How can we explain that? I think it is quite simple. If you can imagine that the retained gas in some terminal alveolar unit is rather like a balloon that has been blown up and it may not burst right away. It can stay there under tension. So very rarely you will have a delay in onset.

When DJ Kidd, who is a Surgeon Captain in the Canadian Navy and I were writing a paper together, we found that we were disagreeing quite vehemently, to put it mildly, on the presentation of gas embolism in diving. My basic training at that stage was in submarine escape buoyant ascent and his was in sports scuba diving. It seemed to us that there was a quite distinct difference. In sports scuba diving the onset was not nearly so often as dramatic as it is in the submarine escape training tank. So you may see the dramatic presentation that I described but just because it is not dramatic and immediate does not mean that it is not pulmonary decompression barotrauma and gas embolism.

Of the 88 cases that happened in the 20 years at HMS DOLPHIN, there were 65 with central nervous system manifestations, 30 of them presented as unconsciousness. (Is this relevant to the 5 pm game we are all playing?) 20 of them were disorientated, which included very minor degrees of disorientation, a slight feeling of “not-with-it-ness”. The fact that the guy is just feeling a little bit giddy and may be walking a little bit asymmetrically is quite enough to put one on guard that he is developing some cerebral manifestations of pulmonary barotrauma. Much easier to diagnose are the cases of paresis, of which we had 15. Five were hemiparetic and six were monoparetic in the arm and four monoparetic in the leg, so it can be pretty discreet.

I would like to describe one of those for you as I remember this particular case well. He was just doing an ear run. Now this is a dry pressure chamber run, not even in the water, with a normal diving rate of descent, with an occasional stop as people try to clear their ears, down to 100 feet. As soon as you get down to 100 feet, you stay there for about four minutes and then bring the chamber back to the surface at the normal diving rate of ascent. Theoretically there should be no problems in decompressing at that rate of ascent in a dry pressure chamber. Yet when this particular individual got out of the chamber he found he could not slip one arm into the sleeve of his jacket. I think that is an important case. I would like, although we could never prove it, to make one particular point. I could not prove it in this individual, but we know it is true in others. Some novice divers get all het up about the ears, how you have got to keep ahead of the pressure as you go down. They get a little bit of a thing about always clearing the ears. But the novice diver coming up gets a full sensation in the ears as the ears start to push out the gas from the Eustachian tubes and one or two idiots will go and clear their ears on the way up. One cannot prove that this is a cause of pulmonary barotrauma and gas embolism, but it is a possibility.

Diagnosis of Cerebral Gas Embolism

Bubbles in the retina are said to be diagnostic in gas embolism. This I have never seen. Neither have I seen another so called diagnostic sign. This is called Lieberhastner's sign, which I have never even heard of until I saw it in somebody else's textbook and it is a white mottling of the tongue. I believe that to be a load of rubbish only because I have not seen it. Much more important are the dysphasic and visual manifestations, the slowing of speech, the tunnel vision and I think particularly serious, is the diver who says he is going blind. I know of only one case of such a diver with gas embolism who has actually survived. The three that I myself have seen, who said that they were going blind, all subsequently died. So to me, if someone after an ascent says that they are going blind, it carries a pretty frightening prognosis. However, Jefferson Davis presented a case at the last course I was teaching on, of a man who went blind but recovered. So if you do go blind after your next dive, do not worry, I am sure you will be one of the survivors.

Pathology

Of the five fatalities three were immediately fatal. The various causes I have already hinted at. No one can tell whether respiratory centre embolism, or vertebrobasilar embolism causing reflex arrhythmia or a coronary embolism has occurred. All one can do is to resuscitate to the best of one's ability and see if you can pull him through. Two of the deaths were delayed, one about twelve hours and the other about thirty-six hours. Both had gross cerebral oedema at autopsy, which could have been due either to the bubbles or to the hypoxic episode during treatment. But their cerebral oedema is an indication, I believe, for giving corticosteroids early in serious cases of pulmonary barotrauma. However that is not generally accepted. Because of my personal experience I think it is a good idea to give all CNS cases of gas embolism from pulmonary barotrauma a corticosteroid. However, not everybody would agree.

In one case a post-recompression X-ray showed mediastinal emphysema and some large cysts in the base of the lung. However, these cysts were not the cause of the man's pulmonary barotrauma as he had had a normal X-ray three days before making the ascent. They were the result of the barotrauma.

I would emphasise that in order to get pulmonary barotrauma you only need to have taken one breath of compressed gas, one single breath and the ascent need not have been from a depth greater than 5 or 6 feet. We have on record one CNS case in HMS DOLPHIN which was from a special tank in which there was only 6 feet of water. Now, that is important, because you will find a number of diving superintendents and other experienced divers who, after a person has become paralysed, will say to you, "Look, that can not be the bends - the guy was not down long enough". That may be perfectly true, but it could most certainly be gas embolism.

We do find a tremendous number of mixed cases in which both pulmonary barotrauma and decompression sickness are present in the same individual. It is really much more common than is recognised and I shall hypothesise about this a bit more later on. I would remind you that barotrauma and embolism can follow any dive. There is a habit in some dive teams for the dive masters to stay at the surface and snorkel down keeping their tanks ready for an emergency. I think this should be discouraged for I know of one fatality at least. This diving instructor went down to his scuba class and while he was down there took a puff from an octopus. He then forgot that he had taken a breath of compressed air and went back to being the snorkel diver that he had been all that morning. So he surfaced without breathing out at all and he was dead within a few seconds of reaching the surface. This happened in the Cayman Islands only a few months ago. Snorkel divers should really be discouraged from coming down and grabbing a mouthful of gas from a diver.

DECOMPRESSION SICKNESS

Decompression sickness requires a dive of sufficient depth and duration to take up a sufficient volume of gas which will cause bubble formation when you come up again. In pulmonary barotrauma, the bubble is alveolar gas whereas it is dissolved gas in decompression sickness. The damage begins during the decompression in barotrauma, whereas there is significant latent period in decompression sickness. The significant bubbles are intra-arterial in barotrauma and are intravascular, though everybody emphasises the intravenous bubbles, in decompression sickness. The effects are cranial, in air embolism, while in

decompression sickness, not only can you get cerebral effects, and some brain stem effects, but you get spinal cord manifestations. I know of no case of pulmonary barotrauma which has gone on to paraplegia. That is a dogmatic statement and I will stick with it.

Manifestations

The manifestations of decompression sickness, I mentioned latency, most things will happen inside twelve hours. If anybody comes to you with symptoms that started 36 hours after the dive, that is not decompression sickness. Bear in mind that I said onset. Divers may suppress manifestations and quite frequently do, but when you get an accurate history you will find that the trouble which they may not report for 48 hours after a dive, did in fact begin within that 36 hour latent period. I will try and stick with air diving, but you can well imagine in the deeper dives of professional divers the latency if often reduced to less than zero because problems occur actually during the ascent.

Perhaps we should touch on the causes of decompression sickness. I would just like to give you some thoughts. The first is failure to adhere to the tables. I think that is perhaps the most common. Many people do get away with it. It is just like Russian Roulette, it may catch up with them after a while. This happens to the semi-professional diver, such as the abalone diver who has started work at the beginning of the season, picking up the shells at maybe 60 to 100 feet, fished it out, and then gradually during the season works down to 120 to 150 feet. This is normal seasonal practice in this type of semi-professional. The trouble comes when the man, for some reason, takes a few weeks off in the middle of the season. When he goes back to work he gets a spinal hit on his first dive, back at 140 feet. I have seen two cases like this. The reason for this is the adaptation of divers who dive every day develop to decompression sickness. It may be that you get the gas nuclei all being used up or something like that, but let us just put it down as. being a phenomenon that exists. These people may get away with ignoring a diving table. The most common cause nevertheless is failure to adhere to the existing tables. I think the US Navy air tables are as good as any. However I prefer the Royal Navy tables because they are about 5 minutes more conservative on most bottom times. Really it is the Jesus factor that counts when you are working out the table. How accurately you have estimated the depth. Do you take the next deepest depth and the next bottom time? It is these things that make the tables safe. Certainly if you dive the 180 feet for 20 minutes table exactly to 179 feet for 19 minutes you will get a percentage of bends in the diving population. There is no doubt about that because we did it. So the tables are not intrinsically safe. But they are safe as normally dived by the normal kind of diver.

The second cause of decompression sickness is inadequate tables. That does not apply to people like yourselves and we are really thinking here of Helium bounce diving, where there are a lot of lousy tables. There is really no excuse for people diving using compressed air not to use competent tables, if they use any.

So it is the third cause that I will bring to your attention, individual idiosyncrasy. We can think of the common things such as obesity and all those other things which might well affect gas uptake and elimination. But there is no doubt that the normal Gaussian distribution applies to divers as well as to everyone else. There are people who can do 100 foot dives and come to the surface with no problems. However, I have seen a knee bend occur after a dive to 100 feet for four minutes and respond to treatment. So individual idiosyncrasy can be extreme. The mere fact that a guy has adhered to the table does not mean a thing. If he has got the symptoms of the bends, he has got bent, because he has been under pressure.

To sum up the three common causes of decompression sickness are:

1. Failure to adhere to diving tables.

2. Inadequate tables (usually helium bounce diving tables).

3. Individual idiosyncrasy.

Two less common causes are:-

4. Flying, or ascending to altitude on land, after diving.

5. Diving in mountain lakes.

Presentation

As I said right at the beginning, the classification of decompression sickness into mild and serious can be misleading. If you look at the work which Tony Slark did in the Royal Navy and has also been done by Rivera in the USN, reviewing the presentations of decompression sickness, you will find that a significant percentage of cases present with a limb bend, because that is the most excruciating manifestation. They have in fact got a more serious manifestation, and therefore do in fact belong to the serious category notwithstanding the fact the presenting symptom is a limb bend. In the UK, or rather in the North Sea, we now tend to say that all presentations of decompression sickness should be regarded as an emergency. Just to make sure we are not missing anything, we like to treat everybody, even the simple limb bends, as though they were a serious case of decompression sickness. Because it is far better to give people a table 6 and be confident rather than give people a shorter table and treat them inadequately.

There are various presentations of the so-called minor cases, some of which do not require treatment, which I think can be regarded as warning signs. Anorexia, fatigue and malaise are very significant as warning manifestations of the onset of decompression sickness. Fatigue in the legs is well described. Any diver who, when back on the

surface, gets anorexic must be observed very, very carefully. The “niggle” is another term that we use in Europe, the Americans have adopted it and given it a slight difference in definition. We used to say that a niggle is a limb pain that starts but within ten minutes is already beginning to get better. That is a warning that something else may well happen. If you do not know where the nearest chamber is then you had better find out. On the whole, providing the manifestations start to get better and then disappear altogether within ten minutes, you can regard a niggle as a narrow miss.

Limb bends have been sufficiently well described in the past. Just to refresh your memory, the pains are in or around the joints. They can be in the end of one of the long bones, it does not have to be necessarily in the actual joint line. They can be just a niggling sort of pain or they can be agonising. They can be rending pains with the diver rolling around in agony. Then of course the diagnosis is very easy. One person, who had had osteomyelitis in one limb, got decompression sickness pains in the other limb at a later date, and said the two were surprisingly similar. Of course, unless you have had osteomyelitis yourself that does not really help. The synovial joints are the ones that are affected. I think that every single synovial joint except perhaps the temporo-mandibular joint, has been described as the site of pain, certainly joint pain in the sterno-cervicular joint and the joints of the hand are not at all uncommon, even in the small joints of the feet. There is diminished movement. There may be paraesthesia. If you compress the site there may be a local response to pressure, which is quite useful as a diagnostic point. The pain may flit from joint to joint. It may be in the shoulder for a while and then in the knee. You have got to watch these people very, very carefully. But although you may get occasional redness and oedema over a joint, most commonly there are absolutely no physical signs whatsoever. The next important little statement is that if the diver says he has got pain in the knee, it does not matter what the examination reveals, that diver has got decompression sickness until proved otherwise. As far as I am concerned, proved otherwise means treatment by recompression. No physical signs are required for the diagnosis.

A typical cutaneous manifestation of decompression sickness is purple blotching, a sort of venous stasis which occurs in the skin. It does not require treatment per se, but nevertheless does respond to treatment if treatment is given. One should not confuse this with suit squeeze. There is an increasing tendency in the colder climates now to use dry suits. Unless the dry suit is fitted with a suit inflation valve then you get pinching and nipping of the skin by the dry suit, which is a very painful cutaneous condition which should not be confused with the cutaneous manifestation of decompression sickness. There are some more unusual forms of decompression sickness which are not very often seen. One is pitting oedema of the hand which is completely painless. It is thought to be due to the formation of bubbles in the lymphatics. If you believe Guyton’s hypothesis of the formation of the lymphatic fluid, the lymphatics have got the lowest tension of all the vessels in the body, and so a priori the lymphatics would be a place where bubbles form. Therefore in some individuals the bubbles will get dammed and you get lymphatic oedema. Another is pitting oedema of the lower part of the chest wall and other parts of the body. One diver was actually diagnosed as having mumps. He thought it was rather curious that he should suddenly get mumps just 20 minutes after surfacing from a dive, so he went to another doctor who made the diagnosis of decompression sickness, because on the whole, mumps does not fit.

The serious manifestations of decompression sickness are neurological and spinal. I think it is worth repeating that the most common onset is that the diver says that he has got a few pins and needles in one foot or a cottonwool feeling. A feeling that then ascends all too rapidly to become paraplegia. This is the commonest presentation of spinal decompression and one which I think is fairly ominous. But of course there are many, many others. So far as the spinal cord lesions are concerned I think that the worst situation that you can be in, is with the man whose paraplegia extends to quadriplegia and he is just left with the phrenic nerve pumping away. At that particular time you wish you had taken up some other specialty.

Labyrinthine decompression sickness or the staggers, has a very dramatic onset. It occurs particularly after rapid ascent, with nausea, vomiting, nystagmus and possibly tinnitus.

Cerebral symptoms include dysarthria, visual manifestations and changes in personality. Changes of personality are very, very difficult to diagnose. Although it is easy to say at a lecture that if a diver changes his personality after a dive he should be recompressed, in fact you usually find that the guy is being recompressed with reluctance and possibly for some other reason. It is only after the recompression you suddenly realise that the man has flipped back to normal and how very abnormal his behaviour was before the recompression. It is something to watch for and it is where the diver’s buddies are going to be very useful in assisting to make the diagnosis.

The pathognomonic sign in the respiratory manifestations of decompression sickness is the retro-sternal catch on trying to take a deep breath. It is best seen in divers who smoke, because they come out of the water, light up a smoke, take a deep breath and catch their breath because of the retro-sternal pain. If it is left untreated shallow, rapid respiration, pallor and shock develop. Shock is not a presenting manifestation. It tends to be rather overlooked and it is most common in cases which have been maltreated elsewhere. As a result they develop a postural hypotension associated with significant haemo-concentration. This used to be a presentation of decompression sickness in aviators too, until they discovered that it was not illegal for aviators to have decompression sickness.

Once it was quite a respectable illness, the aviators started reporting limb bends as well as collapsing after high altitude runs. If you read some of the older text books they mention shock as being a presenting symptom of decompression sickness. In my view, it is not a presenting manifestation. However you will find haemo-concentration very early on and even with limb bends.

Diagnosis

The investigations that one should make before coming to a diagnosis are quite simple, zero. My background is naval and commercial. As far as I am concerned, if a diver has the symptoms of decompression sickness I put him into the chamber, recompress him and examine him at depth to see if anything is left. With luck, there is not anything left. As far as I am concerned any delay of treatment, even to examine the patient, makes the treatment less likely to succeed. In my opinion it is good management to make the presumptive diagnosis on the symptoms, recompress the patient and then examine at maximum depth to make certain that the treatment has been effective. Jefferson Davis does not agree with this approach because he sees a different type of diving accident. I see divers who are diving close to a chamber so we can virtually get a 100% response even with rapid onset decompression sickness. In sports diving you have the problem of delay, which can be 5 to 36 hours after the onset of symptoms before they come for treatment. Under these circumstances it is reasonable to conduct a fairly rapid but meticulous neurological examination to obtain a good base line for judging progress so allowing better management of the case, but also it is much more allowable to commence ancillary treatment. It is much easier to put up a drip and put in a catheter before putting the patient into the chamber, rather than struggling with these in the confines of the chamber. But nevertheless the important message to take home is that you do not have to examine the patient, and even if you do examine the patient, you do not have to find anything wrong with him, to come to the conclusion that decompression sickness exists. The symptoms are sufficient, the feeling of numbness, the feeling of cottonwool feet, the feeling of pain in the limbs, the feeling of difficulty of deep breathing, all these require treatment as a medical emergency. That is the take-home message.

Pathophysiology

Let us consider the concept of decompression sickness. The old idea was that a safe dive was one during which no bubbles occurred. If you got bubbles, then you got the bends. Now we all know that is absolute rubbish. The Doppler Bubble Detector will detect bubbles in lots and lots of people who make a perfectly safe dive and never have any trouble at all. So we have quite a large overlap of bubbles occurring in safe dives. I think it is true to say that there are no dives that result in decompression sickness in which bubbling does not occur, although there are reports of this in animal experimentation.

Where do the bubbles begin? I have already mentioned the extravascular bubbles in the lymphatics. Catchpole and Girsch showed bubbles occurring in various extravascular tissues such as myelin sheaths which I think are important as possible causes of spinal decompression sickness. Extravascular bubbles occur in the fatty tissues of the body, particularly in the bone marrow and perhaps then the gas bursting out of these fatty tissues into the capillary bed and into the veins. What we normally notice first are the venous bubbles. Venous bubbles on the whole are filtered out by the pulmonary bed. Just occasionally a few might get through to the arterial side. We also know that arterial bubbles get through the systemic capillary bed very easily and will go through the venous side. It is generally agreed in the old text books that the pain of decompression sickness is due to bubbles and that all the other manifestations are due to bubbles acting as little plugs in the blood vessels so causing all the manifestations of decompression sickness.

I think the story is significantly modified now. It is important to recognise also that arterial gas emboli may occur in decompression sickness. I would like to stress this point because people who have not read the papers by John Hallenbeck and myself, assume that our hypothesis of venous infarction of the spinal cord is exclusive. It is merely an hypothesis for one mechanism of spinal cord decompression sickness. It is not exclusive. I think the final pathology can be a single end result of one or more pathogenetic pathways, of which arterial gas embolism is one. The original Haldanian hypothesis that gas embolism to the fatty myelin rich parts of the cord causes spinal cord decompression sickness can not be excluded. We think there are at least two other hypotheses which are more likely, one being the venous hypothesis and the other being the one which Brian Hills and Phillip James have re-emphasised recently, the formation of bubbles de novo in the myelin tissues of the spinal cord.

Bubble surface activity is pretty well known. It is mostly known from open heart surgery days and bubble oxygenators. A lot of work has been done to demonstrate the mechanism of the blood-gas interface in causing various changes. These are all proven effects. The activation of the Hageman factor is not only responsible for the cascade part of the blood coagulation cycle, but is also responsible for simulating a whole stack of other things, the release of various vaso-active substances and kinins and so forth. The activation of enzymes is another proven effect of the blood-gas interface. The de-naturation of proteins causes the red cell clumping which is well seen in decompression sickness. The rouleaux

phenomena has been described for more than 100 years now. The significant thing is platelet aggregation with the release of all the nasties the platelets have got stored within them. Already we are getting into a situation which is like a seamless web. A specialist in this particular field of haematology and blood-gas interface effects used the phrase "seamless web". I think it is a very good analogy. If you twang a spider's web in one part the whole web vibrates and you do not quite know which part of the web is going to shed the next drop of dew. This is also true of the blood system. Those of us who have worked in this field realise that Mother Nature has not got just one card up her sleeve but an infinite number. It does not matter how we play with the system, whatever therapeutic effect you put into an experimental decompression model, there is always some other pathway out of which comes a counter-effect which can nullify what we are trying to do. So it is an extremely complex situation.

Finally, and quite separately, the denaturation of lipo-proteins. I have kept that separate, because the lipid emboli that are formed as a result of that particular blood-gas interface effect do not have any other ramifications in haematology, they merely float around the blood stream and can cause various effects as emboli. They are frequently found in the autopsies of divers.

Here is a working hypothesis of decompression sickness which I think is quite useful. We have the so-called silent intravenous bubble, silent because there are no outward manifestations of decompression sickness. However, they can be detected by Doppler. The silent bubbles cause cellular aggregation of red cells and platelets, the release of kinins and other vaso-active compounds and the formation of lipid emboli. These cause a subclinical pulmonary embolism with tachypnoea, fatigue and malaise. That has been well established as being due to a sub-clinical pulmonary oedema.

We now get into an area of dispute. Vasospasm and bronchospasm lead to the severe pulmonary distress of the chokes, I think that is well established. But then it is suggested that the pulmonary congestion back pressures the vertebral venous plexus and causes spinal decompression sickness. The only point to make at this stage is that the hypothesis of venous infarction of the spinal cord does not depend upon the pulmonary part of that story to exist. It exists as its own entity as far as pathogenesis in concerned.

Studies in splenectomized dogs, using radio-active labelled albumin, show no change in plasma volume in "no-decompression" dives. After a dive in which severe chokes and paresis occurred there was evidence of loss of circulating protein into the tissues, of the formation of oedema, and of haemoconcentration.

In the chamber at Duke University in which we did this work we had an X-ray machine that was pressure proof. We could take it into the chamber and take it down to 300 feet. In fact, we only took it to 155 feet. Besides taking X-rays of the vertebral venous plexus we exposed the spinal cords of the dogs and made movies of the vertebral venous plexus during the dives. Quite definite congestion occurs after a dive. In one slide the congestion has even filled up one of the spinous process veins. In another slide the bubbles can be seen along the vertebral venous plexus. Just looking at the vertebra you can see it is almost as though it has been cleaned out, there is not a blood vessel to be seen in that area, although in the pre-dive slide of the same orientation we could see the vertebral venous plexus very well. What we are really saying is that in this particular experimental model, the vertebral venous plexus just gets wiped out with bubbles.

Using a C-14 pyrene technique one can measure instantaneous blood flows, but only once, as you have to kill the dog to do in millilitres perfusion per 100 gms of tissue. Using this technique one can show a normal flow in a normal spinal cord, but after the onset of paretic decompression sickness there are areas of virtually no flow.

Histology

In paretic decompression sickness there are, besides haemorrhagic areas, vacuolations in the myelin. This we can consider as being due to bubble formation in the myelin sheaths. There are two hypotheses as to why this should occur. One is that the bubbles will form there de novo anyway, and the second, and separate one, is that they form there as a result of venous slowing and stasis, the damming up of the circulation. The only difference is that in the venous infarction of the spinal cord hypothesis, which wipes out whole levels of the spinal cord at once, the stasis of the venous system for more than a silicone clotting time allows clot formation to occur in the smaller vessels of the cord. This obviously is bad news so far as the owner of the cord is concerned. In all our slides from paretic animals there are areas of haemorrhage and bubbles in the myelin.

We consider that there is sufficient pathology just with the vascular effects, the formation of clots, the damming back of the blood flow in the capillaries, to explain the histology.

The third hypothesis is based on the fact that although the spinal cord is soft, flexible tissue, it is not very expansile. Therefore the formation of gas in the myelin increases the internal tension within that segment of the cord. The increased tension will squeeze out all the blood from that particular part of the cord and cause spinal decompression sickness. That is a perfectly acceptable alternative hypothesis. The work on that is not yet complete.

We have got three aetiologies for spinal cord decompression sickness to consider arterial gas embolism, venous infarction and in-situ bubble formation. John Hallenbeck and I

consider this to be a secondary effect to the fact that clots were forming in the venous system. It is obvious that the pathology is the same. It is only that the explanations are at variance.

We have a scanning electron micrograph that we did showing one of the vessels of a nerve root very close to the spinal cord. Using increasing magnifications you can see strands of fibrin and red cells on the side wall of that vessel, which is quite positive evidence that it is a pre-mortem clot. We feel confident that we demonstrated in that animal that there was venous clotting and that venous infarction of the cord was the cause of that particular animal's paralysis.

We have covered the sorts of things that are going on at the time when the diver is saying "Please doc, my feet are going numb". The take home message is that it is not just a question of bubbles getting stuck in the spinal cord, there is a whole lot of other pathology going on and that it is very much more complex than anybody would care to claim to understand.

John Hallenbeck and I did this work about ten years ago. I quit about five years ago. He is now working not so much on spinal cord decompression sickness, but on cerebral gas embolism. I particularly commend to you the edition of Stroke of December, 1979. He has not published anything in an easily accessible journals since then. He has been working on the effects of gas embolism to the brain. What he was concerned about was the fact that once you stop the brain's circulation there is no recovery. The majority of people still say that after four minutes the brain is dead and there is nothing you can do to restore circulation. I am pleased to say that John Hallenbeck's work has shown that to be a load of rubbish. In essence what he has done is to examine the various factors which are responsible for the so-called "no reflow phenomenon". Even if you restore the blood pressure and remove the embolus, you cannot restore the circulation to that particular part of the brain that suffered ischaemia. He has demonstrated that there are various endothelial and other factors which contribute to the "no reflow phenomenon". He reported in this particular paper that he gets a 65% recovery of function in animals which otherwise would have made no recovery at all by the use of prostaglandin and indomethacin. He made a superb presentation only about four weeks ago on this. This is work that is still going on, demonstrating that there is some hope that in those divers who get gas embolism we will perhaps be able to prescribe some medication which may help to restore function. I had John Hallenbeck come over and present his paper just a year ago to the Institute of Neurology at Queen's Square. It was one of those electric occasions when the audience is all a little bit bored and shuffly, and suddenly you could just feel everybody stop and pay attention to what John Hallenbeck was saying. It was a tremendous occasion. The work is continuing at Bethesda and it is well worth keeping in mind not only for gas embolism cases, but also it may well have an application in cases of embolic stroke and so forth.

DISCUSSION

Chairman: Dr John Knight

You did not mention rapid ascent as being a common cause of the onset of decompression sickness. Certainly in the cases which are treated in Sydney and the cases that occur at Nauru, it is a very common cause. Some of these are inside the tables but they have all shot to the surface, having run out of air, or seen a shark, and need treatment. A mild example is a friend of mine who was diving in Portsea Hole with four friends when he saw the anchor of his boat go past. He was at 80 feet. You can only dive there at slack water, so he was after the boat like a rocket. His knee started to hurt about two hours later and it went away after about three days. He did not bother the doctor in the interval.

Dr David Elliott

From an academic point of view I am delighted to hear this. I am sorry for the guys, but this in fact is what I was implying when I said let us not be dogmatic and divide decompression disorders into the two principal extremes. There are obviously mixed cases. By that I mean that we have got quite a lot of cases on file now of people who should have been pulmonary barotrauma or might have actually produced pulmonary barotrauma, and who then developed decompression sickness. They should not have really had decompression sickness at all because their dive was perfectly safe.

In my own courses we do a tremendous lot of case history discussions. I think that they are a really important part of that kind of meeting. The case which I will now present has just made me think of one other aspect of decompression sickness, that I have not stressed at this particular meeting.

A diver was at about 50 feet, playing with one of those research submarines in Jamaica. He had been down 50 minutes or so at 50 feet, certainly well within the non-stop times, and made a rapid ascent. Within half an hour he had a classic onset of decompression sickness. I think that is the sort of example that you could be talking about.

This particular guy gets rather quadriplegic, so how about recompression? That particular island in the Caribbean does not have a chamber. So they shipped him to a recompression chamber. I hasten to add that the chamber was not the British Sub-Aqua Club chamber at Cayman Island, but a chamber on another island. It was run by a Navy and not, I hasten to add, the Royal Navy. It was run by a Navy which shall remain anonymous. This poor quadriplegic was treated quite correctly by recompression. However the physicians had not been properly trained in diving medicine. They were very concerned about this paralysed

diver, because he had an acute abdomen. So they brought him to the surface and they opened him up. What do you think they found? He had a catheter in so it was not a full bladder. They found that he had a paralytic ileus. Not very surprising really. So they sewed him up again, and he continued to be a quadriplegic with a burst abdomen. That went on for a long time. You will be pleased to know that he is wandering around on sticks now.

He was a classic case of a mixed presentation. The guy who rushes to the surface after 50 minutes at 50 feet, expected to be the classic gas embolism presentation, yet he showed classic decompression sickness.

Dennis Walder was the person who first put forward some kind of an hypothesis for this. I gave him credit for this hypothesis in a paper, and when I told him he said "That is not what I meant at all". He suggested that in some alveoli there might be air trapping which might cause micro-barotrauma. That some of the smaller alveoli might well distend with gas during ascent and blip off bubbles into the pulmonary venous system. These bubbles could seed and as they went through areas of high inert gas loading, those bubbles would grow producing decompression sickness.

Remembering that there are a lot of people who do a rapid ascent and produce sub-clinical EEG signs or mediastinal emphysema from decompression barotrauma, it is a perfectly reasonable working hypothesis that a person can come rapidly to the surface, discharge into his bloodstream a shower of small bubbles, which really do not matter a damn. Except in those individuals whose tissues are already loaded with gas. They are within a no-stop time, but they have got a fair load of nitrogen in their system. The bubbles and the inert gas load act synergistically to produce decompression sickness in the person who has made a rapid ascent.

Chairman: Dr John Knight

What you have been saying confirms what John Miller was saying to us last year that the people that he gets from the Caribbean usually have a combination of illnesses. It also confirms that if you really look for neurological signs you can often find them.

Question: Dr Bob Hare

I would like to ask a couple of questions about buoyant ascent. Firstly, I am curious to know whether, when you are breathing out for something like a minute and a half ascending from 100 feet, you get a desire to take a breath in. Secondly, could airway collapse be prevented by breathing out against resistance through pursed lips, in much the same way as someone with emphysema does.

Dr David Elliott

To take the second question first. The answer is yes. If you can make the pressure gradient less, by breathing out through pursed lips, maybe that would help, because it is an equal pressure point down the airway which causes the trouble. There is no problem in regard to your first question, in fact as one comes up exhaling, one is just washing CO_2 out.

Buoyant ascent to me is an ascent at approximately 6 to 8 feet per second, when you are in fact using positive buoyancy. Free ascent is not a term that I personally use in association with diving. A free ascent is really confined to a submarine escape training tank. A person who is breathing compressed air at depth from an air lock, who then uses for buoyancy merely his expanded chest containing compressed gas. That is a highly specialised method of ascent. Because you have taken a breath of compressed gas at depth, you have got no gear on, so the only buoyancy that you have got is a full set of lungs. You have got to use your full set of lungs to get your body back to the surface and yet you have got to blow out enough so as not to burst your lungs. That is free ascent. When I did it, I blew out too much and became negatively buoyant. Once you are negatively buoyant you can only go down.

Question: Dr Bob Hare

Say you are scuba diving at 100 feet, and something goes wrong and you wish to go up to the surface faster than usual. Theoretically you should take a minute and a half to do that, exhaling all the time. Does one get a desire to breathe in on the way up? If not it must mean that the stretch receptors must have a lot to do with the desire to breathe in.

Dr David Elliott

The answer to your final phrase is an absolute "YES". There is no doubt about it, the stretch receptors are very important. Without them snorkel diving would be a pain. As far as the minute and a half ascent from 100 feet goes, I see no problem. CO_2 washout is going to help you all the way. I would say once again that if you are having difficulty making it so slowly, flare everything. Make sure that your fins are at right angles to your legs.

Question: Dr Mike Page

During the first talk you showed slides where a diver's X-ray was normal before the ascent and after the ascent it had basal emphysematous bullae. Was that an expiratory film? Is it best to do an expiratory film in routine screening rather than normal inspiration chest X-rays?

Dr David Elliott

Dr John Harrison, who actually reported on those films, went through a phase of saying that every diver should really have an inspiratory and an expiratory film in order to detect bullae that would otherwise be hidden. On the very first one he picked up bullae that would otherwise have been hidden behind the heart. The guy was a professional instructor who had never had any kind of incident. I am

not quite sure whether it helped or not. The answer to your question is that if you really want to be meticulous, do both films.

Question: Dr Tony Slark

You did not mention DIC (Disseminated Intravascular Coagulation) at all. Is this the next thrilling installment?

Dr David Elliott

No, not at all. There was about seven or eight years ago lots of discussion as to whether or not the various effects of the blood gas interface would in fact cause DIC. The haematologists got to work and started looking for fibrin degradation products and doing all the other clever things that they do. The net result was that DIC as such does not exist in decompression sickness. We say quite specifically that intravascular coagulation does occur in decompression sickness. Therefore the word that we have to be a little bit semantic about is disseminated. We are really saying that it is only in those parts of the circulation where stasis occurs longer than a normal silicone clotting time that time the blood will coagulate.

Question: Dr Tony Slark

How then do you account for slides that you show and that others have shown many times in the past of extravascular clotting?

Dr David Elliott

Extravascular clotting? You mean haemorrhage. I would say that haemorrhage is a very common finding in the pathology of spinal cord decompression sickness. I would not like to guess at what actually causes the haemorrhage.

Chairman: Dr John Knight

About five years ago anaesthesia was besieged with cases of Disseminated Intravascular Coagulopathy. It occurred in everything. Over the past five years a certain amount of sanity has returned. Instead of saying that everybody, everytime anything happened to them got DIC that you could not detect unless you did very specialised blood tests, now we are accepting that DIC is in fact rare in ordinary human trauma.

Question: Dr Mike Ramsay

I would dispute that. What you are talking about is a condition, not a disease. A full blast DIC is as rare as hen's teeth I agree. Thank God you do not meet many of them.

I would like to know whether it is intravascular coagulopathy or intravascular coagulation. To me there is a difference. The first is a response to a stress. You looked at the dog's spinal cord. Did you examine its belly vasculature?

Dr David Elliott

No.

Question: Dr Mike Ramsay

Was the vascular status in the rest of the animal any different from that of the spinal cord?

Dr David Elliott

You mean did the animal have a Disseminated Intravascular Coagulation throughout its entire body? Is that your hypothesis? Then one would expect surely, that the animal would not only be suffering from a spinal cord lesion. The spinal cord manifestations are not a presentation of DIC. This was an animal which had done a pretty horrendous dive. It would have survived about three to five hours after the dive ended if it had not been sacrificed.

Question: Dr Janene Mannerheim

In the presentation of decompression sickness how common are limb bends that are not very painful? Is severe headache on its own ever a presentation?

Dr David Elliott

Both of these are good questions. I will deal with the limb bends first. The trouble about limb bends is that it is totally subjective. What the patient feels has to be interpreted by him and passed on to the doctor. If you put a novice diver in a team with experienced professional divers, you will find that the novice diver is always having pains here and there. Things that the experienced diver just accepts as part of his way of life. If you see a person doing a repetitive movement, without even being aware of it, keep an eye on him. Because maybe an hour or so later he is going to say that he has got pain. From my own experience I would not be surprised if one or two of you had not had similar feelings of discomfort. One is just not quite sure what it is.

Yes, headache is a presenting manifestation and it tends to be a migranous type of headache. The hypothesis (we are great on hypotheses, it is proving them that is difficult) for what it is worth, is that the platelets are all stirred up with the bubbles floating around. It is not unlike the mechanism of migraine. Those who dive who are migraine sufferers say that if they get a migraine following a dive, it is one hell of a migraine as opposed to a normal headache.

Question: Dr Peter James

Can the neurological signs of decompression sickness be transient? How should you act if they go away?

Dr David Elliott

Yes, the neurological manifestations can be transient, in fact if you go back to Paul Bert of one hundred years or so ago, before recompression was used for treatment, you will find that a large proportion of people got better without any treatment at all. The trouble is that in this modern day, when we have treatment, you can not predict which guy is going to be the one who gets better, and which is the guy who is going to get worse.

Question: Dr Peter James

This is an interesting point. I had one patient with slurred speech that lasted fifteen minutes and went away completely. The two chambers that I rang both said that he was not down long enough to be bent. He had dived within the no-decompression times.

Dr David Elliott

The thing about these people who do get better spontaneously and do not require treatment, for there is nothing to treat, is that they should be handled in the same way as a person who has been successfully treated. That is, they should be kept in the immediate vicinity of the chamber or got to the immediate vicinity of the chamber. Because there are plenty of people who have had such transient episodes, who maybe six hours later get a recurrence which was not so transient. I think those chambers made two mistakes.

Question: Dr John Knight

Do you expect somebody who presents with chokes twelve hours from a chamber, to be dead by the time he gets to the chamber? John Miller said last year that all untreated chokes were fatal, but I have met divers who have said that they have had the chokes and were not treated.

Dr David Elliott

It could be a very mild form of chokes. The chokes is a mass of venous gas emboli going to the lungs. I suppose there is no reason why they should not resolve normally and be a transient phenomenon just like anything else.

Dr Tony Slark

There is another answer and that is that the majority of people do not report minor chokes. It is not a problem so far as I can see with divers, though it may be with aviators. It is a very rare thing with divers and particularly with scuba divers.

Dr John Knight

Going on from that, two years ago I came up from a dive much faster than I normally do. About thirty seconds after I got to the surface I became extremely breathless. I also started to wheeze. I just could not control my respiration. There was no hope of me swapping to a snorkel. This lasted for about five minutes. I could not get myself into the boat. All I could do was to float in my compensator. I thought that I possibly had done what I saw in the abstracts of the UMS meeting, bubbled my blood on the way up and got a load of bubbles in my lungs. Thinking back on it, in the old text books of anaesthesia, in the days when they allowed spontaneous respiration for neurosurgery, one of the things that you were told was that if the respiratory rate suddenly increased, the patient had sucked air into a vein and developed gas embolism of the lung. I wondered whether you had had similar stories told to you, or had seen anything similar? Douglas Walker put it down to a tight wetsuit.

Dr David Elliott

No. I think it is because the population that we deal with do not do that kind of diving and so do not get that kind of problem.

Question: Dr George Thompson

I wonder whether David could speculate or tell us the mechanism of the feeling of impending blindness in gas embolism?

Dr David Elliott

In fact it is just that the guys feel that they are going blind and they do go blind. One can only assume that it is occipital or somewhere else on the visual pathway.

Question: Dr Terry McGrath

I know of a diver some time ago who went blind in one eye. He said it was like watching a television screen go off. Not being very experienced in this, I sent him off to an ophthalmologist who said that he had a retinal artery thrombosis. Slowly over the ensuing weeks his vision came back again.

Question: Unidentified Speaker

I have a question about micro-bubbles and rapid compression fracturing micro-bubbles.

Dr David Elliott

I think what you are referring to are micro-nuclei and the fact that if you compress sufficiently you will in fact re-dissolve the micro-nuclei. But this is the sort of thing you do with shrimps by banging them down to 1,500 feet in two seconds. It is not good for humans.