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DIVING ILLNESSES AND INJURIES

EYES AND DIVING

Dr Peter Cohen

The main eye problems have with diving are common sense ones. I think more problems come from divers on the surface than underwater. By this I mean, it takes 2.5-3 hours to get out there and you are under for an hour and then you come up and go home. I practice in a coastal suburb and I do see some divers - and people who go out on the ocean. The common problem I see is ultraviolet sunburn. They come in with the equivalent of a welder's flash with a sore eye. The treatment is quite obvious. Keep out of the sunlight for 2 days and the eye usually gets better.

The few professional divers that I know, just like professional fishermen and amateur or professional yachtsmen, get pterygiums. It amazes me that they get out there and there is a dead calm mirror ocean yet they never wear sunglasses. They end up with pterygiums. If you lop them off and the men go back onto the ocean they grow back. And they grow viciously and are very nasty.

Another problem that is practically confined to inexperienced divers is mask squeeze. I do not think an experienced diver will run into this problem. Mask squeeze can present some problems in that people may get, with severe mask squeeze, subconjunctival haemorrhages because the conjunctival vessels are unsupported. I consider sub-conjunctival haemorrhages are innocuous. I do see a fair number in my practice, not necessarily from diving but from any episode of raised venous pressure, and I generally tell them just not to worry. But with a severe mask squeeze they can get chemosis and the conjunctiva will prolapse outside the eye, which does tend to make people panic. However with a tulle gras dressing and an eye pad it should settle down without anything further needing to be done.

Coming on to people with medical eye problems that would be a contraindication to diving. One would be a very high myope who has a history of retinal detachment or retinal hole. I would very much predispose to getting either another hole or a retinal detachment. A retinal detachment in high myopes is a very severe thing. They often do not do well. Their retinas tear very easily as the retina is very stretched and the retina comes off easily. It is very difficult to get it back on. The technique of our surgery with retinal detachments, nowadays, is that we put encircling bands around the eye and we use a plomb, which is a piece of silicone which is pushed down to indent the sclera. We are trying to indent the sclera over where the hole is. With a high myope the sclera is very thin and you can easily perforate the eyeball. Then you are in more troubles than when you started.

The other thing that I am told would also be dangerous, but I have never been confronted with it, is a person with advanced glaucoma with a severe loss of visual fields. Both these conditions of high myopia and advanced glaucoma tend to occur in people who would not dive. So I do not worry about it too much. A high myope is not going to see very much underwater - so why dive? He is going to have trouble with what techniques we have trying to correct vision underwater.

On the whole I am consulted by divers about refractive errors. I am a myope, and I wear contact lenses underwater. But they do have their hazards which I will tell you about. A lot of people do wear lenses glued on to the mask, that is the only alternative. One girl I know actually has wired in, and sealed with some Araldite, a pair of spectacles so that they sit on her nose when she puts the mask on. But it is a very difficult thing to do. The problem with putting lenses in masks is that you have already got a restricted visual field from using the lens. The size of the lens is so large that you get peripheral aberrations, quite severe peripheral aberrations, in a lot of cases. If you are quite happy with having a further restriction to your visual field there is probably no trouble. A lens is adjusted to being a certain distance away from your eye. When you move it further away from the eye and glue it to a face mask and then start looking down and sideways through the lens you start to get oblique astigmatism and this really interferes with clear vision.

In my practice I am quite a fanatic. I believe in people wearing contact lenses. I seem to advise them all the time. Perhaps they think I am a nut because a lot of them lose them.

Over the years we have gone through a development of certain types of contact lenses. The first ones were the scleral lenses. This is the very earliest type of contact lens, and we still fit a few of them. They are made out of hard polymethyl methacrylate. They go right over the cornea and extend onto the limbus and under the eyelid. The first ones were made in about 1857. In those days they used glass. But these lenses were very difficult to construct. They have the advantage that they do not fall out underwater. One of the problems under water with any contact lens is that the lenses stick on with surface tension. If you flood your mask you can very easily have them come out. I do not fit these scleral lenses. I refer them on.

I know one ophthalmic surgeon in Sydney who does a few each year and he says that about his only call for these lenses, for optical purposes, is that he prescribes them for swimmers, especially water polo players where they really need to see what they are doing. They do have a practical role because they do not fall out. They can be used for cosmetic purposes as you can paint an eye over this lens for someone who has got a very scarred eye that is non-functioning. One problem with them is that they have a quite limited wearing time. Often the patient can only tolerate wearing them for 4 hours at a time. They are very difficult to make. To give you an example of why they are difficult to make. I will explain the procedure.

One places a small cup on the anaesthetised eye. There is a tube poking out from the cup. You get a 2 cc syringe and you squirt a quick setting substance down the tube. This gives you a mould of the front of the cornea. And from that negative mould you make a positive mould in plaster or something a bit harder than plaster. On that positive you start to make the lens. After which you start to grind the lens. It is a real hassle to make it and people must really want to have to make it. But for people who want to use contact lenses underwater in a situation where they could very easily lose them and they will be under for a short time they are a very practical answer.

The other types of lens that we have, and it has been around for a long while is the corneal micro lens which is made out of a hard material. The commonest material that is used is the same material from which scleral lenses are made, poly-methyl methacrylate. Nowadays they are mainly used in people with astigmatism and who are hypermetropic. Once upon a time they were the standard lens, before the soft lenses came in. The problem with them is that they are more easily lost and especially they are very easily lost in divers. You have just got to get the slightest amount of water in the eye and they lose surface tension and pop out. They have problems with adaptation. I have a continual parade of people who come in and say “I just cannot wear these things”. They are a lot harder on the cornea. Lately the materials they have been made of have changed so that the manufacturers say that they are of a semi-soft material. It is cellulose acetyl butyrate and has a higher oxygen transmission and is supposed to be better. But I am not quite sure. And we have got even later materials which I will not go into. The advantage of them over other lenses is that they are very easy to prescribe. The softer lenses in some people are quite hard to prescribe but these often are a bit easier. It depends on whether the patient has a lot of astigmatism or not. But the problem is that if people have high hypermetropic errors trying to prescribe our current plastics in soft lenses is a very difficult problem, if they want good vision and they want to wear contact lenses.

The soft lenses are the modern contact lenses. Someone added a water group on to the plastic molecule. That is what led to the start of the soft lenses. The interesting thing that it looks very easy to add water. It was done in Czechoslovakia and the firm that did it has since made about $600,000,000 out of putting that extra radical on the molecule. It is the dominant plastic that is now used in soft lenses. And soft lenses are a big business. They are very much a big business now. They are very flexible. They go over the corneal-scleral junction about 1.5 mm. Generally in a person who is myopic they are easy to prescribe. For someone who has myopic astigmatism they are reasonably easy to prescribe. And for people over about 50 years old who are long-sighted they are not too difficult. But our problem with them is that they have a low oxygen transmission and they are only about 38% water content. Which leads people who are often active in sport who want contact lenses to run into trouble.

There have been some plastics which have come out over the last few years, that are 50% water content. They are claimed to be better. But I find that the problem is that as the water content of the lens goes up they become much more of a mechanical problem for the patients to look after. They tear very easily. They become also more expensive. We have even got up to the stage now of having a type of soft lens material that has 75% water content. The manufacturers of this material boast that it is a permanent wear lens. The interesting thing is that the ophthalmologists all refer to it as an extended wear lens because there is a problem with wearing these lenses all the time. With the normal soft lenses you have got to take them out every night. Clean them overnight and put them in the morning. But with these extended wear high water content lenses they are more oxygen permeable because of the high water content. The problem is that the lenses are not being disinfected. There have been a number of instances, even in Australia, where this has led to a number of corneal abscesses. You have only get to have one corneal abscess in a person who wants to wear a lens for cosmetic purposes to put you off these lenses for a very considerable period. The other big problem with a high water content lens is that, because of the high water content, they are very friable. The patients have to consult you quite frequently. You have got to be around all the time in case they run into trouble. You have got to see them once a month. It becomes both time consuming and expensive for the patient. So the practical application of these very high water content lenses, not the normal soft lens, is in aphakics and that is only in some people who are long-sighted. The biggest problem a lot of long-sighted people, really long-sighted people, have with contact lenses is that they cannot see the contact lens to put it in their eye, when they have got to put it in first thing in the morning.

Originally when the soft lenses first came out we could not prescribe corrections for astigmatism. We have reached the stage now when we use the same plastic and we can. We truncate it, that means we cut off the bottom of the lens. This helps the lens stay correctly positioned. We make it so that it is thicker at the bottom than at the top. The normal thing that happens with a normal contact lens when people blink is that it rotates. It spins around. And you can easily see what I mean when if you look at an eyelid closing slowly. It closes like a zipper from the outside to the inside. And that just spins the lens more or less inwards and upwards and out. The idea of making the soft lenses thicker at the bottom is so that every time they blink the lens settles down between blinks. It is not always easy to get these soft lenses sitting as you would like to think they should. We have various ways of doing it. We can make them even a little bit thicker at the bottom but this sometimes leads to problems in that they complain that every time they blink they can feel the contact lens between their eyelids and a lot of people really do not like it. We do get a lot of problems with these lenses.

Soft lenses are reasonably cheap and easy to make. When I mean by cheap is that the price is around $100 to $114 for a normal spherical correction. But these special astigmatism lenses in Sydney are $184 a pair. And you really would think twice about diving with them on, when you have got a very good chance, if you flood your mask, of losing them. For unlike the complete spherical lens these lenses will float off easily. We can make the same lens, completely spherical with the ballast just incorporated in a part that we would normally cut off. However I find I run into fitting problem.

Some attempts have been made and are still being made, and I am sure as time goes will be successful, of making silicone lenses. That is using silicone rubber. The problem with silicone rubber is that it is hydrophobic, it repels water. We were at one stage told these were the ant's pants for people after a cataract operation. They are easier to put in as the silicone lens is semi-rigid. Because they are hydrophobic and repel water the manufacturers put a chemical on the outside so that they would not repel water. If you do not you would get the tears sitting as little balls of water on the front of the contact lens, therefore interfering with vision. I was not the only one involved in this trial and we prescribed them. After about a year the patients all came back because the chemical had come off the front of the lens. Which was embarrassing when they had paid $150 for a pair of contact lenses. At the moment attempts are still being made, and some have been successful, to get these silicone elastomer lenses in some form that we can put into people that does not have all the disadvantages of the soft lenses.

The biggest disadvantage of the soft lenses is that they do take a fair bit of maintenance. We have got to spend about 10 minutes a night looking after them properly, sterilizing them. It does not worry me but it is a trial for some people.

That is basically all I want to say about eye problems of diving. As I said most problems are optical, not so much related to medical problems.

EAR AND DIVING

Dr Bill Hurst

I was asked to talk tonight about the ear. It is very difficult to talk to a group like this on topics that we have had year after year, You all know just about as much about ear barotrauma as I do. So I thought I would change it around and talk about dizziness because most people talk about deafness.

THE EXTERNAL EAR

When water that is either above body temperature or below body temperature enters the external ear convection currents are set up in the semicircular canals. This fools the ear because the fluid is moving either up or down depending whether your are using cold water or warm water. The vestibule on that side or the lower canal on that side thinks that there is motion, and because the opposite ear has not got an equal and opposite stimulus it throws the computer between the ears into confusion. Then we become dizzy. If in any way you get cold water into one ear more than the other you may get a caloric response. This will last only a short time. It is a true rotating nystagmus and it will settle down as the water warms up. Things that will cause this are wax blocking one ear or ear plugs. I put wax first because no diver in their right senses puts plugs in their ears. You do occasionally get some fool who has been scuba diving with ear plugs in and then you have to fish a plug out of the ear about six inches in. This causes quite a bit of trauma.

THE MIDDLE EAR

You all know the problems of descent and ascent, Eustachian tube obstruction and traumatic perforation. When you get a traumatic perforation there is exquisite pain, acute pain. Then you get a caloric stimulation. You get pain and immediate vertigo. Now the things that will cause a perforation are forceful auto-inflation, or more usually the fool who keeps going down when he has got pain in his ear. He keeps going down, down, down, and eventually it pops. He comes up screaming like a Polaris missile. The same can happen in ascent, this is the reverse Eustachian tube problem. Usually it is a passive mechanism in that as you are coming up the air goes down the Eustachian tube and escapes. I think that I might omit discussing locking on descent because someone might like to ask a question about that at the end of the discussion.

If you get a traumatic perforation while you are diving, the treatment is do nothing, unless you have a discharge from your ear. Strangely enough if you get a perforation in salt water usually there is no infection. This is true in the Melbourne waters. I am sure you would get a high plankton count in the middle ear here and you would probably get it infected. Certainly if you perforate your ear in fresh water, as we see often in water skiers, then you can get a discharge from the ear only a couple of hours after that injury. It is quite dramatic. In fact a lot them come in and they think that the water is still draining out of their ears from the incident. If you get a perforation you can also get cold water pouring into the middle ear and the caloric effect is even more dramatic than it was when it just goes into the external ear, because the cold water is close against the vestibule.

THE INNER EAR

I think that we really should put a lot of thought to the inner ear. Fistula formation is the in vogue condition with divers. ENT surgeons are making their name on seeing these little pools of perilymph. I think that we should be well aware of the possibility of divers coming in complaining of dizziness and deafness. I think really this is the thing that we have to exclude. But when trying to exclude it, you have got the problem of trying to make up your mind whether this is a decompression problem. And this is very, very difficult as they both have similar presentations, if there are no other symptoms apart from either dizziness or deafness. I do not know how you can do it. ENG (Electronystagmography) is not going to differentiate, because you get the same response. So I think you have to rely mainly on the history, and just determine whether this person has done a dive that exposed them to the risks of decompression sickness. If they did, they should be recompressed, because of the fact that their hearing will go and their labyrinthine function will not return without recompression.

But if you feel reasonably confident that your patient has a fistula of that their symptoms followed forced auto-inflation then they should be explored. It is a very simple procedure. You can roll the drum back without causing too much trauma. Then you see a little pool of fluid coming from either the round window or the oval window. I have no experience so for heaven's sake do not ask me questions about it. I understand that you scarify the area and just stick a hunk of fat there and hope that it stays there and does not float away. You should treat a fistula as soon as possible because the hearing tends to deteriorate with time. A few of them recover, but apparently a lot of them do not. So get in there quickly.

Perhaps I should briefly mention the causes of fistula. There are two theories that I would accept. There are a couple of others that I think are pretty airy-fairy. The first one is the forceful auto-inflation, where you go down having a bit of trouble, or and all of a sudden you equalise and air really goes up the Eustachian tube and the drum goes boom. If he has not perforated his eardrum he has pulled the ossicles right out and that will naturally pull the stapes out of the oval window. That is pretty straightforward. Also, if you get a lot of gas expanding in the middle ear as you come up, that could do it. Alternatively as you are descending, the drum can be forced in to such a degree that it forces the stapes actually into the vestibule. The other mechanism is a rise in CSF pressure coming through the cochlear aqueduct. When you strain as you do during Valsalva, it raises CSF pressure This comes through the cochlear aqueduct into the labyrinth and you get the pressure pushing the stapes out.

MISCELLANEOUS CAUSES

These really probably are not true vertigo. Syncope of ascent that is during rapid ascent while breathholding, is due to poor venous return and consequent poor cardiac output.

Motion sickness is something we are not really aware of. If you go down over some weed, turtlegrass or something like that, and watch it going backward and forwards, and you are just hovering over it, it is very motion sickness inducing. That really is just a what we call travellers' nystagmus being induced.

Chairman (Dr John Knight)

Peter Cohen ought to meet a ENT surgeon from Melbourne who went diving in the Seychelles. He had never been diving before. He did a resort course, but nobody told him about breathing out into your mask as you went down. He got down to 30' and could not see a thing. So he came up again. He could see again. Once again he could not see anything. When I saw him three weeks later his sub conjunctival haemorrhages still obscured the whole of the normally visible sclera. What happened was that his eyes and the surrounding soft tissue had been pushed into his goggles as he descended.

Dr David Cossar delivered a paper on sinuses. He outlined the development, anatomy and physiology of the sinuses and discussed the problems caused by the changes of pressure associated with diving.

COMMENTS

Dr John Miller

Apart from the mechanical aspects of dealing with the eye there are some other **aspe**cts that are of importance not directly in ordinary diving but in special sorts of situations. Particularly the effects of oxygen upon retinal perfusion. As you may know, if you have a patient breathing 100% oxygen during a compression and you continually look at the retina through an ophthalmoscope you will see at a pressure that ranges somewhere between 1.2 and 1.5 atmospheres absolute a sudden constriction of the arteries and of the arterioles in the retina. It seems to be a constant for the same person on a day to day basis but the normal range is somewhere between 1.2 and 1.5 atmospheres. There are some people who are really very sensitive to this and show the same sort of retinal spasm at very much lower partial pressures at something like 0.75 and 1 atmosphere absolute of oxygen.

Now this becomes particularly important in some of the older age groups who are exposed to these partial pressures of oxygen particularly for

relatively long periods of time. These are patients who are undergoing cycles of hyperbaric oxygen therapy, or somebody, an elderly person, undergoing or taking part in a prolonged saturation dive. The result from the prolonged retinal spasm appears to be a significant deterioration in vision that most times, but not always, is reversible. We see this frequently in the patients that we have with osteo-radio-necrosis of the mandible. These are patients who have had tumours of the head and neck, had radiation therapy, and then gone on to develop osteonecrosis of the mandible. This particular condition lends itself to regeneration of the bone, due to presumably revascularization, with hyperbaric oxygen. These people have a significant deterioration in vision for a fairly long period of time, like up to three months. The occasional one stays with permanent injury to vision. Recently during one of our long saturation nitrogen-oxygen treatments one of the nurses we had in there underwent the same visual deterioration after an exposure of six hours to 1.2 atmospheres of oxygen. She is a woman in her 50's who has been working for us for quite a long time accompanying patients into the chamber. She was exposed to air at 165 ft, for that length of time. It took her about a week and a half to get a significant return of vision. So that is one of the elements that I think it is important to recognise, that there are things that can happen at the back of the eye as well as the front of the eye.

Coming to the discussion of things to do with the ear and in particular the mechanisms involved and the history in trying to discriminate between a round window rupture and vestibular decompression sickness. The distinction has to be vital because in vestibular decompression sickness it would appear that the time between the onset of symptoms and treatment is absolutely critical. And is very short in the order of a maximum of 45 minutes. So that when this happens you have to make the correct diagnosis otherwise the individual is going to lose the inner ear on that side. These patients who have lost an inner ear on one side then compensate over the succeeding weeks. So that unless you do very precise testing they will frequently appear to be completely normal.

It is vitally important that a person who has had a decompression injury to one vestibule not dive again if that person wishes to avoid being made a cripple. The reason is if you wipe out both vestibules you wipe out the balance organs. Then you have to rely entirely on your eyesight to maintain your balance and orientation. So you can imagine that walking into a dark room becomes a real problem for those people. Closing their eyes at any time is a real problem. You also appreciate that, certainly in the law courts of North America if not the law courts of the South West Pacific, a customer who has a bilateral vestibular injury can be made to look a very dramatically damaged person in a court room.

The distinction between a round window rupture and a decompression accident must rest primarily as Bill Hurst was saying on the history. Almost invariably, at least in my experience, people who develop round window ruptures have clearing problems on descent. I have not seen a patient with this sort of problem who does not have a history of a clearing problem on descent. It may not be a very major clearing problem. It is usually associated with attempts to clear. It is not necessarily terribly painful, but sometimes it is. That history is the main thing that you can go on. An individual who has had a clearing problem and subsequently goes on to develop dizziness and nystagmus following the dive. In fact many times when inner ear windows rupture the person becomes so dizzy and so disorientated that the dive has to be aborted.

There are situations where someone could have clearing problems and go on to develop vestibular decompression sickness maybe and then presents with exactly the same thing with a history that is confusing. Dr Joe Farmer at Duke, who consults on a lot of these cases and who has in fact done a lot of the work describing these mechanisms, presently is consulting on a lawsuit case which is precisely this. An individual has had clearing problems, persisted, had a clean dive on the bottom. Subsequently he decompressed and become dizzy. The question is whether or not he had a round window rupture or whether it was a vestibular decompression sickness. The management is different in each case. Recompression on the one hand and other methods such as surgical repair on the other.

So the distinction between the two things must be made on the history. Statistically a round window rupture from a clearing problem is going to be much more common than from some other cause from the group of causes that Bill Hurst mentioned. Secondly, except under special circumstances, again I cannot really quote the numbers but I am saying essentially what my experience is, the only time I have ever seen a vestibular problem due to decompression sickness is an air-diving situation, as opposed to a deep helium dive, it has been associated with other symptoms of decompression sickness. In deep helium saturation, 1500 ft to 1800 it, this may be the presenting form of decompression sickness with no other symptoms at all. I know of a couple of people who had irreparable injury to their inner ears as a result of that particular syndrome without any other symptoms of decompression sickness.

With regard to any of the air spaces in the skull that communicate with our normal breathing passages, including in a sense the conjunctiva, may I remind you what Ed Lamphier taught in his basic course on diving medicine. That most of these structures are surrounded by bony cages which are non-compliant and the only thing that can really happen if you push it is that the pressure will equilibrate. And the only way that it can equilibrate is by bleeding. A very rapid compression, such as a submarine escape rather

than an ordinary descent in the water, can cause these sort of problems without very much pain but with sudden equilibration by bleeding.

Chairman (Dr John Knight)

I would like to remind everybody that the latest SPUMS Journal (March-June 1980) contains reports of a series of fistulae reported from Norway and one from the Royal Victorian Eye and Ear Hospital. That man did not equilibrate during his 10' breath hold dive. He got giddy on the bottom. Just before I left Melbourne a girl came back from Vila where she and her friend had dived. My girl swore she had no ear clearing problem but when you asked her about it she only cleared twice on the way down to 50'. And she had, clinically, a fistula. The other girl had Peter Freeman lift the eardrum and plug a fistula. So I think it is a much more common thing than people have thought in the past and I think we are going to see them.

Question: Dr John Miller

Bill Hurst what are the sequelae of an untreated round window rupture?

Dr Bill Hurst

They either get better, stay the same or get worse. One of the treatments of a fistula is absolute bed rest and just let the fistula heal itself. Even with surgical treatment the results can be poor. Being a true surgeon, it stands to reason that the later these people are operated on the worse the results are going to be. So if we can operate on them pretty quickly we can limit the damage that may be done from the leak and hopefully solve their problems for them.

QUESTIONS, ANSWERS AND COMMENTS

Question: Dr Mike Davis

Bill, I had hoped that you would like to talk about the mechanism of reversed block.

I thought that I would tell you a little case history of this particular problem that we had quite recently in Christchurch. He was a very competent scuba diver. He had been diving for 20 years. He never had any trouble with his ears in his life. He had had a perfectly ordinary dive doing what we tend to do round Christchurch which is trying to find crayfish. Be was in a cave having had a maximum depth of about 40'-45'. He was on a cave at 10' getting a crayfish out right towards the end of the dive. Came the time to come out of the cave and to ascend. He was totally unable to leave the cave because of a tremendous pain in one ear. He stayed there. His buddy, who happened to be a doctor as well, could not understand what was going on. This chap was just lying in the cave and every time that his buddy pointed to his contents gauge trying to get him to go up he said no by shaking his head. Finally his air ran out and he had to ascend. Hand over hand as slowly as he possibly could to the surface and for the whole time he was in excruciating pain.

When we examined his ear all you could see was a blob of blood. Two days later this had regressed and there was no apparent injury to the ear drum at all. What we assumed had happened was that air had actually tracked round the insertion of the drum separating the layers and evaginating the squamous cell outer layer of the drum down into the ear canal. By 48 hours later the drum was normal. There was no rupture. But one assumes that he had a severe ear problem.

Dr Bill Hurst

Without actually looking at the person's ear it is very difficult. Was it seen by an ENT Surgeon? (Yes). Reverse Eustachian tube obstruction presumably is due to blockage either by swelling of the Eustachian tube lining or a plug of mucus in the Eustachian tube. It is difficult to see how this guy could actually get a reverse block when he paused at 10'. I do not know. There is another thing that one can get. You being an anaesthetist can appreciate that you can get subcutaneous emphysema. Why not get it in the Eustachian tube?

Chairman (Dr John Knight)

I have a favourite theory about why these reverse blocks happen. I think that these people have a chronically unbalanced middle ear pressure, just slightly below ambient all the time during the dive. This leads to a collection of fluid in the middle ear which I suspect clots and forms a cork that is driven into the Eustachian tube by the expanding gas on ascent. It is as good as any other theory.

Question: Dr John Knight

A man or woman who suffers from presbyopia and the arms are not long enough for them to read their depth gauge. And they have to stand off the tube worms so far that they cannot really see the fine detail. I know what I do, but I would like to know what the experts would suggest.

Dr Peter Cohen

One advantage of diving is that when you dive there is magnification.

Dr John Knight

It is not enough for me to see close up detail.
Dr Peter Cohen.

Other than that there are standard lenses, very small ones, that you can glue on the face mask. They do come in varying sizes. There are

available, advertised in American Journals, stick on ones that you can buy.

There is just one point that I want to make. Your colleague who when he got to 30' could not see his main problem was retinal artery spasm, because the ambient pressure was just compressing his eyes completely. Short term it does not do any harm.

Dr John Knight

He was not wearing a mask, he was wearing goggles and they were just pushed in.

Dr Bill Hurst

I borrowed a face mask from somebody and I swam round in circles all afternoon. He has a tiny little lens down in the bottom corner of the mask. It is a one eye bifocal lens that enables him to read his watch.

Chairman (Dr John Knight)

Those of you who have seen me diving will have noticed that I wear a pair of spectacles outside my mask. So I have a bifocal that I can slide up and down the glass of the mask so that I do not have to squint through one eye.

Dr Peter Cohen

John Miller mentioned a very interesting thing that he found people who were exposed to hyperbaric oxygen developed visual problems with the retinal artery vasoconstriction that comes on with raised ambient oxygen pressure. The most fascinating thing is that I have never heard of it in adults. It has a very practical application because with retrolental fibroplasia people run around the neonatal paediatric units trying to monitor PaO2. And yet it occurs every year in any major neonatal paediatric unit. Children who develop retrolental fibroplasia without being exposed to high oxygen tensions at all. Some are even described as being normally born, normally full term and still getting it. The hypothesis has been advanced that just the change in oxygen tension from the foetal pressures to atmospheric pressures is enough to trigger the process.

Dr John Miller

Is that not the mechanism that is postulated for the shut down of the lenticular vessels?

Dr Peter Cohen

That is right.

Chairman (Dr John Knight)

I would like to let everybody know that a lot of high myopes do dive One of Australia's leading diving doctors is a high myope and he is always having his mask stolen by people who find that they can see very much better with his lenses than they can with no lenses at all. He has also had a retinal detachment and he still dives. So it is very difficult to persuade doctors to be sensible.

Dr Mike Davis

One of the mechanisms that has been postulated for fistula formation that we have not mentioned is the Valsalva itself. One of our ENT Surgeons has three cases of non-divers developing fistulae with manoeuvres which would probably increase central venous pressure.

Many years ago he was visiting a renowned French surgeon who specialised in the surgical management of this condition and he assured his visitor that he had 100% success. There were certain things that he would not allow them to do. That very afternoon they saw a young lady who came in with a recurrence of her symptom. The first question that the French surgeon asked was whether she had had intercourse and she rather bashfully admitted that she had. He said "Ah, well, what do you expect?"

Chairman (Dr John Knight)

One might say that too much sex will not only send you blind but make you deaf as well.

I was once asked to give an anaesthetic to a man who had a fistula. He was a diver but he had not burst his round window diving. He had burst it lifting a stone at work. He worked in a stonemason's yard. He picked up something rather too heavy and fixed his thorax and abdomen. Raised his intracranial pressure. And bingo, became giddy and deaf. So even if he is a diver it is not always due to diving.

Question: Dr Nick Cooper

Would there be any point in surgically draining an acutely painful sinus immediately after the dive?

Dr David Cossar

If one has a trocar and cannula in the **bo**at, yes I think that would probably be the thing.

My partner George Gray tells me that what you do is that if there is a vacuum situation and it seems more especially to relate to the frontal sinus, because of the length of the fronto-nasal duct, is you take a 3" nail and you clout the 3" nail through the floor of the frontal sinus. I said to George. Well that is pretty good because you would cure him either way. If you hit in too far you do a pre-frontal leucotomy. So he will lose his pain either

Question (unidentified voice)

Do you think that surgery has a place in the treatment of divers who have chronic problems with their sinuses?

Dr David Cossar

I think that is a very real point. I think that there is a justification for doing an endonasal antrostomy on a person who has problems

of this nature if it is a recurring problem on diving. If their desire to dive is such that they are prepared to undergo surgery. I think that it would be better to cure the mucosal cause of the blockage first. And I think that is more likely to be the conventional line of treatment. I think that there is a case that could be made for doing an endonasal antrostomy on some patients.

Question (unidentified voice)

What about using Quickenstet's test and the fistula test to distinguish between inner ear window rupture and labyrinthine decompression sickness?

Dr John Miller

Labyrinthine decompression sickness is rare.

Dr Bill Hurst

Why do Quickenstet's when you can get them to Valsalva? That is going to elevate the CSF pressure. I think that you are really going to compound the problem. For the fistula test you apply pressure to the external ear canal in people who have got the cholesteatoma. If the cholesteatoma has actually eroded into the lateral semicircular canal increasing or lowering the pressure in the ear canal is transferred through the cholesteatoma, to the semicircular canal and this causes dizziness. It is certainly not as dramatic as you get with getting a patient to Valsalva. However if you have an intact drum you are not going to get your pressure through the middle ear. So I doubt whether the fistula test would work.

Chairman (Dr John Knight)

While the experts are arguing I will make some comments. The incidence of labyrinthine decompression sickness in air diving is very low indeed. If we see one we are not going to see him within 45 minutes of it happening. The usual interval between a diver getting giddy and presenting is five days to three weeks. So we need not worry. We have got to look inside his ear and cure the fistula if he has got a hole and if he has not got a hole we cannot help him.

Dr John Miller

To continue with that. However even though there is nothing you can do for labyrinthine decompression sickness at the time, you are going to see it. What you can do for the individual is to recognise what has happened. And make sure that the patient understands the consequences of his continuing to dive.

Because if he wipes out the other labyrinth he is in problems. I generally regard this as an absolute contraindication to continuing to dive, one wiped out labyrinth.

Dr David Cossar

You keep on asking the novice to give his opinion. This is a bit hard for me. I would have thought that if you had a fistula through the round window and you subjected the middle ear to a significant pressure change while watching the ENG you would get a dramatic response on the ENG.

Dr Bill Hurst

I agree I have not seen this in the literature. I would love to get somebody with a fistula and try it out on him.

Question: Dr Janene Mannerheim

With regard to retinal artery constriction with raised oxygen partial pressures. How soon does it come on?

Dr John Miller

It happens right as you go to pressure. You do not have to wait for it at all. On compression, between 1.2 and 1.5 atmospheres usually, you just see the arteries clamp down. It happens right there. You can do it on yourself.

Question: Dr Mike Davis

Do they open up during the air breaks?

Dr John Miller

I do not know. We do not give our hyperbaric oxygen patients air breaks. They are paying for oxygen and they get oxygen.

Question: Dr Tony Slark

A bit more on this oxygen effect on the eyes. Is it useful at all in oxygen tolerance testing of divers?

Dr John Miller

I do not think so.

Dr Tony Slark

Is it a constant level of partial pressure of oxygen which causes the vasoconstriction that remains constant for each individual throughout his life?

Dr John Miller

I do not know if this persists throughout life at the same level but it certainly persists for a month at a time. This is the only length of time that I have looked at it and the only situation that other people have looked at it.

If you will accept the model of the retinal circulation being a mimicker of the cerebral circulation then may be the brain circulation is shutting down. And that happens. However recent studies using fairly sophisticated laser-type techniques, which can look at enzyme activity which are specifically oxygen linked, show that oxygen utilization is normal. Oxygen delivery is

normal and perfusion appears to be normal. Yet you see this absolute blanching of the retina. There are differences between the retinal circulation and the cerebral circulation that are sufficient to explain the difference.

Question: Dr John Knight

The man who did the very deep breathhold record dive, Jacques Mayol, wore hard, airspace containing, contact lenses. I wonder if Peter Cohen knows anything of the technology of this. Because it would be very much easier to carry your own private air pocket that did not fog up inside your contact lenses than to have to keep on swishing water around inside your mask so that you can see.

Dr Peter Cohen

No comment

PROPHYLAXIS OF MALARIA

A recent letter to The Lancet (November 15 1980, p1079) from the medical committee of the Hospital for Tropical Diseases, London gives this advice.

"The following drugs are recommended for the prevention of malaria:

"Adult doses are given. Children under 1 year: quarter dose. Children 1-5 years: half dose. Children 6-12 years: three-quarter dose. Antimalarial drugs are safer than malaria in pregnancy.

(a) *Africa, Arab States, Pakistan, India (except Eastern India), Pacific Islands*

1. *Proguanil ('Paludrine') 200mg* daily (first choice), or

2. *Chloroquine 300mg weekly.*

(b) *Eastern India, Bangladesh, South-East Asia, Central and South America, Papua New Guinea*

3. *'Maloprim' (pyrimethamine and dapsone) one tablet twice weekly; or*

4. *'Fansidar'* (pyrimethamine and sulfadioxine) one tablet weekly. Not to be taken by persons sensitive to sulphomamides. The manufacturers do not recommend Fansidar in pregnancy.

For further information see *Preservation of Personal Health in Warm Climates*, published by the Ross Institute of Tropical Hygiene, Keppel Street, London WC1."