CARDIOVASCULAR PROBLEMS AND DIVING

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In the US the Diving Alert Network has a list of physicians for medical consultation. I am one of the cardiologists on the list and I get about a call a day about some cardiac question that raises issues of safety or fitness for diving. I will discuss the commoner categories such as coronary artery disease, congenital heart disease, valvular disorders, arrhythmias, conduction disorders, pacemakers, some of the common cardiac drugs used in the treatment of hypertension and some other relatively benign medical conditions that raise questions about the use of the drugs rather than the condition itself.

Coronary disease

Coronary disease is the most common cardiovascular problem in divers and in particular divers above the age of forty. This is because of the increased incidence of cardiovascular disease in the population as age increases.

In most cases the history gives the most reliable information about coronary disease. If a patient has a history of myocardial infarction, or angina, coronary disease is present. If they have had bypass surgery or an angioplasty or any other procedure to relieve coronary occlusion, they have coronary disease. We realise now that when we see a single lesion the vessels are more diffusely involved, although all the disease may not show clearly on a coronary angiogram. One can now put small echo probes inside the coronary arteries and visualise the arteries from the inside. When that is done one finds the extent of disease is much greater than the visible narrowing on an angiogram.

Another warning is increased dyspnoea with mild exertion. Some patients do not get pain or tightness with angina, but rather get a sensation of dyspnoea. Dyspnoea on exertion, particularly of recent onset, needs to be thought about as an anginal syndrome, although one has to look for pulmonary disease as well.

Arrhythmia with exercise is a fairly ominous finding and it is always worthwhile asking people if there is a history of sudden death in their families or among relatives, because arrhythmia with exercise can be a variant of angina. Ischaemia not only produces pain or tightness in the chest, but it also can produce primary arrhythmias because of ischaemic irritability of the myocardium. Some patients do not get angina but get ventricular tachycardia as the first sign of angina. People with a history of syncope with exercise need to be considered as a special group and the whole symptom complex has to be sorted out before one can allow them to dive. In the United States, the most common cause of sudden death in diving is coronary disease.

One needs to look at the risk factors which make people more likely to have coronary disease. The common ones are cigarette smoking (the most common reason why people have coronary disease) and then hyperlipidaemia, high blood pressure, stress, lack of exercise and last of all family history which is probably the least important factor except in the very extreme hyperlidaemias.

The incidence of coronary disease increases with age. Oestrogens protect women from coronary disease until the menopause and then their rate of rise of coronary disease exceeds that of males up into the sixties and seventies. The age adjusted rates are higher in women compared with men after the age of fifty. This is important because there are many people in the United States who complete their lifetime work requirements in their fifties or sixties and want to take up a sport. Many people in their late fifties, sixties and even early seventies learn to scuba dive as a recreation. At these ages women are at the same, and probably greater, risk than men from coronary disease. So both males and females need to be tested to be sure that they do not have ischaemic cardiac disease when they start a new program, such as scuba diving, later in life. Age is a risk factor and it is the only one for which there is no pill to reverse.

High blood pressure is a risk factor and one can take a pill to reverse it. High blood pressure means a systolic pressure of greater than 150 mm Hg and a diastolic pressure greater than 95 mm Hg. High blood pressure is an important risk factor which is easy to detect. It is the number that is important. If the number is normal all is well. If the number is abnormal then something has to be done about it, as one should not tolerate high blood pressure, or even borderline blood pressure, because there is an increase in risk. Individuals who have hypertension and hyperlipidaemia, are older, smoke, do not exercise and want to dive are at very high risk. They need screening to be certain that there is no underlying cardiac disease that might put them at risk of sudden death while diving.

A person with coronary artery disease gets a progressive increase in plaque formation and reduction in lumen until an occlusion occurs and an infarct follows. There is not much change in flow reserve until around seventy percent reduction in cross sectional area, so it can be a long time before a person becomes symptomatic. It is important to test people with high levels of exercise because otherwise one cannot tell whether they have occlusions, or partial occlusions.

There are other, rarer, risk factors. For example, a person develops Hodgkin's lymphoma at age 17 and is treated with chest radiation, neck radiation and perhaps a laparotomy to look for nodes. He or she is cured. Twenty years later they want to learn scuba diving, but they have coronary disease from the radiation injury to the coronary endothelium. Someone who has had chest radiation, even though they may appear to be at relatively low risk, ought to be looked at to make sure they do not have intrinsic coronary damage producing ischaemia.

There is also coronary spasm which can cause narrowing on an angiogram. It is not a fixed lesion, but a transient one caused by active smooth muscle in the region of narrowing. Spasm is associated with local endothelial injury. The endothelium produces background vaso-relaxation so when the endothelium is not functioning normally, local spasm can occur and cause ischaemia. Local spasm in a person who has otherwise no coronary disease is very dangerous because if there are no collaterals protecting an ischaemic circulation, this will often induce ventricular tachycardia as the first symptom. Coronary spasm is very difficult to deal with because the usual treatments, such as angioplasty, stenting and even bypass surgery do not work.

An individual with a coronary artery narrowed down to about 90% would be at risk for ischaemic events while performing exercise of any kind and usually has symptoms at rest. Such a person would be at risk for ischaemia while jogging, while playing most sports, and while doing anything related to the physical activity of diving. Such a lesion, if it is known, would prevent anyone from going through a diving program. Could this person test normal? The answer is no, such a lesion would show a filling defect using thallium. Collateral circulation is unlikely to compensate for a lesion of this nature.

One such patient was a fifty five year old businessman, a very aggressive man who refused any further therapy other than anti-coagulation and aspirin. A year later, when we restudied him the narrowing was gone. Thrombosis on a minor narrowing had produced almost complete occlusion. As time went by and the thrombus cleared, the artery reopened and in fact he passed a stress test. He did not want to go diving, but he certainly wanted to go back to playing tennis and jogging, and we let him.

One should ask older candidates for diving if they have angina or if they have chest discomfort. If they experience chest discomfort while having dinner one certainly does not want to clear them for diving unless the chest discomfort is proven to be dyspepsia. If it is true ischaemic chest discomfort at rest then clearly the person should not be allowed to undertake any kind of physical activity until sorted out.

Coronary artery problems

Look for the patient who has risk factors. A forty year old man who runs three marathons a year, has a cholesterol of 110 mg, a normal blood pressure and does not smoke has a very low risk for coronary disease. If on the other hand he is 19 kg (40 lb) overweight, smokes, has diabetes, hyperlipidaemia, hypertension and takes no exercise he is at risk for a first coronary event while diving. History and the risk factors are important.

I think it is most important to look at the individual as a whole. Remember that coronary disease in the absence of any major myocardial injury shows no abnormalities on physical examination. For people with risk factors it is not at all appropriate to do a clinical cardiac examination and a resting electrocardiogram and say that they can dive, because one can not detect coronary occlusion or partial coronary occlusion purely by physical examination alone. One has to generate some stress on the myocardium and determine that the individual does not have ischaemia during that stress. That can be done by history taking if the person does a certain amount of heavy physical exertion every day without any symptoms, or if he is running a marathon three or four times a year and training doing 64-80 km (40-50 miles) a week of running. One asks about the intensity and duration of the exercise, the presence or absence of symptoms and gains the information that the individual can handle stresses. If one does not have that information and if the individual is in a high risk category then one should do a stress test to make sure there is no ischaemia present.

The easiest thing to do is a regular exercise stress test which will identify the person at risk for exercise. It will not always reveal if there is coronary disease. The problem with doing a regular exercise stress test in that one will miss the presence of some coronary disease in about twenty percent of individuals. However if there is critical coronary disease that would cause ischaemia with exercise this will usually be picked up at the rate of 90-95% on a regular stress test. It will not pick up the sub-clinical 40-50% narrowing of an artery but that is not going to cause sudden changes in the patient's status. One can pick this up with a stress test with myocardial imaging using thallium injection. Thallium goes into the myocardium using the same pathway as potassium, so if there is appropriate blood flow it will be imaged as normal distribution in the myocardium. If the individual has an occluded artery at fifty per cent or greater thallium will show that there is a partially blocked artery by demonstrating an area of inadequate uptake in the region of narrowed blood vessel.

If an individual shows severe ischaemia with exercise one must make a judgement about whether it ought to be treated. In the United States, most patients with an ischaemic response on a stress test have a cardiac catheterisation. This behaviour is changing because

outcome data show that there are many patients whose outcomes do not change whether we do an angiocardiogram and intervention or treat medically. Airline pilots with a positive stress test must have an angiogram to be able to go back on flight status. The same applies to military pilots and some other military personnel. If they have a positive stress test they need an angiogram to determine whether it is a true positive and what the extent of disease is. I would not recommend somebody have a cardiac catheterisation just to take up sport scuba diving. If one finds a high risk situation from the initial part of the examination then one could recommend it to the individual who may be at risk from other things such as the workplace or other kinds of activity.

By the time that most sport divers develop coronary disease, they already have a certification card and in the United States there is no need to come back to get recertified or requalified. People show up on a dive boat with a midline sternotomy scar and no other information. Hopefully the conscientious sport diver who has coronary revascularisation will return to a physician before going back to diving. The question that one has to ask about coronary revascularisation is whether it is complete? In some individuals one can identify a single blood vessel, perhaps a very large left anterior descending artery, that could put the patient at serious risk if it occluded. That is an indication to do a revascularisation either by a percutaneous intervention or a internal mammary bypass graft. If that is the only lesion, the ventricle is intact and no infarct has occurred, when that artery is bypassed, the person should have excellent exercise tolerance if revascularisation is complete. To get that individual back in good condition, wait for the wounds to heal and then continue very rigid risk factor modification to prevent any further progression of disease. Such people go back to their workplace without limitations, to their sports and to diving, because when tested there is no evidence of ischaemia.

Most thoracic surgeons nowadays do midline sternotomies for almost everything except descending aortic surgery. They are fairly careful to stay out of the pleural spaces. That is not always possible to do but a large number of patients with midline sternotomies do not have their pleural spaces opened. Once the sternal wound has healed the chest dynamics are pretty well restored. There are many people diving safely after having had a midline sternotomy. If the revascularisation was complete the next question is “Has there been a re-stenosis?”. This is particularly important in angioplasty, stents and anything that is done percutaneously. With balloon angioplasty the plaque is crushed and the artery torn leaving a denuded, torn, segment of blood vessel, so it is no wonder that there is re-stenosis. All these processes have a very high rate of re-stenosis. I think it is lucky that fifty percent of them heal without re-stenosis, but then fifty percent of them heal with re-stenosis. I generally tell a patient that a routine angioplasty has a fifty percent re-stenosis rate in six months. Some people get angina, have an angioplasty done and postpone their annual diving trip by a month so they can get the procedure done. They should really postpone it by six months to get past the early re-stenosis period. Having an angina attack during exercise is not a good thing while diving. They should not be diving until absence of ischaemia can be confirmed by stress testing.

Another question is “Is angina present?”. Bypass surgery and other revascularisation procedures do not always abolish angina. They may raise the threshold because there may have been severe multi-vessel disease and what was taken care of was the worst of the vessels, but not all of them. The patient still has ischaemia and will get angina with enough exercise. But the angina threshold is now high enough to allow a normal life, and return to work, but it may not be adequate to return to a sport like diving. Can one see ischaemia with reasonable workloads, and are there any arrhythmias? Again one would need an exercise test.

Congenital heart disease

People with cyanotic congenital heart disease should not be diving. Most have real trouble just existing. They are prone to pulmonary oedema. They are prone to thrombotic events because of their high haemoglobin, they have very poor exercise tolerance and their arterial oxygen saturation is usually down around 60-70% in many cases.

The atrial septal defect (ASD) is the ultimate residual of a patent foramen ovale. The flap that is supposed to close the opening does not grow, leaving a fixed opening between the two atria. This is a secundum defect. The first septum closes the juncture between the ventricles and the atrium. A septum primum defect is a much more complex lesion, often connecting the ventricles and the atrium, and often resulting in cyanotic heart disease. In secundum type atrial septal defect, the shunt goes from left to right as the left atrial pressure is higher than the right. Later in life when the lungs have been damaged by this high flow, the shunt can reverse. Even when the shunt is primarily left to right anything that raises venous pressure, whether on a long term basis or short term basis, may cause the shunt to reverse. Standing on one’s head, doing a Valsalva manoeuvre and water immersion are all ways that a primarily left to right shunt can instantaneously reverse and carry objects over into left atrium and so into the arterial circulation. Such episodes can cause strokes, so any uncorrected ASD is a contraindication to diving. Cardiologists generally recommend repair even for relatively small atrial septal defects. If the pulmonary blood flow is 1.5 times the systemic blood flow we recommend closing the shunt. In fact, we are getting even more conservative now, because of the risk of embolic events all through life, in the presence of even a small ASD.

Ventricular septal defect (VSD) is the most common shunt-producing congenital anomaly. Most are very small VSDs in the upper septum. They produce small left to right shunts, are inconsequential in terms of haemodynamics and they do not cause paradoxical embolisation. They usually produce a murmur. The only concern we have for a small ventricular septal defect is the risk of infection. These individuals are given prophylactic antibiotics when having dental work to prevent endocarditis, but often surgical repair is not recommended when the shunt is very small. Such a VSD is not a contraindication to diving or any other sport. Haemodynamically significant VSDs require surgical closure.

Patent ductus arteriosus is a left to right shunt into the pulmonary artery, the result of an incomplete closure of the foetal ductus. It does not reverse unless the patient gets severe pulmonary disease later in life and develops pulmonary hypertension. Early in life, until the thirties or forties, these can go undetected. They will produce a small murmur that is not always easy to hear. Eventually somebody will hear the murmur, do an echocardiogram and find the patent ductus arteriosus. This is not a contraindication to diving. They do not produce paradoxical embolisation. The concern is the size of the shunt because a large shunt will ultimately damage the pulmonary vasculature. If an individual has a small patent ductus and it has been decided it is not in need of surgical repair, then he or she can undertake diving without additional risk.

We heard a very good review on patent foramen ovale (PFO) from Paul Langton.[1] Basically there is an oblique hole in the atrial septum to allow arterialised placental blood to go into the left atrium and be distributed by the arterial system in utero. At birth, as left atrial pressure rises above right atrial pressure the two sides of the hole are forced into contact. In most people the flap scars down against the septum and produces a complete closure. Somewhere about 25-30% of the population has some patency of this foramen, however, the flap is always pressed up against the septum so it is haemodynamically inconsequential. There is no shunt unless one does things that force right atrial pressure higher than left atrial pressure, such as standing on one's head, being immersed in water, doing a Valsalva manoeuvre, or a cough. All those can force the foramen open and, if there are objects in the right atrium at the time, they can cross to become arterial emboli. There is a study underway (May 1995) in the US on unexplained strokes in young people. There appears to be a risk that small thrombotic emboli can pass through PFOs and cause strokes.

Wilmshurst[2] reviewed one hundred and nine asymptomatic divers, 24% had PFOs on transthoracic echocardiogram. In fifty divers with early decompression sickness (DCS) he found that 66% of them had a PFO. Late presenting DCS divers had 26% with a PFO, the same as the controls. It is interesting that he had a small group of divers with limb bends who had a low incidence of PFO, but in 14 divers who had skin bends as a primary event, 86% had a patent foramen ovale. This needs to be looked into further because it has not been corroborated by other studies. I know personally people who get skin bends frequently, with no other symptoms. One wonders whether that is related to a PFO. The 66% was statistically significant, although some people argue that the way Wilmshurst did his statistics was not quite valid. However Wilmshurst's data suggests that a PFO may contribute to early decompression sickness.

Cross[3] examined 78 professional and sport divers with echocardiography and about a third (26%) had PFOs. They had all done a lot of diving and none of them had ever had decompression sickness. I think the message here is that is if one dives safely and does not develop a lot of bubbles, it is likely that one will not get a problem even if one has a PFO.

I have done a meta analysis of four studies[2-5] of PFOs and divers (Table 1) which allows calculation of the risk of DCS. The incidence of DCS in sport divers, about 0.03%, is used as a base for the general incidence in the diving population.[6] The risk ratio for DCS is increased by a factor of about 5 for individuals with a PFO. For sport diving the overall risk remains low and the significance of these small differences is questionable. In my analysis there is a slight increase in risk of DCS in divers with PFOs. If one is diving safely it is very unlikely that there will be a problem from a PFO. One third of us here have a PFO and one third of us are not going to get bent this week.

There have been a number of studies on multiple dive profiles. Significant numbers of divers bubble, particularly in multi-dive, multi-day type exposures, four or five dives a day, five or six days a week. One will find a large number of divers who have at least one or two incidents of bubbles in that period of time.

In the DSAT studies of the PADI tables the six dives a day for six days was stopped because there was a single incident of decompression sickness, but a third of the divers had some bubbles at one time or other. In the four times a day for six days about ten percent of the divers had bubbles. When investigators went on some live-aboards and did uncontrolled surveillance of divers who were doing five or six dives a day for six consecutive days they found 18% of the divers had at least once incident of bubbles some time during their exposure. We can bubble, and it appears that bubbling occurs with a large gas load. That makes sense. Now some organisations are recommending no more than three dives a day and to take a break in the middle of a week of diving to get rid of residual nitrogen. The idea is to reduce the number of bubbles formed. I think that if a patent foramen ovale is going to cause trouble it would be in the kind of environment with increasing gas loads over a five or six or seven day period with the possibility of

TABLE 1

METANALYSIS OF PFO AND DCS PAPERS

All DCS

DAN 1994 report estimated total divers at 2,500,000.
1,164 were injured, incidence 0.05%. DCS 871, incidence of DCS 0.03%.

Source	PFO present No DCS	PFO present DCS history	No PFO No DCS	No PFO DCS history
Moon et al. 1989	19	11	157	19
Moon et al. 1991	18	66	72	24
Wilmshurst et al. 1990	26	47	83	50
Cross et al. 1991	26	-	52	-
Totals	89	124	364	93

Sensitivity	Probability PFO present and DCS history	0.571
Specificity	Probability No PFO and no DCS	0.804
Posterior Probability	Probability DCS history and PFO	0.00101
Posterior Probability	Probability DCS history, no PFO	0.00019
Posterior Probability	Probability No DCS, no PFO	0.9998
Posterior Probability	Probability No DCS and PFO	0.9990

Type II DCS

DAN 1994 report estimated total divers at 2,500,000.
1,164 were injured, incidence 0.05%. Type II DCS 644, incidence of Type II DCS 0.03%.

Source	PFO present No DCS	PFO present DCS history	No PFO No DCS	No PFO DCS history
Moon et al. 1989	19	11	157	19
Moon et al. 1991	18	29	72	30
Wilmshurst et al. 1990	26	33	83	17
Totals	63	73	312	66

Sensitivity	Probability PFO present and DCS history	0.525
Specificity	Probability No PFO and no DCS	0.832
Posterior Probability	Probability DCS history and PFO	0.00080
Posterior Probability	Probability DCS history, no PFO	0.00015
Posterior Probability	Probability No DCS, no PFO	0.9999
Posterior Probability	Probability No DCS and PFO	0.9992

Data taken from references 2-6.
Posterior Probability is the inverse of sensitivity and specificity calculated via Bayes Theorem.

bubbling some time during that exposure.

Valvular disease

The important valves are the mitral and the aortic. The pulmonary valve can be stenosed, which is usually a benign condition unless severe, but mitral valve disease and aortic valve disease are still fairly common. People with mild mitral stenosis, a leaking aortic valve, or a prosthetic valve, may all want to go diving.

Mitral valve prolapse is present in probably 10-12% of the population and as a single entity it does not have any consequence. In mitral valve prolapse the mitral valve

leaflets move behind the plane of the AV ring during systole. This process alone is a benign finding. In essence the valve can be considered too big for the orifice and it bulges back into the atrium. At 10-12% prevalence it is similar to being left handed. It would be quite unreasonable to say that everyone left handed has some kind of disease. Arrhythmias or mitral regurgitation can be associated with mitral valve prolapse. There is a very rare incidence of sudden death because of the prolonged Q-T interval syndrome. With arrhythmias, treat the person as an arrhythmia patient, with mitral regurgitation treat as a mitral regurgitation patient and with sudden death syndrome or an identified prolongation of the Q-T interval, do not let them go diving.

The concerns for both mitral and aortic valves are regurgitation and stenosis.

Critically stenosed aortic valves give the heart a relatively fixed output. They are very risky valves during exercise as vasodilatation of the skeletal muscles will reduce peripheral resistance and the blood pressure will drop. As a result the patient gets syncope with exercise and one day may not wake up. Aortic stenosis is associated with syncope and with sudden death during exercise. The question to ask is "Do you get light headed or pass out with exercise". If the response is "Yes", one needs to rule out aortic stenosis. This lesion is dangerous and is a contraindication to diving.

However there are some children with congenital aortic stenosis who grow up getting along fine. I have patients who jog five miles a day in spite of significant aortic stenosis. I say do not do it. And they come back a year later still jogging five miles a day. But for the most part, aortic stenosis, with a critically narrowed valve (about one square centimetre or less) is a contraindication because of the risk of syncope or sudden death during exercise.

Mitral stenosis is of concern because of elevated pulmonary venous pressure. When the cardiac output goes up with exercise the flow across the mitral valve increases, the left atrial pressure rises, pulmonary venous pressure rises and the patient develops pulmonary oedema. This seldom produces sudden death. In patients with mitral stenosis occasionally the first evidence is pulmonary oedema with exercise. The patient with mitral stenosis should not be diving for the same reason that they cannot go jogging or do other forms of exercise, because they get lung congestion. If there is a minimal pressure gradient and no significant obstruction with good exercise tolerance, they can go diving. But moderate to severe mitral stenosis is a contraindication.

Severe mitral regurgitation, like mitral stenosis, will cause elevations of pulmonary venous pressure during exercise witth lung congestion and pulmonary oedema. People with moderate to severe mitral regurgitation should not be diving. When one is immersed in water about 500 ml of blood shifts into the central circulation. This can cause problems. A 58 year old physician, who of course signed off that he was in excellent health and had no medical history, had trouble breathing during a dive. We pulled him from the water in acute pulmonary oedema. Fortunately there was oxygen on board and with that he improved. It turned out that he had significant aortic regurgitation and had actually had endocarditis once. The combination of exercise and the central fluid shift gave him acute pulmonary oedema. We treated him and he was fine, but somehow this physician inadvertently forgot to tell us that he had severe aortic insufficiency!

When mild, aortic regurgitation and mitral regurgitation are of little consequence. If severe they produce heart failure during exercise which means dyspnoea on exertion and sometimes pulmonary oedema during exercise. They seldom result in sudden death during exercise. Mild regurgitant lesions are benign. One has to examine the individual's exercise tolerance and how cardiac function responds to the workload.

Mild stenotic lesions need to be carefully examined to make sure that they are not producing significant obstruction.

There are people with prosthetic valves diving, although the Divers Alert Network (DAN), in the USA, usually tells people with prosthetic valves not to dive. In the absence of any other cardiac disease the prosthetic valve will allow an individual to perform moderate amounts of exercise which is fine for diving. People with prosthetic valves play tennis, jog, play other sports and they do not have serious problems. The issue with prosthetic valves is anti-coagulation. DAN has raised the issue of having haemorrhagic otitis media with an ear squeeze. I have seen twenty or thirty people diving with prosthetic valves and none of them has ever had a problem with bleeding from an ear squeeze. Obviously they need to teach themselves to be careful with their ears. Bleeding does not seem to be a major concern although the individuals need to be warned about injuries and about making sure their ears are clear when they dive. A sinus squeeze would be likely to cause a haemorrhagic sinusitis.

Arrhythmias, conduction defects and pacemakers

People who get supraventricular tachycardia (SVT) with exercise, or spontaneously, should not be diving. SVT with exercise in subjects with accessory pathways can degenerate into sustained tachycardia and ventricular fibrillation. Supraventricular tachycardia is quite common. It appears that somewhere around 60% of young people with attacks of SVT have an accessory bypass track connecting the atrium to the ventricles or the AV node which is inducing the supraventricular tachycardia. They are treatable by catheter ablation or medication and treatment

generally stops the arrhythmia. If somebody has a serious recurring supraventricular tachycardia tell them to have it treated and come back for evaluation when the rhythm has been eliminated.

These arrhythmias can be induced by stimulants, such as caffeine, decongestants that have adrenalin like compounds, amphetamines and stress. Remove the stimulants and often the arrhythmia goes away.

There are some young people, with no evidence of cardiac disease, who have atrial fibrillation. They are well controlled and have excellent exercise tolerance. They may need to be on anti-coagulation because of the risk of embolisation, but they can dive. The problems of anti-coagulation need to be carefully explained.

I do not worry about premature ventricular contractions (PVCs)when they disappear with exercise. If they do not, or the patient has PVCs associated with ischaemia, these PVCs may increase in frequency and induce severe rhythm problems when diving.

There are many pacemakers that sense the need for increased output with exercise and adjust the cardiac rate to accommodate the exercise. Rate controlled pacemakers allow people to exercise normally. For young individuals who need a pacemaker, perhaps after a viral infection destroys the conduction system or congenital heart block, a rate sensitive, dual chamber pacemaker, will allow normal function for all physical activity. The pacemakers are made to withstand pressures to about 39 m (130 ft). As these individuals have otherwise normal hearts they have normal exercise tolerance and can take up sport diving. However they will not get into military or commercial diving.

An implantable defibrillator is implanted because an individual has had an episode of sudden death. Normally sudden death induces unconsciousness and the theory behind the implantable defibrillator is that when one gets a run of ventricular fibrillation blood flow to the brain stops for a few seconds so when the shock comes one does not remember it. Unfortunately in water, going unconscious for twenty seconds before the defibrillator fires means that one is already drowning. I do not advise diving for these people. I know of two divers who entered the water with these devices. One was a seventy year old with severe heart disease, who should not have been diving. His fired and a diving instructor next to him also felt a mild shock! The other was an already certified diver, who at the age of 36 had a sudden death episode and had a defibrillator implanted. He asked, by letter, whether it was reasonable for him to dive. I said that I did not think so, because sudden death in the water is not good for the diver or the buddy. It is very upsetting when your diving buddy dies right in front of you. But while my letter was in the post I received another letter from him saying "All is well, I already did a forty foot dive. Do you think I could go to eighty feet?" I sent him the same letter again saying "Do not do it", but all we can do is advise.

Fitness

One can assess conditioning by measuring maximum oxygen consumption. When one starts exercising there is an increase in oxygen consumption which peaks at a certain work intensity. Everyone has a peak beyond which one cannot increase one's 0_2 intake. Above 70% of one's peak one is into anaerobic exercise, generating a lot of lactate, becoming acidotic and hyperventilating, ultimately one has to stop exercising. The maximum can be varied by conditioning. People with a low maximum oxygen consumption have less exercise tolerance and so are more at risk for getting into trouble when they need to exercise in the water.

The maximum one can achieve declines with age. If you are thirty years old and in reasonably good condition your maximum would be about 40 ml of oxygen per kg per minute. That is about 13 mets. Around age seventy the maximum is down to about 30 ml. The seventy year old diver is not going to be able to handle the extremes of exercise needed to extricate himself from serious problems in diving the way the reasonably fit thirty year old could. The best way to cope with the differences in physical capacity with age is to gain wisdom so that one stays out of trouble.

For conditioning we suggest a target heart rate of 0.7 x (220 - age). Warm up for 5 minutes, exercise at the target heart rate for about 30 minutes then do a five minute cool down. Doing this four or five times a week will get most people in good condition for diving. For a diver it is sensible to do swimming as part of this program because swimming is what one is training for.

Antiarrhythmic Drugs

Most drugs are not of concern in a patient with true heart disease. Someone with heart failure who is on digitalis will not be fit to dive because he has heart failure and poor exercise tolerance. The same applies to diuretics. Perhaps even more, as in a warm environment, with sweating and loss of sodium and fluid by normal means, one does need diuretics. One must warn people taking diuretics that if they are in the tropics they ought to reduce the dose to make sure they do not get dehydrated.

Digitalis is often used, probably incorrectly, to prevent supraventricular tachycardias, particularly in young people. I meet many people in their twenties and thirties who are on digitalis because they had one episode of tachycardia associated with a long night of alcohol intake. I usually stop the digitalis, although if someone has been on a drug for fifteen years it is difficult to get them to stop it.

I do not like to see people diving while taking anti-arrhythmic medications. The reason is that most of them have cardiac problems. The first thing one has to understand is what their rhythm is.

For example we are using amiodarone to prevent atrial fibrillation in people who have paroxysmal atrial fibrillation. Amiodarone is an extremely good drug for managing atrial fibrillation. In fact it is much better than a combination of three or four other antiarrhythmic drugs. A 30 or 40 year old person may be on a small dose of amiodarone to prevent atrial fibrillation, and that is a very effective treatment. A small amount of amiodarone (100 to 200 mg daily) is a benign dose. There is nothing wrong with diving with a small amount of amiodarone. However, amiodarone sensitises the skin to the sun. Many medications have other effects that one needs to warn the patient about. Amiodarone is one that causes skin sensitisation. If somebody takes 800 mg of amiodarone to prevent sudden death syndrome, that is not acceptable, because such doses cause significant problems with the lungs. the eyes, the liver and the thyroid. Large doses of most other antiarrhythmic drugs cause side effects that can be complicated by diving. Small doses, for the most part are not necessary so when a patient is on a small dose of anti-arrhythmic one can be sure, first, that it is not doing very much and, secondly, that the small dose is not going to bother the individual for diving. What one must do is to gently nudge the patient's physician to find out why the patient is still using this medication. In general the antiarrhythmic drugs in small doses are benign, but the question one must ask is what are they being used for.

Anti-hypertensive drugs

Beta-blockers, ACE inhibitors and calcium channel blockers, with or without a diuretic, are the common anti-hypertensive treatments. They are all benign. Beta blockers will reduce exercise tolerance at its maximum. If one is reasonably well conditioned one is not going to need maximum effort so the beta blockers are not going to limit one's exercise capacity. Sometimes the calcium channel blocker will cause orthostasis. I would be reluctant to let anybody taking high doses of antihypertensives dive. But moderate doses which have minimal side effects, are not a problem with any of the medications.

Audience participation

Unidentified speaker

What cardiovascular screening would you do on mature age diving candidates (I judge mature age as anyone older than myself)?

Bove

The age of 40 is a good base line. Well conditioned individuals, who have no risk factors, that is normal blood pressure, not overweight, no diabetes, non-smoker and with a good exercise tolerance by history, between 40 and 50 would not need to be screened with an exercise test.

With risk factors I think one should screen with at least a routine stress test. That is put the patient on a bicycle ergometer or a treadmill, take a continuous electrocardiogram and check the blood pressure while they are exercising. If they do not show ischaemic changes with moderate amounts of exercise, that is exercise up to around 10 or 11 mets, then they are probably fit for diving. But that does not say that they are free from coronary disease. All it says is they do not have a critically obstructive coronary lesion that would put them at risk for any kind of exercise.

Above the age of 50 I would do much the same but I would be more likely to do a stress test even though I knew the patient was exercising adequately. Beyond about 55 I think everybody ought to have a stress test.

Unidentified speaker

What speed of running is associated with 13 mets.

Bove

About an 8 minute mile (4.9 minute km). However this a maximum capacity. A steady capacity would be 70% of 13 mets which is equivalent to a 10 minute mile (6.25 minute km).

Unidentified speaker

The greatest risk, of course, is not at entry into diving, when we get our diving medical done at 17 and we are fit. We should screen 40 year olds and upwards before they start diving. But what should we do for aging divers ?

Bove

A good history of physical activity, with low risk factors, eliminates the need for stress testing.

Unidentified speaker

Is there any evidence that people with mild valvular disease having a higher incidence of bubble formation through induced turbulence?

Bove

No there is not. I spent four summers as a medical student studying cavitation in stenotic valves. We were never able to show that it occurred.

References

1 Langton P. Patent foramen ovale in underwater medicine. SPUMS J 1996; 26 (3): 186-191

2 Wilmshurst PT, Byrne JC and Webb-Peploe MM. Relation between interatrial shunts and

decompression sickness in divers. *Lancet* 1989: ii: 1302-1304
3 Cross SJ, Evans SA, Thomson LF, Leed HS, Jennings KP aand Shields TG. Safety of subaqua diving with a patent forament ovale. *Brit Med J* 1992; 304: 481-482
4 Moon RE, Camporesi EM and Kisslo JA. Patent foramen ovale and decompression sickness in divers. *Lancet* 1989: i: 513-514
5 Moon RE, Kissolo JA, Wassey FW, Fawcett TA and Theil DR. Patent foramen ovale (PFO) and decompression illness. *Undersea Biomed Res* 1991; 13 (Suppl): 15
6 *Report on diving accidents and fatalities*. Durham,South Carolina: Divers Alert Network, 1996

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PATENT FORAMEN OVALE IN UNDERWATER MEDICINE

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Abstract

The foramen ovale, between the right and left atria, exists in the foetal heart as a vital physiological communication. Haemodynamic closure occurs in the neonatal period with most people having permanent fusion of the foramen. In up to a third of adults the closure is functional only and a potential right to left atrial communication persists as a patent foramen ovale. Studies in patients with decompression illness after diving suggest a consistent increase in the prevalence of patent foramen ovale, as detected by transthoracic contrast echocardiography. The association is strongest for those patients with early onset of neurological decompression illness, particularly those cases occurring in the absence of other risk factors traditionally associated with decompression illness. However, patent foramen ovale is a common finding in the general population and the absolute risk of decompression illness, even in the presence of a patent foramen ovale, remains very low.

Key Words

Decompression illness, heart conditions, investigations.

Introduction

There has been considerable interest in the potential contributory role of the foramen ovale in the development of decompression illness (DCI) and arterial gas embolism (AGE) in SCUBA divers. Venous bubble formation is known to occur during hyperbaric gas exposures well within the recommended limits of recreational diving.[1] The relative absence of clinical decompression sickness is thought to be related to the filtering of venous bubbles as they pass through the pulmonary circulation, thus preventing systemic arterial exposure. It is proposed that the presence of a patent foramen ovale (PFO) allows venous bubbles to pass across the interatrial septum into the left heart and then into the arterial circulation, with the potential to cause AGE.

Background

The foramen ovale exists as a vital physiological communication between the right and left atria during foetal life. Atrial division (Fig 1) initially occurs with the formation of the septum primum, a crescentic structure grows from the top of the common atria and fuses with the endocardial cushions that demarcate the atrioventricular junction. As it develops some of the central tissue of the septum primum beaks down to create the foramen secundum, maintaining interatrial communication. The septum secundum then grows from the right superior margin of the septum primum to incompletely divide the atria; it remains deficient inferiorly, against the endocardial cushions. The combined atrial septum (primum and secundum) thus forms the foramen ovale and allows oxygenated inferior vena caval blood (returning from the placenta) to be directed across the atrial septum to the left heart and thenceforth to the developing brain (Fig 2). In contrast, deoxygenated (superior vena caval) blood streams preferentially from the right atrium through the right ventricle to the pulmonary circulation and then via the ductus arteriosus back to the placenta. The foramen ovale remains open in the foetus because of the existence of significantly higher pressure in the right atrium as compared with the left.

The physiological changes that occur at birth include a profound lowering of pulmonary vascular resistance secondary to lung aeration, and a fall in right atrial pressure. At the same time systemic pressures increases, with a rise in left atrial pressure and hence the functional closure of the foramen ovale (Fig 3). In most infants this functional closure is followed by fusion of the flap like membrane, forming the fossa ovalis. In about a third of individuals fusion does not occur and a potential inter-atrial shunt persists as a PFO. For shunt flow to occur however the right atrial pressure must exceed that in the left atrium. The phasic nature and right to left flow of a PFO help distinguish this anatomical variant from an atrial septal