

HEART ATTACKS WHILE DIVING

Recreational scuba diving is a 'safe' activity. Never-the-less, approximately 100 people die every year in North America and 200 people world wide. The most common causes of death are having a heart attack while diving and arterial gas embolism (AGE). I discussed AGE in issues #124/5 (Oct/Nov08 and Dec08/Jan09) of Sportdiving. In this column I will discuss divers who die as a result of having a heart attack while they are diving.

If you review statistics for diving fatalities, the most common cause of death listed in autopsy reports is always 'drowning'. The person died while they were in the water so they must have drowned. Unfortunately, this is the level of investigation in many cases.

What we really want to know is why did they drown? In diving fatalities the information available is almost always incomplete but sometimes we are able to determine the most likely cause of drowning. In these cases the most common cause is a problem with the heart, followed by arterial gas embolism.

There are several reasons why a diver might suffer a heart attack while diving. The most obvious is simple statistical probability. Heart attacks are a relatively frequent event. Divers spend a fair amount of time diving. Therefore, pure chance will result in some people suffering their heart attack while they are diving.

However, there are a few reasons why diving itself increases the risk of having a heart attack while diving. Before we get into these reasons, let's review why people have heart attacks in the first place.

Heart muscle is different from any other muscle in the body. In a normal muscle cell, an electrical potential is maintained across the cell membrane. When you want the muscle to contract, your brain sends a signal down the nerve to the muscle cell causing the electrical potential to reverse and the cell to contract. When the electrical potential in one cell reverses, this change tends to travel to

adjacent cells, causing them to contract as well.

The cells in the heart are different in that their cell membranes 'leak'. Therefore, even if no signal is received from the nerve, the heart muscle cell will spontaneously contract and this contraction will spread to adjacent cells until the entire heart has contracted. For the heart to pump blood effectively, this contraction has to start in the correct location and proceed in an orderly fashion.

In the normal heart there is a small area in the atria where the cells spontaneously contract at a rate of approximately 70 times per minute called the sinoatrial (SA) node (this is faster than other heart cells). The signal is spread from the SA node to the rest of the atrial muscle cells and to the junction between the atria and the ventricles where it is delayed by the atrioventricular (AV) node. This delay is important because it gives the atria time to contract and move blood into the ventricles before they contract. After the delay at the AV node, the signal travels down Purkinje fibers to the ventricles and then from cell to cell causing the ventricles to contract from the bottom up, forcing blood out of the valves at the top of the ventricles; to the lungs on the right side and the body on the left side.

This entire process is automatic but can be influenced by both nerve and chemical signals. Stimulation of different nerves can result in slowing or speeding up of the heart. Releasing chemicals into the blood (like adrenalin) can also result in slowing or speeding up of the heart. When a person receives a heart

transplant, there are no nerves going to the transplanted heart. It beats at its own innate rate. When the person starts to exercise, chemicals released into the blood cause the heart to speed up.

This system of electrical and chemical control of the heart is far more complex than I have explained so far and can malfunction. Some electrical malfunctions result in heart muscle cells contracting erratically and as a result, the heart stops pumping blood. Some malfunctions result in the heart beating so rapidly that there is no time for blood to flow into the heart between beats. This also results in very little blood being pumped by the heart. In both cases there will not be enough oxygen delivered to the brain and the person will lose consciousness. If this happens while they are diving, or even in the water, the end result will usually be drowning.

However, fatal electrical conduction problems in a normal heart are relatively rare. Usually the heart is abnormal in some way and these chronic changes are identifiable at autopsy. These changes increase the risk of a heart attack, but if the heart attack is a result of a conduction problem there will be no additional changes at autopsy.

The most common cause of a heart attack is obstruction of one or more of the arteries that supply blood to the heart muscle. You may never have thought about it before, but the blood being pumped through the heart does not supply oxygen or nutrients to the heart muscle. As blood leaves the left ventricle through the aortic valve, some of the blood enters the two main coronary

arteries, located behind the valve leaflets. These arteries deliver blood to the heart muscle.

Over time, most people develop changes in these arteries that result in slowly increasing obstruction to the flow of blood through them. This is called 'coronary artery disease' (CAD). When we exercise, our heart has to pump more blood to the working muscles. It does this by beating faster and by pumping more blood per heart beat. To do this, the heart muscles have to work harder.

Heart muscle is again different from other muscles in our bodies. Most muscles

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contain energy stored in special molecules (glycogen) that allow the muscle to perform work for a short while even if there is no oxygen being delivered to the muscle by the blood. Heart muscle does not contain these energy stores. In addition, most muscles remove only some of the oxygen from the blood flowing through them and when they are working they can remove more oxygen from the blood. Heart muscle removes the maximum amount of oxygen from the blood flowing through it all the time. As a result, when the heart has to work harder the only way this work can occur is for more blood to be delivered to the working muscle.

If one of the arteries supplying blood to heart muscle is less than 50% obstructed by CAD, the person can usually work without developing any symptoms. However, if more than 70% of the cross-sectional area of the artery is obstructed the person will usually have no symptoms

at rest, but when they work they will experience symptoms.

If we maximally work an arm or leg muscle it will start to hurt. This is due to accumulation of waste products in the muscle (they are being produced faster than they can be cleared). When we rest, the waste products are cleared away and the pain resolves.

A healthy heart can work maximally without problem. However, if blood flow to the heart muscle is reduced by CAD, the working muscle will accumulate waste products and generate symptoms. An arm or leg muscle hurts. A stressed heart muscle will produce symptoms like pain in the left shoulder and arm, a sensation like there is a band around the chest preventing you from taking a deep breath, and/or a feeling of impending doom.

These symptoms start with exercise and go away with rest. The amount of blockage can be estimated by the amount of exercise required to bring on the symptoms. If they only happen with very heavy exercise, the blockage will be less than if they happen every time you try to walk more than a few meters.

This process is called 'angina pectoris' and is often treated with nitroglycerin. Nitro causes the arteries of the heart to dilate, thereby increasing blood flow to the heart muscle. The three major problems with angina are that if it continues too long without treatment the oxygen deprived heart muscle can die, it can cause a sudden electrical problem in the heart, and the plaques that are causing it can suddenly rupture and completely block the artery.

This is a typical heart attack and a medical emergency. Heart cells will start to die in a few minutes if blood flow is not restored. This dead and dying heart muscle does not pump blood. If enough heart muscle is involved, the person will lose consciousness and if they are

diving, almost certainly drown. Anyone with angina has a higher risk of sudden death due to a cardiac arrhythmia or myocardial infarction. Unfortunately, many people do not suffer from angina before they have their first heart attack.

There are two main reasons diving may increase the risk of having a heart attack. First, when we enter the water the effects of gravity are removed. This results in the rapid movement of blood from the arms and legs into the chest and heart. If the heart is abnormal (diseased) this sudden

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Nitrox and Technical Divers (IANTD) since 2000, and is an active cave, trimix and closed circuit rebreather diver/instructor/instructor trainer. David's first love is cave diving exploration and he's been exploring and surveying underwater passages in Canada since 1985. David was responsible for the exploration and mapping of almost 11 kilometres of underwater passages in the Ottawa River Cave System. In 1995, he executed the first successful rescue of a missing trained cave diver. David received the Canadian Star of Courage for this rescue which took place in the chilly Canadian waters of Tobermory, Ontario. He still dives as much as possible, but admits his six year old son Lukas, five year old daughter Emeline and wife (Dr Debbie Pestell) are currently higher priorities than diving!

diving medicine

expansion of the heart might cause an electrical abnormality.

The second reason diving may cause a heart attack is that diving is relatively hard work. Simply carrying all of the gear onto the boat or to the shore, getting dressed and into the water, is far more taxing than most daily activities. Routine recreational diving is roughly equivalent to playing basketball and fighting current while trying to swim back to the boat/shore or getting out of the water can be far more stressful. The end result is that many divers suffer a heart attack while they are diving as a result of the 'exercise' of diving. The first major snowfall in winter always results in emergency departments being flooded with heart attacks after people try to shovel the snow.

As a rough guide, recreational divers should be fit enough to run a mile in less than 10 minutes (6.25 min/km), have a

VO2 max of at least 35 ml/kg/min or be able to exercise at 10+ Mets. For technical/commercial divers I recommend being able to run a mile in less than 8 minutes (5 min/km), have a VO2 max of at least 45 ml/kg/min and be able to exercise at 13+ Mets. In addition, a diver needs enough muscle strength to safely carry and handle their gear. Diving can trigger a heart attack so make sure that you are 'fit' to dive.

