Arterial dissection in scuba divers: a potential adverse manifestation of the physiological effects of immersion

Neal W Pollock[1,2], John Lippmann[3,4], John Pearn[5], John Hayman[6,7]

[1] *Department of Kinesiology, Université Laval, Quebec, QC, Canada*
[2] *CISSS Chaudière-Appalaches (CHAU-Hôtel-Dieu de Lévis), Hyperbaric Medicine Unit, Emergency Department, Lévis, QC, Canada*
[3] *Australasian Diving Safety Foundation, VIC, Australia*
[4] *Department of Public Health and Preventive Medicine, Monash University, Clayton, VIC, Australia*
[5] *Queensland Children's Hospital, South Brisbane QLD, Australia*
[6] *Department of Clinical Pathology, The University of Melbourne, VIC, Australia*
[7] *Menzies School of Health Research, Casuarina, NT, Australia*

Corresponding author: *Associate Professor Neal W Pollock, Université Laval, Quebec, QC, Canada*
neal.pollock@kin.ulaval.ca

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Abstract
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Introduction: Aortic dissections and dissections of cervical, cerebral, and coronary arteries have been previously reported in scuba divers. These incidents may be the consequence of a variety of physiological effects. We review the reported cases of arterial dissection in scuba divers and discuss potential contributing factors related to immersion and diving.
Methods: Medline, CINAHL Plus, and SPORTDiscus were searched for published reports of arterial dissection and the Australasian Diving Safety Foundation fatality database was searched for additional cases from Australia. Identified cases were recorded and scrutinised for possible contributing factors.
Results: Nineteen cases of arterial dissection, both fatal and non-fatal, were identified. These included cervical or intracranial artery dissection (n = 14), aortic dissection (n = 4), and coronary artery dissection (n = 1). There were 14 male and five female victims; mean age 44 years (SD 14, range 18–65). Contributing factors may include a combination of vasoconstriction and blood redistribution, untreated hypertension, increased pulse pressure, abnormal neck movement or positioning, constrictive and burdensome equipment, exercise, increased gas density and circuit resistance with concomitant elevated work of breathing, atheroma, and possibly the mammalian dive response.
Conclusions: Dissecting aneurysms of the aorta or cervical, cerebral, and coronary arteries should be considered as a potential complication of scuba diving. The development of aneurysms associated with scuba diving is likely multifactorial in pathogenesis. Detailed reporting is important in the evaluation of cases. The potential role of the mammalian dive response as a contributing factor requires further evaluation.

Introduction

Underwater diving is used worldwide for a range of recreational, commercial, scientific, and military operations. Although most diving is to depths less than 40 metres of seawater (msw), divers may descend to much greater depths and stay underwater for extended periods. Scuba apparatus has evolved substantially in recent decades, but its use still requires appropriate selection, training, maintenance, and implementation. Although such equipment enables a diver to breathe underwater, usually in relative comfort, it introduces certain potential complications that need to be managed.

Incidents, including fatalities, do occur in diving. Many deaths result from human factors such as inexperience, impaired health and fitness, and failure to adhere to safety guidelines.[1–3] However, more than one-quarter of scuba fatalities have reportedly been associated with cardiac causes, often reflecting health issues such as obesity, poor physical fitness, and an aging diver cohort.[1–4] It is recognised that clinically silent, intermittent cardiac conduction abnormalities can be triggered during diving.[5] Significant ischaemic heart disease and other pre-existing cardiac conditions may also precipitate dysrhythmias while diving.[6] Dissecting arterial and aortic aneurysms are also possibilities. Although rare, cases have been reported in sufficient number for this to be accepted as a diving hazard.[7–25]

Arterial dissection involves tearing of the intima, splitting of the media, with blood tracking through the divided vessel wall, which can lead to malperfusion of end organs, and rarely, arterial rupture.[26] Neck artery dissections may occur through a variety of physically violent events and blunt trauma, but may also occur after less violent events such as exercise stretching or chiropractic neck manipulations.[27,28] Aortic dissection has also been reported in conjunction with weightlifting.[29]

There are several classification systems for aortic dissection, with the Stanford classification the most commonly used. The Stanford classification includes two types: in type A the entry tear originates in the ascending aorta but may continue into the descending and abdominal aorta; and in type B the entry tear originates in the descending aorta but can extend in a retrograde fashion through the arch and ascending aorta.[30] Of the two, type A is the more lethal. Both types can be temporally classified as acute, sub-acute, or chronic in nature.

The risk of arterial dissection in scuba divers is likely increased by a host of factors, including the physiological effects of immersion, notably centralisation of blood, in addition to other burdens associated with diving, such as equipment worn, workload pre-, during, and post-dive, and general or other environmental stressors. It is an open question as to whether there may be a contribution of the mammalian dive response.

The dive response is a complex physiological reflex, well developed in diving mammals. The effects seen during breath-hold include bradycardia, increased systolic blood pressure, constriction of cutaneous, muscular, and splanchnic vessels, and preservation of the blood flow to the brain and heart.[31] The overall effect is to preserve oxygen for these two vital organs while reducing overall oxygen consumption in tissues more tolerant of hypoxia. The practical cardiovascular effect is an increase in systolic and pulse pressures, primarily confined to cervical, cerebral, and coronary arterial vessels and the aorta (Figure 1). The pressure increase is augmented by an increase in venous return to the right heart as a result of peripheral vascular constriction.

The dive response is present in all human neonates, diminishing in intensity by 12 months of age, but remaining as a substantial response in most adults.[32] In humans the response involves the afferent arc of the trigeminal (fifth) cranial nerve; the area of response activation includes the forehead which is innervated by ophthalmic branch of that nerve.[33]

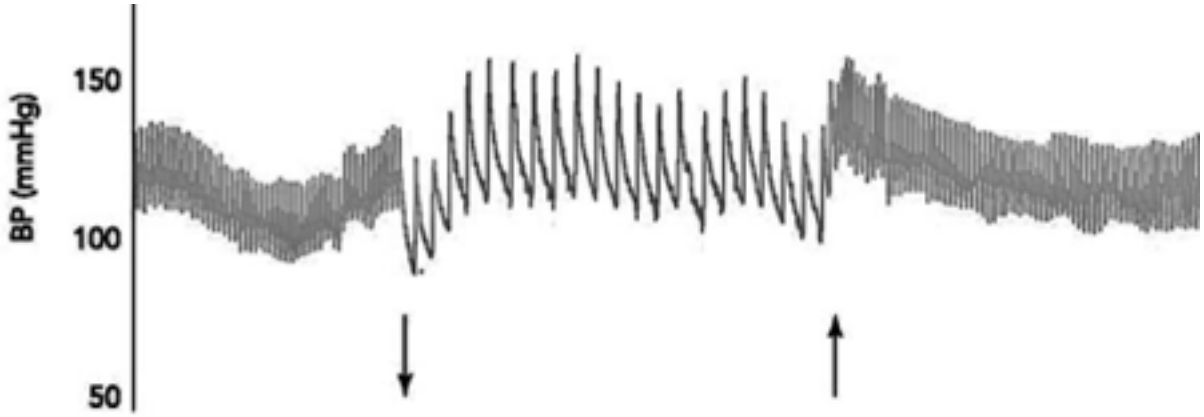
The classic evidence for dive response in humans is seen with breath-hold and facial immersion, particularly in cool or cold water. The effect would be blunted by the continued breathing associated with scuba diving, and likely also by the mask and often a hood covering a substantial portion of the face, but it is possible that the dive response pathway could augment a blood pressure spike if activated. The aim of this report is to discuss potential contributing factors to arterial dissection in scuba divers including the possible contribution of the dive response.

Methods

A literature search was conducted using Medline, CINAHL Plus and SPORTDiscus using the search terms "*arterial dissection*" or "*artery dissection*" or "*coronary artery dissection*" or "*aortic dissection*" or "*dissecting aneurysm*" AND "*scuba*". In addition, a search of the Australasian Diving Safety Foundation (ADSF) diving fatality database

Figure 1

Pulse and blood pressure recording from a rat trained to swim underwater through a tunnel; down arrow – start of dive; up arrow – end of dive. There is a marked bradycardia beginning immediately at the commencement and ceasing directly at the end of the dive. The blood pressure rise is slightly delayed and persists a short time after the dive. The pulse pressure increases from 20 mmHg to 40 mmHg during submersion. From WM Panneton, with kind permission



(adsf.org.au) was conducted to find any further documented fatalities attributed to aortic and arterial dissection.

As the dive response pressure changes also affect the coronary arteries a search was made for coronary artery dissection associated with scuba, using the search terms "*scuba*" AND "*coronary artery dissection*."

Results

The available case data are summarised in Table 1. The results are divided into three sections based on the location of the dissecting aneurysm – aorta, cervical and/or intracranial arteries, and coronary arteries. Descriptions of the dives, potential contributing factors, and the timing of problem development relative to diving were included as available.

AORTIC DISSECTING ANEURYSMS

One of us (author JH) reviewed and reported an early case of aortic dissection in a scuba diver and was aware of a second.[7,8] Two additional cases were more recently recorded.[9,10] All victims were male, and all dissections were type A, with onset evident during or within minutes of surfacing. Documentation of predisposing disease was limited. There was no evidence of contributing factors in the youngest victim (case 1 – 20 years of age), no information for one (case 2), and evidence of ischaemic heart disease and hypertension in cases 3 and 4. Three of the four cases were fatal, each of these reported in Australian waters. The most recent case had the only non-fatal outcome.[10]

CERVICAL AND INTRACRANIAL DISSECTING ARTERIAL ANEURYSMS

Four articles presented case reports, each listing cases of previous cervical artery dissections associated with scuba diving.[15,16,18,19] Five additional cases were identified in the search. A total of 14 cases were identified with age ranges from 18 to 60 years; four were female (Table 1). Unlike the aortic dissections there were no fatalities and the diagnoses were made based on clinical and radiological features. The onset of symptoms relative to diving was variable, including two cases during dives, six cases within minutes of surfacing, three cases within hours of surfacing, two cases within two days of diving, and one case with an unknown point of onset.

Fourteen cases involved the cervical or intracranial arteries (Table 1). Nine involved only the carotid arteries (cases 5, 8, 9, 10, 12, 13, 16, 17, 18), two only the right vertebral arteries (cases 7 and 14), and one involved both carotid and both vertebral arteries (case 6). The nine carotid artery-only cases included six involving only the left internal carotid artery (cases 9, 10, 12, 13, 17, 18), one involving only the right internal carotid artery (case 8), one involving both internal carotid arteries (case 5), and one involving both common carotid arteries (case 16). Seven of the 14 cases exhibited signs of Horner's syndrome (9, 10, 12, 13, 16, 17, 18).

Most internal carotid artery dissections occurred at the base of the skull, proximal to the entry of the artery into the carotid foramen (Figure 2). The one exception involved a 35-year-old woman with multiple dissections (case 6). In this instance, the internal carotid dissections occurred at the origin of these vessels from the common carotid arteries. The two vertebral artery cases occurred before the artery entered a cervical vertebral transverse foramen.

The two intracranial cases (11 and 15) involved the left posterior cerebellar artery, originating from the left vertebral artery, and the left anterior cerebral artery, a terminal branch of the left internal carotid artery.

One patient (case 6), with multiple cervical artery dissections, had evidence of osteogenesis imperfecta, a collagen disorder reported as being associated with dissecting aneurysm.[34] This person had only minimal clinical evidence of the disorder but developed the dissections within minutes of her first dive experience. Two patients were known to be smokers (cases 12 and 17, the latter also diagnosed with stable multiple sclerosis), and one was known to have treated hypertension (case 13). None of the remaining patients had reported conditions predisposing to arterial dissection.

CORONARY ARTERY DISSECTION

Only one case of coronary artery dissection has been reported in association with scuba diving.[25] This was a 65-year-old female (case 19) with no known risk factors. Chest pain developed during descent on her ninth dive in five days. She developed a myocardial infarction as a result of the dissection but there was resolution of the arterial lesion with anti-platelet therapy.

We are unaware of any reports of arterial dissection of the splanchnic and peripheral arteries in scuba divers.

Discussion

Head and neck artery dissections have been reported associated with a wide range of sporting activities as well as from neck manipulations and trauma.[22,27,28] Some involved abnormal neck movements, such as chiropractic manipulation, others blunt trauma, such as in karate, and some seemingly innocuous activities such as jogging. Not infrequently there are no obvious potentially precipitating events.

All four cases of aortic dissection (type A) affected the initial segment of the aorta, which includes the areas directly or indirectly connected to the origins of the cervical arteries. It is logical to infer that the factors leading to aortic dissection are the same as those responsible for the dissections in other arteries. If aortic dissections are included, the evidence for a connection between vessel dissection and scuba diving is substantial.

Table 1

Sex, age, anatomical locations of dissecting arterial aneurysms, and possible contributing factors reported for divers. Depth – reported maximum depth of dive in meters. Timing of symptom onset: 1 – during dive; 2 – within minutes of surfacing; 3 – within hours of surfacing; 4 – within two days of surfacing. ACA – anterior cerebral artery; CCA – common carotid artery; Dash (–) – no information; ICA – internal carotid artery; L – left; NF – non-fatal; PICA – posterior-inferior cerebellar artery; PLVA – posterior left ventricular (coronary) artery, R – right; VA – vertebral artery; y – years

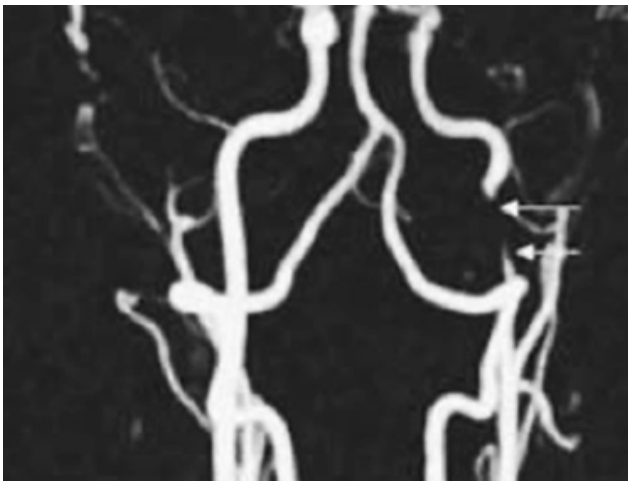
Case	Sex	Age (y)	Site	Dive description	Potentially relevant associations	Timing of symptom onset	Initial presentation of problem	Outcome
Aorta								
1[7,8]	M	20	Aorta	3 dives completed (36, 12, and 42 m) in one day; 15°C water	nil	2	Chest tightness noticed after second dive; chest pain when unsuiting after third dive; clinical check unremarkable; cardiac arrest during the night	Fatal
2[8]	M	63	Aorta	Resort dive (likely shallow and short)	–	2	Collapsed in dive boat post-dive	Fatal
3[9]	M	54	Aorta	3 min initial descent/surface	Ischaemic heart disease	1	Diver surfaces shortly after brief initial descent, vomiting with rapid loss of consciousness	Fatal
4[10]	M	60	Aorta	Developed chest pain ascending from 27 m dive	Hypertension, Dyslipidemia	1	Diver complains of chest pain starting during ascent and persisting post-dive; helicopter transport to hospital; patient survived emergency surgery	NF
Cervical or intracranial								
5[11]	M	52	ICA (R&L)	30 m for 25 min (mostly between 18–24 m); 3rd day of 1 dive/day pattern	nil	2	Headache "*within minutes of surfacing*" and dysphasia the next morning	NF
6[12]	F	35	VA (R&L), ICA (R.?L)	First lifetime dive	Osteogenesis imperfecta	2	Developed neck pain after first dive; evolving 2 days later	NF
7[13]	M	18	VA (R)	–	nil	4	R occipital headache, neck pain, and visual disturbance developed 2 days post-dive	NF
8[14]	F	38	ICA (R)	37 m max with "*several hours of diving*"	nil	1	Loss of consciousness at 2-3 msw at the end of unplanned rapid ascent; headache, L weakness, R anterolateral cervical neck discomfort, and mild aphasia 4 h later (during hospital assessment)	NF
9[15]	M	48	ICA (L)	8 m for 1.5 h	nil	2	Dizziness and stiff neck for 10 min immediately post-dive, neck pain and dysgeusia 2 days post-dive	NF
10[16]	M	51	ICA (L)	20 m (15 min near max) for 45 min total dive time; uneventful	nil	3	Developed slurred speech and difficulty finding words 3 hours post-dive (lasting 15 min); difficulties in swallowing and expressing words 2 days later; miosis and L ptosis	NF
11[17]	M	32	PICA (L)	–	nil	3	Persistent drowsiness experienced post-dive (reported 3 days later)	NF
12[18]	F	37	ICA (L)	Uneventful holiday dive; 22°C water	Smoking, multiple sclerosis	2	L facial paraesthia, L cervical pain, and L ear tinnitus starting within minutes post-dive; L nightly tension-neck pain noted 3 days later	NF
13[19]	M	52	ICA (L)	"*Scuba diving in cold water*"	Treated hypertension	4	Considerable and persistent neck pain radiating to left retroocular region starting the day after diving; L side facial edema, heaache, ptosis, miosis and neck pain worsened over days	NF
14[20]	M	27	VA (R)	"*Cold water*"	nil	3	Vertigo and gait imbalance developed 2 hours post-dive	NF
15[21]	M	51	ACA (L)	20 m for "*1 h*"	nil	1	Sudden R hemiparesis developed during dive (diver assisted to surface by instructor)	NF
16[22]	M	46	CCA (R&L)	–	–	–	Headache	NF
17[23]	M	32	ICA (L)	–	Smoking	2	L cervical pain and feeling of pressure in L ear starting within minutes post-dive; ipsilateral miosis and ptosis evident upon exam	NF
18[24]	F	60	ICA (L)	12 m max; uneventful	nil	2	Epistaxis and L frontal headache extending to L ear upon surfacing; L partial ptosis developed the next day; persistent ptosis and headache for 3 weeks	NF
Coronary artery								
19[25]	F	65	PLVA	30 m for 30 min (aborted from max depth); 9th dive in 5 days	nil	1	Sudden oppressive chest pain developed at 8 m during initial descent; cardiac signs and symptoms seen over following week	NF

The risk of dissection rises with increasing arterial pressure, such as in the case of individuals with untreated or poorly managed hypertension. Immersion and cooling stress increase arterial pressure through centralisation of blood volume. The increased partial pressure of oxygen produces an additional vasoconstrictive drive with concomitant increase in arterial pressure. An increase in arterial pressure can also be seen with constrictive suits or equipment, and also with breathing circuit resistance, more so as depth and respired gas density increases. It is unknown if the dive response may play a role in further augmenting arterial pressure in a spontaneously respiring diver, but it is at least theoretically possible. The reported cases of dissection in scuba divers all occurred in vessels where the dive response has been shown in animal studies to produce increased pulse pressure.[31] There are no reports of dissection involving peripheral or splanchnic arteries where the dive response is associated with vasoconstriction, but not with increased pulse pressure.

The cervical artery dissections identified in this review occurred at anatomical sites within those arteries that are distinct from the locations typically associated with neck manipulations or trauma. This finding suggests the involvement of different factors in their pathogenesis. In an earlier review of 24 patients with carotid artery dissection, none reportedly associated with scuba, one case involved the common carotid and the remainder the proximal internal carotid, the most mobile part of that vessel.[35] None involved the subcranial portion of the vessel, the region where the scuba-associated dissections occurred. The suggestion that neck hyperextension or other positioning or movements with diving play a key role in precipitating dissection in divers seems unlikely, but it is possible that restrictive equipment could create an additional burden of strain.

Figure 2

Magnetic resonance angiography from case 11; arrows show the dissection site in a sub cranial location where there is a 2 cm long with near total occlusion of the left internal carotid artery. From Prof M. Brodmann with kind permission



Cold stress has been demonstrated to induce the dive response[36] and/or to increase arterial pressures.[37] Exposure to cold water leads directly to increased parasympathetic stimulation and peripheral vasoconstriction, regulating body temperature homeostasis by increasing venous return to the heart. While the mask, continuous breathing, and thermal protection worn by divers may attenuate or abolish the dive response, activation is possible. Cold stress typically increases as a function of depth as neoprene insulation is compressed to become less effective and as divers cross thermoclines into colder water. It is possible that a quick chilling could initiate some degree of dive response.

Cervical artery dissections were generally seen to occur proximal to the entry points of the vessels into narrow bony spaces, these being the carotid canal for the internal carotid artery and the transverse foramina of the cervical vertebrae for the vertebral arteries. An enhanced pulse wave would be restricted at these points resulting in increased intraluminal arterial pressure. Instead of affecting the entire vessel, it is the specific region of arterial distension caused by the pulse wave that undergoes further stretching in accordance with the law of Laplace.[38] This increased distension could lead to elevated vessel wall shear stress with the potential for intimal tearing.

Pinnipeds (seals, sea lions and walruses) possess an elastic and bulbous ascending aorta,[39] an adaption that serves to reduce pulse pressure. It may be the lack of such an anatomical modification that promotes arterial dissection in human diving.

The dive response may also be activated in surface swimmers where there is total facial water exposure, yet reports of dissecting aneurysms in this group are relatively rare.[21] Other factors, such as muscular exertion, necessitating increased blood flow to the limbs, and regular breathing may serve to wholly or partially overcome the diver response. Surface swimmers also do not experience the strain of breathing circuit resistance and the depth-related increased gas density that divers must manage.

Three of the four reported cases of aortic dissection occurred in Australian waters, a region where only a small proportion of world diving occurs. One case occurred in a sinkhole diver in cold water, but the other two were in northern Australian waters that are relatively warm. It is unclear if water temperature may have played a contributing factor given the potential confounding of protective garments. Australia is noted for a robust system of dive fatality and dive incident investigation and reporting; it is possible that further cases have occurred elsewhere but remain unrecorded.

The only non-fatal case of aortic dissection was associated with quick recognition of a serious problem and helicopter evacuation to a medical centre, followed by transfer to

another hospital where emergency surgery was conducted. None of the cases of aortic dissection include detailed timelines to determine if the speed of transportation played a role in patient survival.

Coronary artery dissection is a rare cause of heart attacks and sudden cardiac death. It may be difficult to diagnose both clinically and radiologically. Even at post-mortem examination dissection may be confused with haemorrhage into an atheromatous plaque. The one case report of dissecting coronary artery dissection in a scuba diver could be an solitary event coincident with diving but it too may represent a failure of reporting or the failure of diagnosis of similar cardiac events.

IMPLICATIONS

Dissecting aneurysms should be considered among other conditions potentially affecting the collapsed diver, particularly when there has been no untoward event during the dive and in cases where there has been no response to recompression. Cardiopulmonary resuscitation will generally not be beneficial to a patient with aortic or arterial dissection.

Dissecting aneurysms of cervical vessels may present with neck or head pain, neurological disturbance, and Horner's syndrome. Horner's syndrome was observed in six of the eight patients with dissections confined to the internal carotid arteries. Horner's syndrome consists of a constellation of signs, including miosis (contraction of the pupil), ptosis (drooping of the upper eyelid), and anhidrosis (absent or reduced sweating on the affected side of the face). The syndrome is due to interruption of the sympathetic nerve supply to the eye and face.[40] This sympathetic supply reaches these areas through a network closely applied to the external layer (adventitia) of the carotid artery. Physical distortion of this network from arterial distension can result in the interruption of sympathetic transmission. Miosis is the most readily clinically recognised Horner's syndrome feature. Neck pain or tenderness, unequal pulses and disparity of pupil size should be looked for as signs of arterial dissection. Even with symptoms such as headache and confusion clinical signs may be absent and angiography necessary to establish a diagnosis.

With every dive incident, detailed dive history and profile should be recorded, together with dive conditions, including the water temperature, all equipment and thermal protection worn, the state of equipment and garments at the point of compromise, and a comprehensive timeline of all events. Prior medical disorders should be known before scuba diving. In fatal cases a meticulous autopsy is required, and an accurate cause of death established.

We speculate on the potential contribution of the dive response but cannot offer any estimate on the possible magnitude of the effect. Dive response effects are most commonly associated with breath-hold, which is not a normal situation for compressed gas divers. Further work is needed to determine whether any contribution goes beyond the theoretical to be practically important.

LIMITATIONS

The number of reported vascular dissection cases is small and inconsistently reported. There may be considerable under-reporting of scuba associated dissecting aneurysms both due to failure of diagnosis and failure of recording. Dissection of vertebral arteries may not be detected due to limited autopsy technique and aneurysm of a coronary artery may be misdiagnosed as haemorrhage into an atheromatous plaque. Single case reports are also not encouraged by many journals.

The available reports are inconsistent in descriptions of dive profiles (e.g., depth, duration, decompression obligations), water conditions (e.g., temperature, thermoclines, sea state, and entry/exit requirements), equipment worn by divers (e.g., scuba device, total weight, thermal protection), and event timelines through to completion. Incomplete and inconsistent autopsy records are also a problem. In fatal cases the vertebral arteries may not have been examined and the coronary arteries not specifically studied with serial transverse sectioning.

The impact of the diving activity in the two cases in which symptoms reportedly developed either on the next day or two days after diving (cases 7 and 4, respectively) is difficult to assess. It is possible that these were co-incidental and not causal events. Indeed, even in cases more proximally associated with diving a causal relationship cannot be confidently assumed.

Conclusions

Dissecting aneurysms of aorta, cervical, cranial and perhaps coronary arteries should be considered as a potential complication of scuba diving. The development of aortic and arterial aneurysms associated with scuba diving is likely multifactorial in pathogenesis. Detailed reporting is important in the evaluation of cases. The potential role of the diving response as a contributing factor in the pathogenesis of dissections requires further evaluation.

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