not be passed off as merely "part of diving''.

The instructions to the diabetic diver and the diving officer are at pains to emphasise that the symptoms of low blood sugar may mimic those of neurological decompression sickness or a gas embolism and vice-versa, e.g. confusion, unconsciousness, fits. In this situation, first aid therapy must be given to the diabetic casualty as though both conditions were present i.e. 100% oxygen and treatment for low blood sugar. In the event of there being an incident in the water or on the boat, the diabetic diver should be brought to the boat or shore as soon as possible. The blood glucose should be measured using the equipment in the diabetic emergency kit if this can be swiftly performed. Oral glucose should be administered to the subject with low blood sugar if conscious; otherwise, an intramuscular injection of glucagon (1 mg) should be given. Medical attention and recompression facilities should be sought as soon as possible.

Experience to date

The BS-AC and SAA have admitted diabetics who fulfil the medical criteria set out above to dive since November 1991. During this time, more than fifty diabetics have registered with the BS-AC. Their ages range from 17 to 46 and are of both sexes in roughly equal numbers. Both insulin-dependent and non-insulin dependent diabetics are registered. Some of the diabetics who contact us wishing to dive state that they will be diving to a depth of no more than 15 metres and will be diving in warm tropical waters but the majority are diving in British waters. Currently, there are three National Instructors (the highest teaching grade) within the BS-AC who are registered diabetic. Thus far (October 1993) there have been no reported incidents involving diabetics or their diving buddies. We hope that the standards set out above are such that this will remain the situation, but the situation is kept under constant review.

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SINUS BAROTRAUMA

A BIGGER PICTURE

Carl Edmonds

Abstract

The major English language publications on sinus barotrauma are reviewed, and a new clinical series of 50 cases is compared with a previous survey. The various symptomatologies, complications, investigations and treatments are discussed in relation to these series.

Background

Fifty years ago sinus barotrauma from aviation exposure was well described by Campbell [1,2]. The injury was due to the changes in volume of the gas within the paranasal sinuses during ascent or descent, when those changes could not be compensated by the passage of air between the sinus and the nasopharynx. It was the clinical manifestation of Boyle's Law, as it affected the sinuses.

The pathological changes found within the sinuses due to these aviation exposures included: Mucosal detachment; submucosal haematoma; blood clots in membranous sacs; small haemorrhages within the mucosa; and swelling of the mucous membrane (especially in the absence of previous sinusitis).

Weissman et al.[3] described a series of 15 cases of frontal sinus barotraumain aviators. Mostwere Grade III. They used a grading systems as follows:-

Grade I A transient discomfort which cleared promptly and had only a slight oedema but no X-ray changes.

Grade II Characterised by pain over the affected sinus for up to 24 hours. There was thickening of the mucosa seen on X-ray. If such a sinus was opened, small amounts of blood-tinged fluid were found. Serosanguinous fluid sometimes drained from the sinus, with or without the use of decongestants.

Grade III A severe pain or a "bee sting" or "being shot" sensation. If the pain was not quickly relieved by the Valsalva manoeuvre, the pilot had to ascent rapidly to relieve symptoms.

Usually Grades I and II did not seek medical aid, and were usually treated simply. Grade III cases resulted in oedema and congestion of the sinus mucosa with submucosal haemorrhages. As the sinus mucous membrane was pulled away from the periosteum by the negative intrasinus pressure, a haematoma formed. Sinus X-rays showed an air fluid level or a polypoidal mass. Incising this mass brought forth a spurt of old blood, with clots.

Reviews of the diving related sinus barotraumas were not easy to find. Flottes[4] in 1965 described sinus barotrauma in divers. Other authors, whom I can not translate, included Ameli and Caligaris,[5] Sliskovic,[6] Conde,[7] Czech and Chojnacki.[8]

Sinus barotrauma has been previously described in various texts on diving medicine[9] and aviation medicine,[10] but initially without any specific clinical series being documented. A reasonably large clinical series of divers with sinus barotrauma was first described in Australia by Fagan, McKenzie and Edmonds in 1976[11] and quoted widely thereafter.

The initial Australian series

The Australian series included 50 consecutive cases of sinus barotrauma as they were observed in a Navy setting, where all such cases were referred for medical opinion, irrespective of severity. In the majority of these cases the divers were inexperienced, undergoing their first open water diver training course. This series was valuable in that it quantified the imputed symptomatology and the extent of specific provocation factors. Unfortunately it did have the disadvantage of including many cases that might otherwise have not attended for treatment.

In that series 68 % of the presenting symptoms developed during or immediately upon descent, and in 32% during or immediately upon ascent. Pain was the predominant symptom present, in all the cases on descent, and in 75% of the cases on ascent. It was referred to the frontal area in 68%, the ethmoid in 16% and the maxillary in 6%. In one case it was referred to the upper dental area.

Epistaxis was the second most common symptom, occurring in 58% of cases. It was rarely more than an incidental observation, perhaps of concern to the diver but not usually of great severity. It was the sole symptom in 25% of the cases of ascent barotrauma.

Even though these were inexperienced divers, in 32 % there was a history of previous sinus barotrauma, and this had been produced by scuba diving, aviation exposure or free diving. Half had a history of recent upper respiratory tract inflammation and others gave a history of intermittent or long-term symptoms referable to the upper respiratory tract e.g. nasal and sinus disorders, recurrent infections, hay fever, etc.

In 48% of cases, otoscopy showed evidence of middle ear barotrauma on the tympanic membrane.

Radiologically the affected sinuses did not replicate the frequency of the clinical manifestations. The maxillary sinus had either mucosal thickening or fluid level in 74% of the cases, the frontal in 24% and the ethmoid in 15%. This is in reverse order to the clinical manifestations. A fluid level was present in 12% of the maxillary sinuses.

Most of these divers required no treatment, or responded to short term use of nasal decongestants. Antibiotics were prescribed if there was a pre-existing or subsequent sinusitis. Neither sinus exploration nor surgery was required in any case. This series has unfortunately, and inappropriately, been used to imply that such intervention is never applicable in the treatment of sinus barotrauma. The fact that it was a series covering the range of cases diagnosed as sinus barotrauma, meant that most were of minor severity.

Despite the fact that this survey was done almost two decades ago, and before computer imaging techniques became commonplace, there has been little in the way of clinical series documented since.

This is unexpected, because sinus barotrauma and its complications remain a common medical hazard of diving. The importance has been stressed by many workers.[12-15]

There have been many single case reports in the literature, mostly reiterating the observations of previous authors or those of the diving and aviation medical medical text books.

The second Australian series

The initial Australian series, by design, comprised relatively minor cases of sinus barotrauma, not necessarily typical of those that present for medical consultation on their own volition. The diagnosis was made on the symptomatology alone, based on symptoms produced with pressure changes, but verified by radiology. The Australian

group has, by default, been used as being typical of all sinus barotrauma cases, even those that present with recurrent or delayed symptoms, or complications, in emergency wards or ENT consulting rooms. That extrapolation is not necessarily valid.

To offset that presentation, a new series of 50 cases of sinus barotrauma, more accurately represents the type of case that is likely to confront the ENT or Diving Medical consultant, has been collected by the Diving Medical Centre. It consists of the last 50 cases that have presented with the following criteria:-

1 They were seen within 1 month of the most recent incident.
2 Incontrovertible or overwhelming evidence was present to verify the diagnosis of sinus barotrauma or disease. This usually included a CT scan of the sinuses, but with sinus endoscopy and/or surgery in some cases.
3 The divers were referred by primary care physicians and came specifically for treatment at their own instigation.

This second series, because the symptomatology was far more dramatic and often repetitive, dealt with more serious cases. The cases were self-selecting because the divers with repetitive or more significant problems were more likely to present for medical treatment. The investigations frequently involved CT scans of the sinuses, sinus endoscopy and occasionally MRI.

Despite the difference in selection procedures, the two series were very similar in their major symptomatology and provoking aetiologies. The pain, provoked by descent and/or ascent, and the extrusion of fluid on ascent was similar to the first series. This fluid takes the form of a thick mucus secretion (especially if there had been chronic sinusitis in the past) or blood, from the nose into the face mask or down the back of the nasopharynx. This is due to the expansion of gas with ascent, producing a "washout" of the damaged sinus.

The main differences from the first series

Diving Experience

Unlike the initial series, 88% of the divers in the second series were experienced (over 50 dives). The distribution was strongly skewed to the extremely experienced, with 70% of the divers having over 5 years diving, and many being dive masters, dive instructors or professional divers.

Because of the extreme amount of diving exposure in this group, it is presumed that the sinus ostia or ducts become scarred and narrowed from the repeated insults they sustain.

Symptoms

In some cases the diagnosis was not evident when the client was initially seen by the primary care physician. This was especially so in 12% of the cases, where the presenting headache developed and progressed whilst at depth. With questioning it could usually be elicited that the headache would be made worse with subsequent ascents or descents, but the initial development of the headache during a time when there was no substantial change in depth, did apparently cause some confusion in the emergency physician's assessment.

A small degree of negative pressure[16] is sustainable within the sinuses, without symptoms. Exceeding this may be sufficient to cause a gradual effusion to develop, and the full or heavy sensation within the sinus may take some time to develop. Extrapolation from aviation physiology would suggest that diving related barotrauma could occur with a reduction in sinus air volume of 5-10%, i.e. at a depth of 0.5 to 1 m below the surface.

We now must accept that headache developing during the dive, with the diver neither ascending nor descending, should not exclude the diagnosis of sinus barotrauma. When this develops at considerable depth, the sedative effects of narcosis may distort the clinical features. Diagnostic complications may arise. Also, small changes of depth are not particularly noticeable when swimming in mid-water, so producing a misleading history.

Because of the method of selection of the initial 50 cases, such symptomatology was not considered. On the contrary, patients were selected who developed their symptoms only during ascent or descent. In retrospect, this was clearly a design fault in the survey.

"Pop"

In 8% of the cases there was a very clear-cut and dramatic sensation of a bursting or popping, during depth changes. Half were on descent and half on ascent.

It has been described in aviation medicine as; the "popping of a champagne cork", a "gun shot", "like a bee sting over the eye", "like being struck on the head with a club or bat". It is presumed, both from the observations of Campbell[1,2] and Mann and Beck[16], and from this series, that the sensation is due to a haemorrhage stripping up the mucosa of the sinus, produced by the negative intra-sinus pressure with descent encouraging hyperaemia and stretching of the mucosa.

A similar sudden sensation can also occur from the rupture of an air sac or release of pressure from a distended sinus during ascent. This may be followed by a "hissing" sensation of air movement, which may then relieve discomfort and pain. One of the cases involved the ethmoidal

area, and there was a subsequent small oval shaped haematoma noted over the ethmoid region, within hours (Figure 1).

Figure 1. Oval haematoma at root of nose

Maxillary Nerve Involvement

In 4% of the cases the pain was referred to the upper teeth, on the same side as the maxillary sinus affected. This is presumably an involvement of the posterior superior alveolar nerve. In another 4% there was involvement of the infra-orbital nerve, with numbness over the skin of cheek on the same side.

Two separate branches of the maxillary division of trigeminal nerve can be affected[17,18] as they traverse the maxillary sinus. The infra orbital as it runs along the wall of the maxillary sinus and the posterior superior alveolar nerve as it runs along the lateral or inferior wall of the maxillary sinus. The former produces a numbness or paraesthesia over the cheek. The latter a numbness over the upper teeth, gums and mucosa on the same side. In some cases pain and hypersensitivity are noted. Problems with neurapraxes are more common with ascent than descent, suggesting that the pathology is pressure induced impaired circulation is more frequently than congestion or haemorrhage of the nerve.

There is a possibility of involvement of any division of the trigeminal nerve, including its maxillary division, with involvement of the sphenoidal sinus.[19]

Atypical Symptoms

In a small number of the cases (8%) some additional symptoms did not appear to be easily explicable on the basis of local sinus pathology. These included; nausea or vomiting, a sensation of impending syncope, and disorientation at the time of injury. These all occurred in the more dramatic cases of the sinus barotrauma.

Differential Diagnosis

In 6% of the cases an initial diagnosis of decompression sickness was made, with the case subsequently demonstrated to be sinus barotrauma, often with complicating sinus infection. At the time of presentation, which could be some hours after the dive, the clinical pattern was confused with cerebral decompression sickness, and treated as such. These were understandable mistakes and there should be no hesitation in repeating them, if there is any doubt regarding the diagnosis.

It would be preferable to miss and mistreat a case of sinus barotrauma than miss and mistreat a case of cerebral decompression sickness.

The only other case of incorrect diagnosis was one subsequently attributed to a dental aetiology (barotrauma associated with pneumatisation around a carious tooth) and this case was therefore not included in the series.

Inappropriate Diving Techniques

This was not fully appreciated in the initial series, and so not documented. In 10% of the second series, repetitive incidents of sinus barotrauma appeared to be provoked by inappropriate diving and equalisation techniques. Often there was a head first descent, and/or swallowing as a method of middle ear equalisation. The substitution of a feet first descent (preferably down a shot line), together with frequent positive-pressure middle ear equalisation manoeuvres, appeared to rectify the situation. These are fully described in medical texts[20] used by divers.

A similar problem developed if descents were slow, due to the discomfort noted in the sinus. The blood or effusion gradually accumulating in the sinus equalises the pressure and reduces the degree of pain and discomfort. This might be appropriate for an emergency dive, but is not reasonable if damage is to be avoided. On the contrary, divers inappropriately used the development of the pathology (blood or effusion, mucosal congestion, etc.) as a "treatment" to replace a contracting air space in the sinus during descent, and allow the dive to continue.

Divers were advised of the correct methods of descent and to use positive pressure middle ear equalisation (e.g. a Valsalva manoeuvre). This can aerate the sinuses before major pathology and haemorrhaging develops.

Sphenoidal Sinus

Lew and his colleagues[19] not only referred to the symptomatology of sphenoidal sinusitis, but also referred to its association with "deep sea diving".

Sphenoidal sinus involvement occurred in 6% of the current series. It is important because of the tendency of clinicians to not recognise it and to not appreciate its potentially serious complications.

Another case, which is well known to the author but was not part of this series, sustained sphenoidal sinus barotrauma and caused considerable concern because of the proximity of the space occupying lesion to other important structures near sinus, and the possibility of the lesion being more ominous than a mucosal cyst or haematoma. Although operative intervention was contemplated in that case, the lesion cleared up within a few weeks, following abstinence from diving.

Spenoidal sinusitis is not easy to demonstrate radiologically, but is often obvious with tomography or CT scans.

Complications

Acute Sinusitis

Acute sinusitis developed some hours after the dive, and extending into subsequent days, in 28% of the cases, usually with an exacerbation of pain over the affected area,

The cases of sinus barotrauma that subsequently developed a sinus infection, possibly did so because of the haemorrhage and effusion in the sinus. This becomes a culture media for organisms introduced by the flow of air into the sinus during descent.

For this reason we now vigorously treat with antibiotics any symptoms following sinus barotrauma, which commence hours after the dive or continuing into the following day.

Chronic Sinusitis

The criteria for this diagnosis included a continuation of sinus symptomatology longer than one month. In 18% of the cases the initial barotrauma episode and acute sinusitis continued into chronic sinusitis.

In another 14% the chronic sinusitis was initially present, with recurrent barotraumas developing.

Complications not evident in the second series

Pneumocephalus

None of the cases in this series sustained a pneumocephalus, either radiologically or by CT scan. The presence of pneumocephalus, in association with sinus injury in general medicine, has been well recorded by Markham[21] and it is one of the dangers associated with sinus barotrauma.[9] It has been well demonstrated by Goldmann.[22]

Surgical Emphysema

This did not occur in any of the cases in this series. Nevertheless, it has been demonstrated elsewhere.[9] The tracking of air into the tissues can present as orbital surgical emphysema (usually from the ethmoidal sinus though a fracture of the eggshell-thin lamina papyracea). In other instances the air has passed from other sinuses, such as the mastoid.

Treatments given

These can be divided into groups:-

1 The first group were those that cleared up spontaneously, and were advised to not dive until this had happened.

2 Those that were using inappropriate diving techniques. They usually responded to appropriate regimes of:
feet first descent
positive pressure manoeuvres to autoinflate both middle ears and sinuses, on the surface, immediately before descent, and then at regular intervals of 0.5-1 m during descent
avoiding diving with respiratory tract inflammation.

3 Those who responded to medical treatment of the nasal pathologies. This included the topical use of steroid nasal sprays, cromoglycate, topical or generalised decongestants, avoidance of nasal irritants and allergens, and cessation of smoking.

4 Infective sinusitis cases required treatment of the infections, usually by decongestants and antibiotics.

5 The intractable group required sinus exploration, usually with endoscopy and reconstruction, or nasal surgery. In some cases surgery was required to produce patency of the ostia and to remove polyps or redundant mucosa that caused obstruction to the ostia. Othertimes it was needed to improve nasal air flow.

6 The sixth group continued to have difficulties and usually gave up diving.

All were strongly advised to not dive during times of upper respiratory tract inflammation (infections, allergic or vasomotor rhinitis, etc.). As in the original series, over 50% of the divers had a history of diving with such conditions at the time of the barotrauma.

Some patients moved between treatments, as various measures failed to completely resolve or prevent problems.

It was difficult (and would be misleading) to allocate percentage incidence of success to each of these treatments. Many were involved in more than one type. Also, many of the patients could not be traced 1 - 2 years later. Divers are an itinerant group.

Also, other environments, countries and consultant practices would result in different clientele and treatment indications. Our general impression is that approximately equal numbers fell into each “treatment” group.

Investigations and pathology

The appearances were often described by radiologists as suggestive of haematomas, mucous cysts, mucocoels, polyps or polypoid masses, opacification and, most commonly, a thickening of the mucosa. Our series was no different in the various radiological descriptions. However, the CT scans showed more identifiable and definitive pathology. Magnetic resonance[23] using T1 and T2 weighted imaging would be expected to be more useful in differentiating blood from mucosal thickening.

Most of the effects of sinus barotrauma rapidly regress if diving is suspended and the underlying or consequential inflammatory pathology of the sinus treated.

Campbell [1] stated that infection occurs only rarely, and his series may be comparable, in terms of selection, with our initial survey.[11] If, however, one considers our second survey, with its more serious cases, then infective complications are more frequent.

The use of CT scans of the sinuses made diagnosis and treatment more definitive in most of these cases. Sinus endoscopy, sinus surgery and/or nasal surgery was needed in 12% of the cases, often with excellent results. Bolger, Parsons and Matson[24] in 1990, have questioned the value of surgery in aviators with sinus barotrauma. Their guarded enthusiasm for functional endoscopic sinus surgery is tempered by the possible complications of this procedure.

Nevertheless, endoscopic sinus surgery is advancing rapidly, and may offer value to the more serious and chronic cases. With current endoscopic surgical procedures, the maxillary, ethmoid and sphenoid sinuses can be treated so as to widen the sinus ostia, preventing sinus barotrauma. The frontal sinus is less amenable, but may be assisted in some cases

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FACIAL PARALYSIS AFTER SCUBA DIVING A CASE HISTORY

Noel Roydhouse

Introduction.

The occurrence of facial paralysis after scuba diving has been reported by Molvær[1] and Becker.[2] Their cases suffered transient ipsilateral facial paralysis associated with middle ear overpressure during ascent. When it occurs it produces a marked and unmistakable deformity which prompts the seeking of medical advice even if it is transient. Bell's palsy is the commonest clinical variety to be seen. Other causes are acute otitis media, chronic otitis media with or without cholesteatoma or mastoiditis, skull fracture, tumours, and iatrogenic. Viral peripheral neuritis and vascular spasm of perineural vessels are also rare possibilities. In the course of all middle ear and mastoid operations the facial nerve is at risk from trauma. It emerges from the lower border of the pons passing laterally down the internal auditory meatus, then between the cochlea and the vestibule until it reaches the medial wall of the middle ear. It bends sharply backwards to travel in the horizontal part of the facial canal which is a rounded eminence above the fossa ovalis. At the posterior margin of the fossa ovalis the nerve arches downward behind the middle ear between it and the mastoid region.

In about 40% of cases the bony facial canal during passage through the middle ear has dehiscences. This lack of bone covering the facial nerve varies from complete to small 2-3 mm gaps. At the time of operations such as total stapedectomy, a procedure rarely carried out now, an instrument may press against such an exposed nerve. However rarely is there any problem but facial paralysis for up to 6 months has been known to occur. It takes 6 months for re-growth of the fibrils from the middle ear to the muscle motor end plates. However without a break in the neural tubes the paralysis may last 5 minutes to a day or two.

Case History.

This 16 year old male patient was doing his first diving session in a PADI scuba diving course. He was diving in a swimming pool with a depth of 3 m and had carried out about 6 descents and ascents over a period of 45 minutes. After descent he would swim around the bottom of the pool before ascending. He stated that he was able to equalise his middle ear pressure with the ambient pressure without difficulty. His method was to hold his nose and blow hard through his nose and his ears would pop. On descent he inflated his ears in response to the feeling of pressure in his ears. There was some soreness on descent but he developed more soreness in his ears on ascent. Both ears were affected equally.

He first noticed his facial symptoms when he had finished underwater and had come out of the water. His left lower lip felt numb and 5 minutes after this he noticed the numbness had spread to the side of his face and that his left lower lip was hanging down and he could not whistle. By this time he had removed his scuba gear and was surface swimming. He found that he could not close his left eye fully and that water was getting into it and it was sore. This failure to close the eye was noticed by other people. This was about five minutes after the initial numbness that he felt in his left lower lip. This condition lasted for 15 minutes when there was a fairly sudden return to normality of his face.

About one and a half hours after getting out of the water he noticed some deafness in his left ear as he used the telephone. This ear felt as though there was water in it and felt blocked. The deafness was definite. The morning after the incident of the facial asymmetry he reported to his General Practitioner who syringed out a lump of wax from the left ear with the relief of the left deafness.

In the past he had had ventilating tubes inserted into his eardrums along with the removal of his tonsils and adenoids at the age of five years. His mother never saw the ventilating tubes come out. This is not unusual. He stated that his ears as a rule did not get itchy nor did they block but he did rub them at times. He did not use cotton buds in his ears. He denied grinding his teeth but admitted that he