also required the highest standard of air pump and Siebe's pump was superior, hence his diving rig became the preferred one for the Royal Navy. From here the saga of the origins of the closed diving dress started. Siebe's son, William Henry Siebe, and son-in-law, William Augustus Gorman, and later Sir Robert Davis, a chairman of Siebe Gorman Company, were probably responsible for the propagation of the story that A Siebe was the first to design the "standard" diving dress. For greater detail on the origin of the "standard" diving dress the reader is referred to Bevan's excellent historical book *The Infernal Diver*,[16] from which these details have been taken.

Augustus Siebe died on the 15 April, 1872 from "chronic bronchitis". His son, Henry, and son-in-law, William A Gorman, had taken managerial control of Siebe's business in 1868 which became known, in 1870, as the Siebe Gorman Company.[12,16]

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BILATERAL DEAFNESS ASSOCIATED WITH DIVING

Alfred Buchner and Matthias Heppe

Key Words
Case report, ENT.

Case report

A 61 year old male German diver went to Egypt and the Red Sea for a live-aboard diving holiday. He had 30 years experience and more than 500 dives in the former German Democratic Republic. Before he went he had medical examinations for fitness to dive and was considered "fit to dive without any restrictions".

During the first day on board he performed two relaxing dives without any strenuous events. The profiles were 10 to 15 m (30 to 45 ft) for about 30 minutes. He did not use a diving computer and there were no records available.

On day 2 he performed three dives. The first dive was 30 minutes at 14 m (42 ft). After a surface interval of three hours the second dive was 30 minutes at 20 m (60 ft). The surface interval was 3 hours 30 minutes before the third dive, which was to 35 m (105 ft) for 20 minutes. Dives 1 and 2 were in a group of divers with a divemaster while dive 3 was a camera dive with a buddy. He did regular safety stops on all dives. The last dive was "not very much controlled" according to the diver's report. He had no equalisation problems at all and did not do any Valsalva manoeuvres during the dives.

After the third dive he complained of "stomach problems", nausea, dizziness, sweating and pallor. He vomited once but there was no vertigo. He felt tired and went to bed early. The divemaster reported that this dive party consumed a lot of alcohol. The patient stated that he drank much "as normal". Waking up the next morning he realised that he was completely deaf in both ears. There were no symptoms or signs of decompression illness.

A doctor from a mobile service was called on board. He stated that there were no haemorrhagic portions of the tympanic membranes nor any perforation. Nevertheless he diagnosed bilateral barotrauma to the middle ears. One and a half litres of saline, and steroids, were given intravenously. Oxygen was administered by mask through a Wenoll system.[1] There was no change in the deafness after this treatment.

The patient was then referred to a hyperbaric oxygen chamber in Hurghada (Egypt). According to the medical report he was still on oxygen 48 hours after the last dive. There were no other complaints apart from the hearing loss. He was treated with a US Navy (USN) Table 6. He was still deaf after 4 hours and 45 minutes of treatment. The next day a USN Table 5 was performed (2 hours and 15 minutes). Again there was no change of the existing deafness.

Six days after the last dive he eventually arrived at the EURO-MED-CLINIC Hyperbaric Oxygen (HBO) Centre, in Fürth, Germany.

He had severe hypertension (210/140 mmHg), diabetes mellitus and obesity (107 kg, 182 cm). With oral hypoglycaemic agents and anti-hypertensive medication he returned to normal values. He was still deaf in both ears with no sign of barotrauma, no rupture of the foramen rotundum (round window) as diagnosed by the ENT Department. No vestibular lesion was found. Magnetic resonance imaging and CAT scanning displayed no tumours, no haemorrhage nor any trauma. The only finding was a slightly affected maxillary sinus on his left side. Neurological examination was found normal apart from the deafness and a late SEP (somatic evoked potential) of the posterior tibial nerve bilaterally.

We commenced treatment with intravenous hydroxy-ethyl-starch, 1,000 ml daily, and Pentoxifylline with a loading dose of 100 mg on day 1 and dose increments of 100 mg on day 2 and day 3. A daily dose of 300 mg was continued until day 9. No steroids were given because of the patient's coexisting diabetes mellitus. He had HBO treatment for 8 days in a multiplace chamber (HAUX Starmed). Each treatment consisted of three 30 minute periods on oxygen, separated by 10 minutes air breaks, at 2.4 bar absolute pressure. There was no change in his deafness.

After this we transferred him to a Center for cochlear implants for further treatment.

We searched Medline from 1992 to 3/1998, using the key words: "diving + deafness". We did not find a case of bilateral deafness reported as due to diving.

We believe that the dive profiles, with each dive deeper than the dive before, with the pre-existing, but formerly ignored, medical history of severe hypertension, mild diabetes and obesity led to a higher susceptibility to decompression illness. We think that this was bilateral decompression illness affecting the inner ears, probably with a rupture of endauricular membranes.

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