General history of vestibular disorders in diving

ROBERT S. KENNEDY

Behavioral Sciences Department, Naval Medical Research Institute, National Naval Medical Center
Bethesda, Maryland 20014

Kennedy, R. S. 1974. General history of vestibular disorders in diving. Undersea Biomed. Res. 1(1): 73-81.—The history of vestibular symptomatology in hyperbaria is reviewed. Most of this literature deals with vertigo as a symptom of decompression sickness in divers and caisson workers, although other symptoms (e.g. nystagmus, staggering), other influences (compression, mixed gases), and other environments (breath-hold dives, submarines) are included. Studies that report case histories, incidences, and other factors are summarized. After joint pain and its effects, vestibular embarrassment and related ear problems is the next largest symptom complex in decompression sickness.

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Many disorders in diving can be attributed to vestibular involvement. It is helpful to consider vestibular implications in three categories: direct, indirect, and uncertain. The involvement may be considered direct during pressure vertigo or when lesions are present (Heller et al. 1900). Indirect vestibular implications may be seen in the many perceptual problems of spatial orientation occasioned by unarticulated environments in general, and diving in water in particular (e.g. “empty field myopia” [Katz et al. 1964]; “break off” [Clark and Graybiel 1957], “fascination” [Vinacke 1946], and “perceptual narrowing” [Weltman et al. 1971]; “Which way is up?” versus “Where is my habitat?” [Radloff and Helmreich 1968]). Examples of disorders where the vestibular implication is uncertain include difficulties due to inversion of the stomach contents when swimming downward (Fields 1958), past-pointing changes during neutral buoyancy (Whiteside 1960), auditory threshold shifts when inverted (Corso 1972), sea sickness prior to a dive (Cave 1971), differential caloric irrigation due to a dislodged plug of cerumen (Fields 1958), etc. An attempt was made previously by this author (Kennedy 1972) to annotate all the published studies where there was a vestibular implication whether direct, indirect, or uncertain. The present paper deals mainly with literature that describes vestibular symptomatology that occurs following changes in pressure, and which is believed to have a direct relationship to stimulation of the vestibular apparatus or its pathways. For studies dealing with indirect and uncertain vestibular involvement see reference numbers 623-729 in the annotated bibliography (Kennedy 1972).

The literature is reviewed herein in order to: a. assess the incidence and severity of vestibular disorders in hyperbaric exposures; and b. summarize the significance of these findings for those involved in diving research.
HISTORICAL REVIEW

The number of studies that report vestibular symptoms in hyperbaria is presently increasing. About as many appeared before 1900 as between 1900 and 1940. The annotated bibliography (Kennedy 1972) shows that the number of studies approximately doubles each decade since 1900 and presently the number of articles is probably growing at an equivalent or faster rate than the scientific literature in general. The sourcebooks of Hoff (1948) and Greenbaum and Hoff (1954, 1966) and Shilling and Werts' bibliography (1971) show a slower growth rate for compressed gas studies in general. For example, studies that mention vestibular-type symptoms are infrequent between 1940-1955, a period within which the sourcebooks indicate that diving medicine investigations grew at the most rapid rate before or since (about 4,000 citations in the 6-year period, January 1946 to December 1951). It is convenient to consider the diving and caisson literature in four main time frames: prior to 1900, 1900 to 1940, 1940 to 1959, and 1959 to the present.

PRIOR TO 1900

American and British studies

The first author to write about vestibular problems in English was Smith (1873), the medical director for the Brooklyn Bridge. Van Rensselaer (1891) reviewed the literature up to his day and cited certain vestibular symptoms as prodromal signs (p. 414) of decompression sickness. He also reviewed the French literature. Curnow (1894) published the first complete, careful, descriptive report of a case of vertigo. Snell (1896) also provided an excellent account of “...cases of auditory vertigo...not previously...met with...by writers on the subject [p. 74].” Lester and Gomex (1898) reported labyrinthine problems and performed ENT exams before and during compression. These reports represent the most significant studies published in English prior to 1900.

German studies

Also at the turn of the century Alt, Heller, Mager, and von Schrotter (i.e. Alt [1896]; Alt et al. [1897]; Heller et al. [1895, 1900]; and von Schrotter [1904]) published their studies where lesions in the semicircular canals from gas emboli were described (Alt et al. 1897, p. 239) and case histories with vestibular symptoms were reported. Symptoms like dizziness, tinnitus, vomiting, “reeling like drunkards [p. 240],” and balance problems were also listed. In Hill’s (1912) later review of this work as many as 20% were considered to have had vestibular-type symptoms. Hoche (1897—Engl. transl. available) also mentioned spontaneous nystagnus (p. 465), dizziness, and other symptoms in connection with air pressure diseases of the central nervous system (CNS).

French studies

In 1878 Bert recorded “vertigo, dizziness, and vomiting [p. 363]” as symptoms of decompression sickness as well as “reflex movements [of the eyes] caused by the slightest stimulus [p. 381].” He also may have reported an instance of the oculogyral illusion (p. 385) and of nystagnus (p. 385).

1 These articles have been translated and copies are available from this author. To my knowledge, the books (Heller et al. 1900) have never been translated.
1900 TO 1940

In the next decades Keays (1909), reviewing almost 4,000 cases of compressed air illness, reported vertigo as the "... most characteristic symptom [p. 38]" in 5% of all cases and suggested labyrinthine hemorrhage as the cause. Keays did not indicate how frequently vertigo or other vestibular-type symptoms were present in the remaining cases. Bassoe (1913) reported that more than 50% of 161 men had "ear affections [p. 541]." Reporting 700 cases, Levy (1922) showed 6.5% with vertigo and 91.8% with pain as presenting symptoms, but no further breakdown was given of either vertigo as an accessory symptom or other vestibular-type symptoms.

When Vail (1929) summarized some of this earlier work, he expressed surprise that no Navy divers had been placed on sick lists "for damage to the ears considered due to diving activity [p. 114]." He then provided two case histories as examples.

In 1933 Lestienne reviewed the literature of labyrinthine accidents and provided case histories. From these data he hypothesized that labyrinthine problems due to compression tended to recover, but those resulting from decompression accidents affected the nerve and were more likely to be permanent.

1940 TO 1959

In a series of papers, Shilling (1938, 1941a, 1941b) reviewed some of the literature available in light of the U.S. Navy's experience in diving with compressed air. He (1941a) felt that "... Meniere's symptom complex is relatively common and vertigo, either alone or associated with other symptoms, is frequently noted [p. 237]." He (1941b) also claimed that "... joint pathology" and "otological effect(s)" are considered two special problems "... given more notice than before [p. 309]." In the early forties, several articles appeared that are of importance in understanding the role of vestibular symptoms of decompression sickness. These include Behnke's (1942) paper regarding bubble formation and fat; Almouir's (1942) general paper on otology in caisson workers (including Meniere's-type symptoms); Bert's (1878) book which was translated into English in 1943; and Shilling and Everley's work (1942). Shilling and Everley distinguished labyrinthine problems due to barotrauma from those due to bubble formation—they considered that the latter most resemble the Meniere's syndrome and may cause "aural lesions [p. 669]."

During this period about 70 additional pressure chamber studies (mostly low pressure) were published (Kennedy 1972) that reported problems related to the labyrinth. Most of these studies dealt with barotraumatic otitis media (Shilling et al. 1947). Some mention vestibular-type symptoms (vertigo, ataxia, nystagmus, dizziness, disequilibrium and visual illusions of motion, incoordination, nausea, vomiting, pallor, sweating, faintness). In one study the last five symptoms listed were considered circulatory problems (Gray et al. 1946, p. 339). Also during this period three unusual findings bear mention. One was that the symptoms of a migraine-like syndrome in decompression sickness were similar to those with vestibular involvement (Engel et al. 1944). Another was that dental problems (jaw malocclusion) were related to susceptibility to labyrinthine symptoms during pressure changes in several studies. And third, thickened eardrums, hearing loss, balance disturbance, dizziness, and spontaneous nystagmus were found during exposure in "Schnorkel" submarines (Uffenorde 1948).

During the fifties some French articles appeared with new data (Bertoine 1953; Chossegros et al. 1953) focused on vestibular symptoms as precipitators of longer-lasting damage. A
study by Kooperstein and Schuman (1957) suggested that an ability to equalize pressure, coupled with an eardrum inspection prior to exposure, contributed to one of the lowest incidences of general decompression sickness ever reported (.03%).

1959 – PRESENT

Modern studies of vestibular involvement in compressed gas exposures owe a debt mainly to Rozsahegyi (see Rozsahegyi and Soos 1956; Rozsahegyi 1959; Rozsahegyi and Gomori 1961; Rozsahegyi 1966; Rozsahegyi and Roth 1966a; Rozsahegyi and Roth 1966b). He called attention to a central pseudo-Meniere’s syndrome which occurs with significant frequency and is due to a lesion in the medullary vestibular nuclei. His reports have dealt mainly with caisson workers, people with long histories of exposure. The symptoms he reports resemble Meniere’s disease as well as syncope, parasympathetic disturbances, and migraine. In this regard a review study by Coles and Knight (1961) is important. These authors attempted to distinguish vertigo due to syncope versus vertigo due to vestibular involvement. They also report hypovestibular function (caloric) in older divers.

A long book (250 pp.) in Italian by Pagano (1959), *Otopathology and sinusopathy from barotrauma in caisson workers*, has not yet been absorbed in the diving literature. The book was referenced incorrectly in the diving medicine sourcebook (Greenbaum and Hoff 1966), never translated into English, and has never been observed by this author as a reference by others. (A translation of the Summary by this author indicates Pagano was well aware of vestibular problems in diving and his reference list is fairly complete.)

In addition to the studies cited above, those of Picard and Nouritt (1961) and Erde (1963) should be consulted. These authors agree in their concern for CNS problems of decompression sickness and in particular, vestibular problems. Lastly, recent studies by Rubenstein and Summit (1971) and by Edmonds (1971) have shown that the incidence of vestibular problems is greater in deep saturation diving.

GENERAL EPIDEMIOLOGY AND SYMPTOMATOLOGY

Approximately 300 studies dealing with direct vestibular involvement in compressed gas work have been found in the published literature (Kennedy 1972). These studies cover about 100 years of diving and caisson work. The reported incidence of vestibular symptoms in these studies is from zero (i.e., none reported) (Paton and Walder 1954; Bond 1966), to 28% (Heller et al. 1900), to more than 50% (87 out of 161 tunnel workers with ear problems, Basso 1913). In the latter study a further breakdown showed “33 complained of dizziness . . . 6 of vomiting . . . 6 has blind staggers, that is, labyrinthine vertigo, with nystagmus [p. 527].” Therefore, 28% probably involved the vestibular system itself (cf. Hill 1912; Hamilton et al. 1966). In modern saturation diving studies, as many as 50% or more of the subjects have been reported to have experienced vestibular-type symptoms (e.g. “dizziness” and “nausea” [Bennett and Towsie 1971, p. 1154] were complaints made by both subjects). In addition, related CNS symptoms (i.e. Type II [Griffiths 1969], as opposed to Type I [i.e. pain]) seem to occur with greater frequency in saturation diving unless special precautions are taken. Unfortunately, in saturation diving studies group sizes are typically small—generally 2 to 4 persons (Hamilton et al. 1966; Bühlmann et al. 1970; Sundmaker 1972).

However, in a report (Bühlmann and Waldvogel 1967) of 83 saturation diving accidents from 11-23 ATA, 13% involved the labyrinth and nearly all of these (9 out of 11) required treatment. Of the remaining (nonlabyrinthine) accidents, a smaller proportion (49 out of 71) required treatment.
VESTIBULAR DISORDERS IN DIVING

Vestibular symptoms in divers have been mentioned in pressure chambers and open-sea dives for various working conditions, gas mixtures, and depths (Rubenstein and Summitt 1971). They have also been reported in submarines (Uffenorde 1948), caissons (Hill 1912), and following breath-hold dives (Pauley 1965). They have occurred during compression (Shilling 1937), just after decompression (Rivera 1963) or long after (Keays 1909), and under isobaric conditions (Sundmaker 1972). Symptoms appear to occur in guinea pigs and squirrel monkeys under high pressure (McCormick et al. 1971). Vestibular symptomatology (nausea and vertigo) are among the symptoms mentioned in connection with the high pressure nervous system syndrome (Överath et al. 1970; Bennett and Towse 1971; Choueau et al. 1971).

Residual vestibular defects have been reported both in divers (Lehmann et al. 1970; Plante-Longchamp et al. 1970; Kennedy 1972) and in caisson workers (Lestienne 1933; Rozsahegyi and Soos 1956; Rozsahegyi 1959; Rozsahegyi and Gomori 1961; Rozsahegyi 1966; Rozsahegyi and Roth 1966a). Furthermore, Bertoin (1953) feels these labyrinthine symptoms specifically worsen with time. Residual central nervous system deficits (EEG abnormality) were high (50%) in a group which experienced labyrinthine symptoms of decompression sickness (Rozsahegyi and Roth 1966b), although true control groups were not shown in that study.

In terms of the number of instances of vestibular-type involvement in decompression sickness, probably the best data were found in Rivera (1963) where, out of a total of 935 cases, “dizziness or vertigo” was reported 80 times; “nausea or vomiting” 74 times; “visual disturbances” 64 times; “incoordination” 9 times; “equilibrium disturbances” 7 times; and “auditory disturbance” 3 times. Although these symptoms often occurred along with others (notably localized pain), still dizziness, nausea, or visual disturbance was the premonitory sign in 5% of all the cases.

Studies of a history of vertigo only (as opposed to vestibular symptoms in general) in divers have shown that a high proportion of divers (i.e. between 12% and 40%—Lundgren 1965; Terry and Dennison 1966; Vorosmarti and Bradley 1970) have experienced vertigo at least once in their careers.

SUMMARY AND CONCLUSIONS

The main finding to emerge from this report is that vestibular accidents do occur in diving and caisson work and that, while the overall incidence of vestibular embarrassment may be small in comparison to all dives (e.g. 10-15 hits per 1,000 dives), it does occur frequently enough to warrant concern. Furthermore, because a vestibular hit may reflect involvement of the central nervous system it also appears more likely that permanent damage will ensue.

The second finding of this review is that the work of previous authors regarding vestibular problems is often overlooked. Very little mention of the vestibular organ complex is made in the U.S. Navy Diving Manual and other texts although localized pain is treated thoroughly. Vestibular problems may very well be underestimated and a modern review of diving accidents should be undertaken with this factor in mind.

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2 For an earlier, larger, but less delineated review (1,361,461 decompressions), Behnke (1951) should be consulted.
R. S. KENNEDY

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The author is now Head, Human Factors Engineering Branch, Naval Missile Center, Point Mugu, CA 93042.

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