Function of the eustachian tubes in divers with a history of alternobaric vertigo

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Tjernström, Ö. 1974. Function of the eustachian tubes in divers with a history of alternobaric vertigo. Undersea Biomed. Res. 1(4):343-351.—The ability to equilibrate middle ear pressure was studied in two groups of divers. One group consisted of 12 divers who had experience of vertigo which, based on the history, was considered to be alternobaric vertigo (i.e. to have been caused by middle ear overpressure). The other group consisted of 6 divers without vertigo experience. When exposed to simulated ascents and passive clearing of the ears in a pressure chamber, the 12 divers with vertigo experience required significantly higher middle ear pressures for passive opening of the eustachian tubes than the 6 divers without experience of vertigo during diving. Furthermore, 6 of the 12 divers with vertigo experience reported vertigo in the laboratory; nystagmus simultaneous with the vertigo episodes indicated that the latter were of vestibular origin.

alternobaric vertigo        eustachian tube function
                               vertigo

During diving and flying inadequate pressure equilibration of the middle ear may cause transient vestibular dysfunction and a form of vertigo known as alternobaric vertigo (Lundgren 1965). Information about alternobaric vertigo in divers has so far been available only in epidemiologic studies. It appeared from these studies that approximately 0.4% and 40% of divers may experience alternobaric vertigo while diving (Taylor 1959; Coles and Knight 1961; Lundgren 1965; Terry and Dennison 1966; Bayliss 1968; Vorosmarti and Bradley 1970). The same type of vertigo has been observed in flying (Melvill Jones 1957; Benson 1965; Lundgren and Malm 1966).

As for the mechanism of alternobaric vertigo, anamnestic information obtained from divers has provided some clues. Most divers with experience of vertigo during diving have observed that vertigo starts during ascents. This observation suggests that overpressure in the middle ears is important for the development of alternobaric vertigo, and this notion has gained further support from the observation of some divers that the vertigo may be relieved by interrupting the ascent and descending again (Lundgren 1965). Information received in interview studies, however, could only to a limited extent allow conclusions about the mechanism of the vertigo; a better foundation for such conclusions would be obtained by studying the phenomenon under laboratory conditions.

Alternobaric vertigo was earlier investigated in experimental studies of nondiving subjects. A relation was found between a unilateral high middle ear pressure and the occurrence of vertigo during simulated ascents in a pressure chamber (Ingelsted, Ivarsson, and Tjernström 1974; Tjernström 1974a; Tjernström 1974b). In a recent epidemiologic study of alternobaric vertigo in a large group of divers, questions were asked concerning differences between the
two ears with regard to the ease of passive equilibration of the middle ear pressure during ascent (Lundgren, Tjernström, and Örhagen 1974). Such differences were reported by significantly more divers with experience of alternobaric vertigo than by divers without such experience. It seemed worthwhile to apply to divers the experimental technique that had been used in the case of the nondiving subjects.

The aim of the present study was to compare divers with a history of vertigo during diving with divers without such a history with regard to a. the middle ear pressure level required for passive opening of the eustachian tube; and b. active tubal function, i.e. ability to achieve middle ear pressure equilibration by deglutitions.

METHODS

Eighteen subjects were selected for the present study from 2006 divers who had responded to a questionnaire on experiences of vertigo during diving (Lundgren et al. 1974). For economic reasons only subjects from the local area were selected. Out of these 18 divers, 12 had experienced vertigo during diving and 6 subjects had not. Based on the history the vertigo was thought to have been caused by middle ear overpressure. The diving experience ranged from 10 to 600 dives among the former 12 divers and from 60 to 1000 dives among the latter 6 divers. The results of the subjects' ENT examination on the day of the experiment were normal; the hearing threshold was within 0-20 dB (ISO Standard 1964).

In the present study the divers who had experienced alternobaric vertigo will be referred to as V-divers, and the divers who had never experienced vertigo will be referred to as NV-divers. Figure 1 gives an outline of the equipment used in the experiments. The essentials of the method are given here. For a more detailed technical description, see Ingelstetd, Ivarsson, and Jonson (1967) and Elner, Ingelstetd, and Ivarsson (1971a). The following symbols are used in text and figures:

- $P_{atm}$ atmospheric pressure at ground level (in the laboratory)
- $P_m$ pressure in middle ear
- $P_{tm}$ pressure gradient across eardrum
- $P_{ch}$ chamber pressure
- FPL passive forcing pressure level
- $V_{tm}$ volume displacement of eardrum in relation to its neutral position, outwards (+) and inwards (−)
- $\dot{V}_{tm}$ airflow through resistor of the ear canal flowmeter, caused by volume displacement of eardrum
- $\dot{V}_{oc}$ airflow through resistor of the ear canal flowmeter, caused by expansion or compression of gas volume in external ear canal and in flowmeter system induced by changes in the ambient pressure
- $\dot{V}_{ref}$ airflow through resistor of the reference flowmeter, caused by expansion or compression of gas volume in reference system induced by changes in the ambient pressure
- $\Delta$ before a symbol indicates a change of variable. Pressure is expressed in cm H$_2$O, volume in μl or ml and airflow in μl/sec
- $P_m$ and $P_{ch}$ are relative to atmospheric pressure in the laboratory (considered constant for the duration of an experimental session)
Fig. 1. Outline of the equipment used for recording eardrum displacement ($V_{tm}$) at changing ambient pressure ($P_{ch}$). The equipment as well as the subjects are placed in the pressure chamber. (For more details see text and list of symbols.)

A pressure chamber was used in which it was possible to simulate underwater ascents and descents by pressure changes amounting to 90 cm H$_2$O in 25 sec, the rate of pressure change being constant during 90% of the time. The subject, who was enclosed in the chamber, was fitted in each ear with a polyethylene catheter running through a rubber disc that was inserted into the inner bony part of the external ear canal. The other end of the catheter was attached to a flowmeter. Volume displacements of the eardrum giving rise to airflow ($V_{tm}$)

![Diagram](diagram.png)

Fig. 2. Example of a recording of eardrum displacement ($V_{tm}$ in μl) and chamber pressure ($P_{ch}$ in cm H$_2$O). Each recording starts at (a) after a pressure increase of 45 cm H$_2$O. The passive opening of the eustachian tube appears clearly on the $V_{tm}$ curve. $ΔP_{ch}$ is the change in chamber pressure required to force the tube open. After completion of pressure reduction, the middle ear pressure is equilibrated to the chamber pressure by deglutitions (arrows). Before the start of another examination the chamber pressure is increased (b) and the middle ear pressure is equilibrated by deglutitions (arrows).
in the catheter system could thus be recorded. This arrangement made it possible to record volume displacements of the eardrum caused by changes in ambient pressure ($P_{ch}$) or middle ear pressure ($P_m$). Since the airflow through the catheters depended both on $V_{tm}$ and on compression or expansion of the gas in the system, the latter portion of the flow ($V_{ec}$) had to be corrected for. This was achieved by creating a compression or expansion flow ($V_{ref}$) in the opposite direction to $V_{ec}$. $V_{ref}$ was obtained from an adjustable reference volume system operating a separate flowmeter. In the properly balanced system the signals derived from the $V_{ec}$ and the $V_{ref}$ cancelled out each other. The flow signal $V_{tm}$ was integrated to allow recording of $V_{tm}$. (The latter recording was required for separate determinations of pressure-equilibration capacity.) The sudden opening of the eustachian tube appeared clearly on the $V_{tm}$ curve (Fig. 2); hence it was possible to derive from the $P_{ch}$ curve the $\Delta P_{ch}$ required to induce a passive opening of the tube. Because of the elastic properties of the structures of the middle ear, including the eardrum, the actual relative overpressure in the middle ear ($P_{tm}$) must be somewhat lower than the $\Delta P_{ch}$. However, Ingelstedt et al. (1974) have shown that there is only a small difference between $P_{tm}$ and $\Delta P_{ch}$ in subjects with normal ears. The exact calculation of $P_{tm}$ requires extensive separate examinations of the subject, which were considered unnecessary for the present purpose. Therefore, the passive forcing pressure (FPL) was considered the same as $\Delta P_{ch}$ at the moment of passive opening of the tube. Simultaneous with the recordings of $P_{ch}$ and $V_{tm}$, recording of eye-movements was performed by means of electronystagmography (ENG, DC-recording) with the subjects in total darkness and with their eyes open (cf. Tjernström 1973).

Recording of the passive forcing pressure level (FPL) was performed with the subject in both the seated and recumbent positions. The latter position was attempted because it is known to influence the eustachian tubal patency, presumably by causing venous engorgement in the mucosa of the eustachian tube (Ingelstedt et al. 1967; Rundcrantz 1969; Tjernström 1974b). This was thought to offer an approximate parallel to the circulatory changes that might be induced by immersion in water (cf. Arborelius, Ballin, Lilja, and Lundgren 1972). Each period of recording started by increasing the chamber pressure to 45 cm H$_2$O above atmospheric pressure. Simultaneously the subject swallowed repeatedly to achieve pressure equilibration. The eardrum was checked for a neutral position from the $V_{tm}$ curve, after which a continuous pressure reduction started, simulating ascent through the water. After completion of the pressure reduction (totalling 90 cm H$_2$O), the pressure was again increased for a new examination. Between two and seven recordings were made in each posture. The subjects were instructed to avoid active clearing of the ears during the simulated ascents, to keep their eyes open looking straight ahead, and to avoid blinking. They also were to report vertigo when it occurred.

A subject's capacity for active middle ear pressure equilibration by deglutition was tested by changing the chamber pressure stepwise by 10 cm H$_2$O above and below atmospheric pressure and using the $V_{tm}$ curve as a criterion of equilibration between $P_{ch}$ and $P_m$. The subject performed deglutition repeatedly at stable chamber pressure; the fewer the swallows required to normalize middle ear pressure, the greater his capacity for active pressure equilibration. (For more details see Elner et al. 1971b.) Usually only one experiment of each type was made. To test whether active equilibration could provide protection against vertigo, the subjects were also exposed to continuous ascents of 90 cm H$_2$O, during which they were instructed to perform repeated deglutitions.

Calibration of the equipment for recording $V_{tm}$ and $P_{ch}$ was performed before and after each test according to the original method. The equipment for ENG was calibrated before and after each test for 30° eye movements in each direction.
RESULTS

The results presented in Table 1 demonstrate that in subjects without previous experience of vertigo during diving (Subjects I-VI) the individual mean forcing pressure in the seated

<table>
<thead>
<tr>
<th>Subject</th>
<th>Position</th>
<th>Right ear Mean (n)</th>
<th>Range</th>
<th>Left ear Mean (n)</th>
<th>Range</th>
<th>Tubal function</th>
<th>Vertigo</th>
<th>Nystagmus</th>
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<td>I</td>
<td>Seated</td>
<td>45(2)</td>
<td>3</td>
<td>43(2)</td>
<td>2</td>
<td>II/II</td>
<td>–</td>
<td>–</td>
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<td></td>
<td>Recumbent</td>
<td>49(2)</td>
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<td>10</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>II</td>
<td>Seated</td>
<td>40(5)</td>
<td>4</td>
<td>33(5)</td>
<td>5</td>
<td>Ib/Ib</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Recumbent</td>
<td>53(4)</td>
<td>2</td>
<td>44(4)</td>
<td>4</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>III</td>
<td>Seated</td>
<td>37(5)</td>
<td>2</td>
<td>32(5)</td>
<td>3</td>
<td>Ic/Ic</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Recumbent</td>
<td>58(6)</td>
<td>7</td>
<td>52(6)</td>
<td>8</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
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<td>Seated</td>
<td>44(7)</td>
<td>4</td>
<td>41(7)</td>
<td>8</td>
<td>Ib/Ib</td>
<td>–</td>
<td>–</td>
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<tr>
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<td>52(5)</td>
<td>3</td>
<td>45(5)</td>
<td>3</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>V</td>
<td>Seated</td>
<td>30(6)</td>
<td>5</td>
<td>41(6)</td>
<td>9</td>
<td>Ib/Ib</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Recumbent</td>
<td>45(6)</td>
<td>8</td>
<td>58(6)</td>
<td>13</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<td>Seated</td>
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<td>11</td>
<td>28(6)</td>
<td>9</td>
<td>Ic/Ic</td>
<td>–</td>
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<td>Recumbent</td>
<td>61(2)</td>
<td>8</td>
<td>42(3)</td>
<td>4</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>VII</td>
<td>Seated</td>
<td>64(3)</td>
<td>0</td>
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<td>1</td>
<td>Ic/Ib</td>
<td>–</td>
<td>–</td>
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<td>Recumbent</td>
<td>75(5)</td>
<td>7</td>
<td>55(4)</td>
<td>40</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>VIII</td>
<td>Seated</td>
<td>67(5)</td>
<td>8</td>
<td>50(5)</td>
<td>10</td>
<td>IV/II</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Recumbent</td>
<td>82(3)</td>
<td>4</td>
<td>62(3)</td>
<td>5</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>IX</td>
<td>Seated</td>
<td>53(5)</td>
<td>7</td>
<td>64(5)</td>
<td>12</td>
<td>Ic/Ic</td>
<td>–</td>
<td>–</td>
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<td>65(4)</td>
<td>3</td>
<td>71(4)</td>
<td>6</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<td>X</td>
<td>Seated</td>
<td>60(7)</td>
<td>18</td>
<td>53(7)</td>
<td>5</td>
<td>Ib/Ib</td>
<td>–</td>
<td>–</td>
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<td></td>
<td>Recumbent</td>
<td>65(6)</td>
<td>21</td>
<td>67(6)</td>
<td>11</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>XI</td>
<td>Seated</td>
<td>45(4)</td>
<td>4</td>
<td>48(5)</td>
<td>11</td>
<td>II/II</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Recumbent</td>
<td>63(2)</td>
<td>3</td>
<td>62(3)</td>
<td>7</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>XII</td>
<td>Seated</td>
<td>72(5)</td>
<td>33</td>
<td>76(5)</td>
<td>25</td>
<td>Ia/Ia</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Recumbent</td>
<td>87(3)</td>
<td>6</td>
<td>82(4)</td>
<td>3</td>
<td>Ib/Ib</td>
<td>+</td>
<td>+</td>
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<tr>
<td>XIII</td>
<td>Seated</td>
<td>92(5)</td>
<td>7</td>
<td>92(3)</td>
<td>4</td>
<td>+</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Recumbent</td>
<td>–</td>
<td></td>
<td>65(4)</td>
<td>8</td>
<td>Ic/Ic</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>XIV</td>
<td>Seated</td>
<td>–</td>
<td></td>
<td>70(2)</td>
<td>3</td>
<td>+</td>
<td>+</td>
<td>–</td>
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<td>70(2)</td>
<td>3</td>
<td>+</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>XV</td>
<td>Seated</td>
<td>67(4)</td>
<td>7</td>
<td>72(4)</td>
<td>8</td>
<td>Ic/II</td>
<td>+</td>
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<td>0</td>
<td>+</td>
<td>+</td>
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<tr>
<td>XVI</td>
<td>Seated</td>
<td>97(2)</td>
<td>7</td>
<td>113(4)</td>
<td>47</td>
<td>II/II</td>
<td>+</td>
<td>+</td>
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<td></td>
<td>Recumbent</td>
<td>71(2)</td>
<td>3</td>
<td>50(6)</td>
<td>9</td>
<td>II/II</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>XVII</td>
<td>Seated</td>
<td>74(3)</td>
<td>6</td>
<td>55(5)</td>
<td>14</td>
<td>+</td>
<td>+</td>
<td>–</td>
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<tr>
<td></td>
<td>Recumbent</td>
<td>73(5)</td>
<td>5</td>
<td>67(4)</td>
<td>17</td>
<td>Ib/Ib</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>XVIII</td>
<td>Seated</td>
<td>81(4)</td>
<td>5</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
<td>–</td>
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</table>

The pressure is given as mean and range, with number of examinations within ( ). Subjects I-VI had no history of alternobaric vertigo. Subjects VII-XVIII had experienced alternobaric vertigo during diving. Tubal function, vertigo and nystagmus episodes are also given. Total n = 18 subjects.
position was equal to or lower than 45 cm H₂O, which is essentially the same as the mean value (43.9 cm H₂O) of 79 otologically healthy nondiving subjects studied by Ingelstedt et al. (1974). Of the 12 subjects with a history of alternobaric vertigo (Subjects VII-XVIII), 11 were found to have high mean forcing pressures (>60 cm H₂O) on one side, and a comparison of the mean forcing pressure levels of the two groups of divers showed that the V-divers had a mean forcing pressure which was 29 cm H₂O higher than that of the NV-divers (P<0.001, Student’s t test). Furthermore, the V-divers who did not experience vertigo in the laboratory had a mean forcing pressure which was higher by 20 cm H₂O than that of the NV-divers (P<0.001, Student’s t test). Of the 12 V-divers, 6 reported vertigo when exposed to simulated ascents and passive clearing of the ears, both in the seated and in the seated position also did not report vertigo in the recumbent posture. During the vertigo episodes. Shifting to the recumbent position always caused an increase in the forcing pressure. In spite of the increase, however, the subjects who had not encountered vertigo in the recumbent posture also did not report vertigo in the seated position. During the examinations in which the subjects were instructed to perform active clearing of the ears there were no episodes of vertigo or nystagmus. Table 1 also presents the results from the examinations of the active tubal function. The tubal function was assigned according to the classification by Elner et al. (1971b).

Group I equilibrates pressure differences across the eardrum completely
Group II equilibrates pressure differences with small residual pressure in the middle ear
Group III is capable of equilibrating relative overpressure, but not underpressure
Group IV is incapable of equilibrating both over- and underpressure by deglutitions

Subgroups of Group I are:
Group Ia tuba aperta
Group Ib equilibrates small over- and underpressures in the middle ear in 1 to 3 deglutitions
Group Ic equilibrates in 4 to 10 deglutitions

As seen from the table, 10 ears out of 12 of the NV-divers belonged to Group I, and 2 ears of 12 belonged to Group II. In the V-divers 15 ears out of 24 belonged to Group I, 8 ears out of 24 to Group II, and one ear out of 24 to Group IV. This classification of the NV-divers and the V-divers did not reveal any statistically significant differences between the two groups.

DISCUSSION

Subjects with previous experience of alternobaric vertigo during diving had higher forcing pressures than subjects without such experience. It is noteworthy that it was also possible to elicit vertigo as well as nystagmus in 6 out of the 12 V-divers. Thus, the definition of alternobaric vertigo used in the interview study by Lundgren et al. (1974) apparently provided a material which, on experimental investigation, underscored the validity of the selection criteria used in the interview study.

The relation between a high forcing pressure level and the occurrence of vertigo during simulated ascents confirms the findings presented by Ingelstedt et al. (1974) in a group of
nondivers. They found it possible to elicit vertigo in 5 out of 79 otologically healthy
subjects, who were all found to have a high forcing pressure level on one side. The fact that 6
out of the 12 V-divers in this study did not get vertigo in the laboratory is in accordance
with the information obtained in the Lundgren et al. (1974) interview study, i.e. most
V-divers only occasionally experience alternobaric vertigo when diving.

The chances of getting alternobaric vertigo apparently increase for the individual diver as
he accumulates diving experience (Terry and Dennison 1966; Vorosmarty and Bradley 1970;
Lundgren et al. 1974). Yet, in a single experimental session alternobaric vertigo could be
elicited in 6 out of the selected group of 12 V-divers, who at ear examination were normal
and at nose and throat examination were without sign of catarrhal infection prior to the
session. Of these six who reported vertigo in the laboratory, three belonged to tubal function
groups lb and lc (very good tubal function) and the other three belonged to Group II (Table
1). This should be compared with the emphasis placed by Vorosmarty and Bradley (1970) on
pathological processes such as ruptured tympanic membranes or upper respiratory infections
appearing concurrent with episodes of vertigo. From this study it is clear that such
pathological processes as well as barotrauma are no prerequisites for alternobaric vertigo;
however, any condition which reduces the tubal patency might well increase the risk of
forcing pressure level becoming high enough to elicit vertigo. In 5 of the 12 V-divers no
vestibular stimulation appeared, even if their forcing pressures were as high as in some of the
6 V-divers who did report vertigo in the laboratory. Whether this difference was caused by
interindividual inequalities in the pressure levels required for a vestibular stimulation and/or
differences in pressure asymmetry between the two ears could not be evaluated. In an earlier
study (Tjernström 1974b) it was demonstrated that the magnitude of the asymmetry between
the forcing pressures of the two ears did not seem to affect the vestibular response.
From that investigation it appeared that only a certain minimum overpressure in one middle
ear was required. The present results seem to indicate a somewhat lower mean forcing
pressure level in the V-divers who had no vertigo in the laboratory when compared with the
mean forcing pressure level of the 6 V-divers who had vertigo, but this difference was not
statistically conclusive.

All experiments performed on subjects in the recumbent position showed higher forcing
pressures than in the seated position. Subjects who did not report vertigo when seated also
did not report vertigo when examined in the recumbent position. One possible explanation
might be that the overpressure required for a vestibular stimulation also increases in the
recumbent position (Tjernström 1974b). The magnitude of the increase in forcing pressure
in the recumbent position seems to confirm earlier observations (Tjernström 1974b) that an
initially low forcing pressure (<50 cm H₂O) increases more in the recumbent position than
an initially high forcing pressure (>60 cm H₂O).

As reported in the interview study by Lundgren et al. (1974), V-divers reported greater
difficulties in achieving pressure equilibration during both ascent and descent than the
NV-divers, which is in accordance with the results presented by Vorosmarty and Bradley
(1970). The results of the present study showed a statistically significant difference between
the V-divers and the NV-divers with regard to passive equilibration during ascents. They also
seem to indicate a difference in the capacity for active middle ear pressure equilibration
during descent between the two groups, but this difference was not statistically conclusive.

The conditions under which the active equilibration was tested differ greatly from those
with which a diver is faced during actual diving. Diving usually implies much faster ambient
pressure changes than were technically achievable in the present experiments. Therefore,
difference in active pressure equilibration capacity that makes itself felt during actual diving may not necessarily become obvious under the less strenuous conditions in the laboratory. Neither the present experimental studies nor those of Elner et al. (1971b) or Ingelstedt et al. (1974) show any relation between the tubal function according to Groups I-IV and the magnitude of the passive forcing pressures. In the study performed by Ingelstedt et al. (1974) it was demonstrated that the vertigo-prone nondiving subjects, all of whom had high passive forcing pressures, had a normal active tubal function (Groups 1b and 1c). But if a diver, because of a poor capacity for active pressure equilibration, is repeatedly exposed to barotrauma, it is conceivable that this might eventually reduce the patency of the eustachian tubes so as to increase forcing pressures sufficiently to make him vertigo-prone. In view of this possibility it is noteworthy that several studies have indicated a greater diving experience of V-divers than of N-V divers (Terry and Dennison 1966; Vorosmarty and Bradley 1970; Lundgren et al. 1974).

Apart from the possibility that diving itself might induce tubal middle ear or inner ear changes that increase the risk of alternobaric vertigo, it is quite conceivable that subjects who experience alternobaric vertigo during diving have been recruited from that part of the nondiving population which has earlier been shown to have high forcing pressures and to be susceptible to alternobaric vertigo in the laboratory (Ingelstedt et al. 1974). In some instances it might be of value to screen the passive forcing pressure of divers. Such a screening could be made in an ordinary pressure chamber. The subject in the chamber should undergo controlled ascent while it is recorded what pressure reduction is required to make him observe that the eustachian tubes open. Usually divers do not equilibrate actively during ascents, but wait for the ears to be cleared passively. Many divers with experience of alternobaric vertigo have pointed out that active pressure equilibration (also during ascents) eliminates or greatly reduces the risk of alternobaric vertigo. The present investigation confirms that this is a recommendable technique, since it prevents inducement of high overpressures in the middle ears during ascents.

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