Introductory remarks on this issue
On the role of the ampulla in disturbances of vestibular function

Hans Scherer and Satoru Watanabe

1 ENT-Clinic of the Free University of Berlin, Germany
2 Daido Institute of Technology, Nagoya, Japan

Introduction
In our daily clinical work on patients with vestibular disturbances we quite often have to deal with diseases, the pathophysiological basis of which we don’t know. Even the exact location of the pathology in many cases is not clear. While the mechanism of BPPN as a canalolithiasis or cupulolithiasis is solved to a large extent, the origin of sudden loss of vestibular function is as unknown to us as that of sudden hearing loss. Virus infections of Scarpae ganglion, acute disturbances of blood supply to the inner ear or microvascular problems are discussed. Only in rare cases we find the evidence for one or more of these causative factors. There remain doubts, because virus infections may not start as immediately as the sudden loss of vestibular function usually does and quite often young people are concerned having no evidence and risk factors for a circulatory problem.

The symptomatology of sudden loss of vestibular function with a strong horizontal nystagmus and a weaker torsional component is quite uniform. The question therefore arises, if mechanical changes within the vestibular organ may cause this entity, like the otoconias do in BPPN or endolymphatic hydrops does in Menière’s disease. As a horizontal nystagmus mainly is elicited by the sensor of the horizontal semicircular canal, the ampulla, our scientific field of vision is directed to this site of the vestibular organ.

A group of Japanese and German scientists (S. Watanabe, Y. Mizuno, H. Jijiwa; N. Watanabe et al., H. Scherer, K. Helling et al.) started experiments in the ampulla of the pigeon in order to create an animal model with a sudden loss of vestibular function.

The ampulla of the semicircular canals host the sensor for rotating motions in the plane of the canal, the crista ampullaris and the cupula. In 1938 Steinhausen and Dohlman presented pictures of the ampulla of pikes, in which the cupula moved like a swinging door. These movements were artifacts, due to rough dissection techniques, as Dohlman mentioned in later years. Today we know, that the cupula is attached to the ampullar wall and functions like a membrane. Movements of the membrane elicitated by rotating motions of the head take place in the lower part of the cupula with maximal deviation in the middle. Up to now the kind of fixation of the cupula at the ampullar wall is unknown. At least at its top the cupula seems to be attached only by pressure (f.i. inner turgor), as the cupula (fig. 1) as well as the ampullar roof (fig. 2) are smooth at their surface and do not show any sign of anatomical adhaesional structure. The kinocilia of the vestibular sensory cells have a ball-like thickening at their end (fig. 3 and 4). As Nagel et al. showed in electronmicroscopic pictures (fig. 5), the kinocilia, other than the stereocilia expand into the cupula, in which they are fixed by their thickening in a cavity. These cavities also can be seen in our cupular preparations (Helling et al.) (fig. 6). This mechanism probably allows the sensory cells to exert tension by changing its length similar to the
Scherer, H. and Watanabe, S.

functioning of outer hair cells in the cochlea.

In case, the cupula loses its attachment f. i. by shrinking or if the roof of the ampulla is lifted up the membrane effect is abolished and the sensor does not work any more.

Method

The method of preparation will be described here briefly. Exact documentations can be found in S. Watanabe et al. and will be published by K. Helling et al.. After unilateral exposure of the horizontal canal and its ampulla (fig. 7a), the bony canal was opened (fig. 7b). One side of the membranous canal was blocked and the other side punctured (fig. 7c). With colored artificial endolymph we raised the pressure in the ampulla on one side of the cupula gradually (fig. 7d and fig. 8) until the cupula was loosened. In this moment, a nystagmus appeared and the caloric reactivity, which was normal throughout all steps of the procedure, disappeared. The defect of the bony canal was covered with fibrin glue (fig. 7e) and the skin was closed. In the next days and weeks the animals were observed in respect to their static and dynamic disabilities. 6 pigeons were operated in Nagoya/Japan and 6 in Berlin/Germany in the described way. In 2 pigeons we added permanent damages of the vestibulum to the loosening of the cupula.

Results

We succeeded in loosening the cupula by raising the endolymph pressure on one side of the ampulla. Further experiments on this item and the pressure needed to mobilize the cupula are described later in this issue (N. Watanabe et al.). The endolymphatic membrane was not damaged by this maneuver, as perilymph was uncolored
Introductory remarks on this issue

throughout the experiment.

The eyes, being observed with video cameras, showed a nystagmus, which started in the moment, in which the cupula lost its attachment. On the next day the pigeons were able to stand upright, but the head was tilted to the operated side and we saw a compensatory head nystagmus. When provocation occurred (rotation on a chair), the pigeons fell to the operated side. These symptoms declined gradually and vanished totally after 15 - 20 days. The pigeons were able to fly. Pigeons, in which we had added a defect of the vestibulum, did not compensate and they lost their flying capacity.

Discussion

The experiments demonstrated clearly, that the cupula of the horizontal semicircular canal could be loosened by a slowly increase of endolymphatic pressure on one side of the ampulla. We established a chronic animal model with this type of mechanical change of the ampulla with a loss of membrane function. The symptoms we saw in the animals were identical with those of a sudden loss of vestibular function in human. The animals compensated well and quite rapidly, they did not compensate when we destroyed more than the cupula attachment.

These typical symptoms of the loss of vestibular function, arising in pigeons after loosening the cupula from the ampulla roof show, that mechanical changes within the vestibular organ may be the cause of sudden loss of vestibular function in humans. The cupula and its attachment to the ampulla wall seem to play a role role in this disease.

The question, if in case of a sudden loss of vestibular function in human the cupula shrinks or if the constant development of the cupula is disturbed or if the roof of the ampulla can be lifted up, will be investigated by our groups in future.

As a resume one can say, that the ampulla of the semicircular canals shall be taken into consideration in vestibular disease of unknown origin, such as the sudden loss of vestibular function.

In march 2001 a symposium took place in Nagoya/Japan on the morphology and physiology of semicircular ampullas, the papers of which being presented in this issue. This symposium was sponsored by the Japan Society for the Promotion of Science (JSPS), the Deutsche Forschungsgemeinschaft (DFG) and the Daido Institute of Technology. To all these we express our thanks. This symposium brought together the scientists of our both countries, being active in this special field of the vestibular organ. Vestibular research has a long tradition in Germany and in Japan. We hope, that we gain further knowledge by this scientific cooperation.

Conclusion

It is discussed, if the sudden loss of vestibular function may be attributed to mechanical changes within the labyrinth. The mainly horizontal nystagmus of this disease leads our attention to the ampulla of the horizontal canal. The sensor, located there, uses the cupula as a membrane. The kind of attachment to the ampullar wall still is unknown. By loosening this attachment the membrane function is cancelled and by that the reactivity of the sensor.

We created a chronic animal model, in which we loosened the cupula from the ampullar roof by raising the endolymph pressure on one side of the cupula. We could show, that the typical symptoms of a sudden loss of vestibular function occur in the moment, the membrane is open.

These experiments gave the idea to this symposium, the papers of which are presented as follows.

Fig. 7 (a) Schematic drawing of the horizontal canal with its ampulla like seen during the operation. (b) Opening of the bony canal from above. (c) Blockade and puncture of the membraneous canal. (d) Injection of artificial and colored endolymph. (e) Closure of the bony canal with fibrin glue.

Fig. 8 Picture of the operation with small picture of the eye.