Iatrogenic vestibular disorders may result as inadvertent effects of the management of dizzy patients or patients with disorders unrelated to the vestibular system. The word “iatrogenic” is commonly used incorrectly in the narrower context of a drug-induced condition. This is an error since the term “iatrogenic” is derived from the Greek “iatros,” meaning a “physician,” and when combined with the word “genesis,” the term is correctly applied to any symptom or condition created by the physician (Ballantyne 1970). In modern usage, this includes the surgeon. Iatrogenic vestibular disorders include both the avoidable and the unavoidable consequences of:

- drug treatment,
- physical therapy, or
- surgical procedures.

In the broadest sense, they may also be the result of medical counsel to the patient or the patient’s failure to comply with therapy.

The following examples of iatrogenic vestibular disorders do not exhaust all the possibilities:

- drug treatment
  Dizziness and postural imbalance are among the most frequent adverse effects of an impressive list of drugs described elsewhere (Chap. 28). In addition to dosage-dependent reversible side effects, there is also irreversible vestibular loss caused by, e.g., gentamicin or quinine.

- physical therapy
  Chiropractic neck manipulations may cause vertebral artery dissection with subsequent PICA infarction or thrombosis of the basilar artery. Physical liberatory manoeuvres for treatment of posterior canal benign paroxysmal positioning vertigo may lead to transitions to anterior or horizontal canalolithiasis.

- surgical procedures
  Inner ear surgery and neurosurgery of the cerebellopontine angle may damage the labyrinth or the vestibular nerve. Benign paroxysmal positioning vertigo may be caused by ear or head surgery. Selective vestibular neurectomy or plugging of the semicircular canals can cause chronic functional vestibular insufficiency.

- medical counsel
  A simple prescription of bedrest for a non-vestibular disorder can in the susceptible patient lead to benign paroxysmal positioning vertigo.

- avoidable vestibular disorders
  Early identification of a vertebral artery dissection following minimal neck trauma may prevent subsequent Wallenberg’s syndrome or basilar artery thrombosis if treated immediately with anticoagulants. Likewise early immunosuppressive therapy for Cogan’s syndrome can prevent bilateral vestibular failure. Avoidance of exposure to chronic high levels of noise prevents not only noise-induced hearing impairment but also vestibular deficiency in industrial workers or military personnel.

Intratympanic gentamicin in Menière’s disease: desired and undesired effects

Intratympanic injections of gentamicin are increasingly used to treat intractable vertigo or drop attacks due to Menière’s disease (p. 91). Obviously it is possible to damage selectively the dark cells of secretory epithelium (and thereby improve endolymphatic hydrops) before significantly affecting vestibular and cochlear functions. In fact, treatment is often effective even if it does not impair caloric responses. However, Magnusson et al. (1991) warned that the onset of ototoxic effects of gentamicin is delayed. Oxidative damage of the mitochondria (Hutchin and Cortopassi 1994) and blockage of transduction channels of hair cells (Kroese et al. 1998) may trigger hair cell death.
These multistage mechanisms are consistent with the fact that gentamicin ototoxicity is reversible at an early stage but can become irreversible at a late stage (Halmagyi et al. 1994). After 1 to 4 years of treatment 5 of 18 patients developed oscillopsia and ataxia, symptoms and signs of (presumably permanent) chronic vestibular insufficiency (Murofushi et al. 1997). Responses of the vestibulo-ocular reflex following unilateral vestibular deafferentation by gentamicin ablation showed a strong asymmetry with permanent, reduced gains toward the side of the lesion (Allison et al. 1997). The proportion of chronic vestibular insufficiency following this kind of treatment was not lower than that after selective vestibular neurectomy or surgical labyrinthectomy. Therefore, earlier procedures involving serial intratympanic injections of gentamicin have been revised. The current tendency is to treat these patients with low concentrations of gentamicin and one injection a week on an outpatient basis, while carefully monitoring audiovestibular function and suppression of the distressing attacks (p. 91).

**Vertebral artery dissection due to chiropractic neck manipulation**

A survey of 84 cases of vertebral artery dissection reported minor traumas as the main cause; 70% of these patients were in their third or fourth decade of life (Shimizu et al. 1992). The probable traumas preceding the dissection were neck manipulations, especially chiropractic procedures (52%). The delayed onset after the neck trauma could take more than 1 week, but in most cases it occurred within less than 24 hours. Cervical rotation and extension are believed to be the causative movement, and the third segment of the vertebral artery is considered the most vulnerable. Vertigo may be the first symptom of vertebral artery dissection after chiropractic neck manipulations (Vibert et al. 1993). Wallenberg’s syndrome (p. 309) is a common complication of vertebral artery dissection.

Violent head movements and sports may cause vertigo by mechanisms other than that of arterial dissection. A selective vulnerability of the otoliths and the organ of Corti has been reported in cases of audiovestibulopathy induced by high impact aero-bics (Weintraub 1994). Since tinnitus, “ear-fullness,” and sensitivity to barometric pressure change (flying, scuba, swimming) were also present in about 60% of the described 30 individuals, it is more likely that excessive Valsalva manoeuvres caused perilymph fistula (p. 99) in this subgroup. Perilymph fistula can result from strenuous physical exercise, heavy lifting or sneezing (Sakikawa et al. 1994), manipulation of the ear (cleaning cerumen with cotton swabs or sticks (Kubo et al. 1993), or water-jet irrigation (Wurtele 1981; Anon and Miller 1985).