Antivertigo Medications and Drug-Induced Vertigo
A Pharmacological Review

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Contents

Summary ................................................................. 778
1. Pathophysiological Basis of the Drug Treatment of Vertigo  .................................................. 778
  1.1 Neuroanatomical and Neurochemical Bases ................................................................. 778
  1.2 Mechanisms to which Vertigo Is Commonly Attributed .................................................. 779
2. Aims of Drug Treatment ........................................... 780
  2.1 Specific Treatment .................................................. 780
  2.2 Symptomatic Treatment .......................................... 780
    2.2.1 Vestibular Suppression ........................................ 780
    2.2.2 Vestibular Compensation ..................................... 780
    2.2.3 Accompanying Symptoms ...................................... 781
3. Pharmacological Possibilities ..................................... 781
  3.1 Anticholinergics .................................................. 781
  3.2 Antihistamines .................................................... 781
  3.3 Histaminergic Medications ....................................... 782
  3.4 Antidopaminergic Drugs ........................................... 782
  3.5 Benzodiazepines ................................................... 783
  3.6 Calcium Antagonists ............................................. 783
  3.7 Sympathomimetics .................................................. 784
  3.8 Acetylleucine ........................................................ 784
  3.9 Miscellaneous .................................................... 784
4. Therapeutic Use ................................................... 785
  4.1 General Comments .................................................. 785
  4.2 Specific Indications ............................................... 785
    4.2.1 Vestibular Neuritis ............................................ 785
    4.2.2 Benign Paroxysmal Positional Vertigo .................................................. 785
    4.2.3 Ménière’s Disease ............................................. 785
    4.2.4 Vertigo of Unknown Origin ...................................... 786
5. Vertigo as an Adverse Effect of Drugs ............................ 786
  5.1 Drugs Causing ‘Pseudovertigo’ of Nonvestibular Origin ............................................ 786
  5.2 Otoxins ............................................................ 786
  5.3 Drugs Inducing a Central Vestibular Syndrome ..................................................... 787
  5.4 Drugs Hindering Vestibular Compensation ........................................................... 788
6. Conclusion .......................................................... 788
Summary

The approach to drug treatment of vertigo is almost exclusively symptomatic. There are 3 major goals for drug treatment of vertigo. The first one is to eliminate the hallucination of motion. Drugs with vestibular ‘suppressant’ properties are used for this purpose. The major vestibular suppressants are anticholinergic and antihistamine drugs. The second goal is to reduce the accompanying neurovegetative and psychoaffective signs (nausea, vomiting, anxiety). Antidopaminergics are used for this purpose. The third goal is to enhance the process of ‘vestibular compensation’ to allow the brain to find a new sensory equilibrium in spite of the vestibular lesion. Until now, the pharmacological manipulation of vestibular compensation has been assessed in animals but not in humans with vestibular lesions. Vestibular suppressant drugs delay rather than enhance compensation. A variety of other drugs is also used in the treatment of vertigo, including benzodiazepines, histaminergic agents, sympathomimetics and calcium antagonists. Their mechanism of action is poorly understood.

The data base derived from clinical trials evaluating antivertigo medications is often questionable because of methodological limitations. This explains why habits of prescription are mainly empirical, and why striking differences can be noticed from one country to another. We can hope that new treatments may emerge from the present interest in receptor subclasses and neuromodulators of the vestibular system, and we must be ready to evaluate these potential new pharmacological agents with reliable clinical methods in humans.

This article discusses the treatment of ‘true’ vertigo, which is defined as hallucination of motion, generally spinning, caused by a lesion of the vestibular system. This definition therefore excludes motion sickness, orthostatic hypotension, phobic syndromes and other conditions of nonvestibular origin.

It is important to understand that our present understanding of the pathophysiology of vertigo limits a discussion of antivertigo medication to an approach that is almost exclusively symptomatic.[1,2] Vertigo is a symptom common to diverse lesions of the vestibular system, which can vary in location and pathophysiology. Unfortunately, presently available clinical testing and explanations are often inadequate to define the location and extent of many vestibular lesions precisely.

1. Pathophysiological Basis of the Drug Treatment of Vertigo

Healthy persons use a partially redundant system of motion processing incorporating 3 primary streams of sensation: visual, vestibular (inner ear) and somatosensory. These 3 streams of information are combined in the brain to form an estimate of head and body orientation and motion. When there is a mismatch between information carried on two or more senses, vertigo is perceived.

Vertigo is primarily of otological origin, caused by dysfunction of the inner ear structures, because the semi-circular canals in the inner ear are rotational velocity sensors. Disruption of vestibular CNS circuitry that processes motion can also cause an illusion of motion.

1.1 Neuroanatomical and Neurochemical Bases

For reviews of vestibular neurochemistry, see Matsuoka et al.,[3] Raymond et al.[4] and Dewaele et al.[5]

Much of the aim of drug treatment is to manipulate neurotransmitters involved in vestibular transmission. There are several neurotransmitters influencing the ‘3 neuron arc’ between the vestibular hair cells and oculomotor nuclei that drives the vestibulocular reflex. Cholinergic, monoaminergic and glutamatergic synapses have been demon-