Practical Therapeutics

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Treatment of Postural Hypotension
A Review

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Contents

Summary ........................................................................................................................................... 75
1. Nonpharmacological Treatment ................................................................................................. 75
2. Pharmacological Treatment ........................................................................................................ 75
   2.1 Mineralocorticoids .................................................................................................................. 76
      2.1.1 Mechanism of Action ....................................................................................................... 76
      2.1.2 Adverse Effects ................................................................................................................ 77
   2.2 Prostaglandin Synthetase Inhibitors ...................................................................................... 77
      2.2.1 Mechanism of Action ....................................................................................................... 77
      2.2.2 Adverse Effects ................................................................................................................ 77
   2.3 Somatostatin .......................................................................................................................... 77
      2.3.1 Mechanism of Action ....................................................................................................... 78
      2.3.2 Adverse Effects ................................................................................................................ 78
   2.4 α-Sympathomimetic Agents .................................................................................................. 78
      2.4.1 Tyramine and Tyramine/Monoamine Oxidase Inhibitor Combinations ....................... 78
      2.4.2 Mechanisms of Action ..................................................................................................... 79
      2.4.3 Adverse Effects ................................................................................................................ 79
   2.5 Dihydroergotamine and Caffeine .......................................................................................... 79
      2.5.1 Mechanism of Action ....................................................................................................... 79
      2.5.2 Adverse Effects ................................................................................................................ 80
   2.6 Ergotamine ............................................................................................................................. 80
      2.6.1 Mechanism of Action ....................................................................................................... 80
      2.6.2 Adverse Effects ................................................................................................................ 80
   2.7 Dopamine Antagonists .......................................................................................................... 80
      2.7.1 Mechanism of Action ....................................................................................................... 80
      2.7.2 Adverse Effects ................................................................................................................ 80
   2.8 Clonidine ................................................................................................................................ 80
      2.8.1 Mechanism of Action ....................................................................................................... 81
      2.8.2 Adverse Effects ................................................................................................................ 81
   2.9 Yohimbine .............................................................................................................................. 81
      2.9.1 Mechanism of Action ....................................................................................................... 81
      2.9.2 Adverse Effects ................................................................................................................ 81
   2.10 Drugs Interacting with β-Adrenoceptors ............................................................................. 81
      2.10.1 Mechanism of Action ..................................................................................................... 83
      2.10.2 Adverse Effects ................................................................................................................. 83
   2.11 Drugs Influencing Water Balance ....................................................................................... 83
      2.11.1 Mechanism of Action ..................................................................................................... 83
      2.11.2 Adverse Effects ................................................................................................................. 83
3. Approach to Treatment ............................................................................................................. 83
Summary

Many drugs have been used in treating patients with postural hypotension but for a large number the evidence of benefit is small and the potential for adverse effects, particularly supine hypertension, is great. Full clinical assessment is essential at the outset to define the nature and extent of pathophysiological disturbance of autonomic function. Many patients can be treated adequately by sleeping with the head of the bed elevated, and the use of fludrocortisone. Patients without evidence of central neurological deficit may benefit from additional treatment with drugs which alter \( \beta \)-adrenoceptor tone. Patients who respond poorly to these measures should be admitted to hospital, and treatment with desmopressin initiated. Symptomatic postprandial hypotension should be identified early since the response to these measures alone is often poor; caffeine administered before eating, with abstinence for the rest of the day, may be very helpful.

Symptoms of dizziness and syncope due to postural hypotension may result from a wide variety of conditions, many of which are apparent on clinical assessment (e.g. dehydration, blood loss, severe myocardial disease). Occasionally, phaeochromocytoma or Addison's disease may be responsible. However, the most important cause of chronic recurrent symptomatic postural hypotension is autonomic failure (table I). This may be due to pure autonomic failure (Bradbury-Eggleston syndrome) characterised by widespread degeneration of sympathetic nerves, particularly those arising in the intermediolateral column cells of the cord; in multisystem atrophy (MSA) there is additional clinical evidence of more widespread central neurological disease with Parkinsonian features, impairment of ocular and pupillary movements and often spasticity. Simple testing of reflex responses is important in confirming the diagnosis and in defining the extent and nature of the disease (Bannister & Mathias 1988).

1. Nonpharmacological Treatment

Treatment should be oriented towards improving postural symptoms rather than solely improving falls in orthostatic blood pressure. The range of blood pressure over which autoregulation of cerebral blood flow occurs is reset to a lower level in autonomic failure, leading to relative tolerance to profound hypotension (Thomas & Bannister 1980). Diurnal fluctuation, with marked symptoms on rising from bed in the morning, is a characteristic feature which results from overnight recumbent polyuria leading to contraction of plasma volume (Wilcox et al. 1974). Sleeping with the head of the bed elevated reduces overnight fluid loss and has a major role in the nonpharmacological management of these patients (Bannister et al. 1969). Patients with autonomic failure are very sensitive to changes in blood volume and have impaired ability to conserve fluids during dehydration (Wilcox et al. 1974). Education to avoid dehydration and maintain adequate sodium intake is therefore important.

2. Pharmacological Treatment

Drug treatment of postural hypotension is difficult. Response and tolerance to different drugs varies markedly between patients, possibly because

<table>
<thead>
<tr>
<th>Table I. Autonomic disorders associated with chronic postural hypotension</th>
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<tbody>
<tr>
<td>Predominantly peripheral</td>
</tr>
<tr>
<td>Primary</td>
</tr>
<tr>
<td>Pure autonomic failure (Bradbury-Eggleston syndrome)</td>
</tr>
<tr>
<td>Familial dysautonomia (Riley-Day syndrome)</td>
</tr>
<tr>
<td>Congenital noradrenergic failure</td>
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<tr>
<td>Secondary</td>
</tr>
<tr>
<td>Baroreceptor dysfunction (tumour, surgery or irradiation)</td>
</tr>
<tr>
<td>Associated with systemic disease (usually with motor/sensory nerve involvement), e.g. diabetes mellitus, amyloidosis, cord lesions, porphyria, tabes dorsalis</td>
</tr>
<tr>
<td>Predominantly central</td>
</tr>
<tr>
<td>Multisystem atrophy (Shy-Drager syndrome)</td>
</tr>
<tr>
<td>Idiopathic Parkinsonism</td>
</tr>
</tbody>
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