Effect of Abana (an Ayurvedic Preparation) on Rabbit Atrium

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INTRODUCTION
Several herbs are reputed to have cariotonic action according to literature on traditional Indian medicine. Some of these have been proved to have pharmacological actions, e.g. Rauwolfia and Jatamansi. Abana is a compound preparation of several reputed remedies and initial studies have demonstrated the possibility of novel cardiac actions. Hence a detailed study was undertaken on its effect on rabbit atrium.

MATERIALS AND METHODS
Rabbits were killed by a blow on the head and their atria removed quickly and mounted in an isolated organ bath and the rate and amplitude of contraction were recorded on a smoked drum by Starling’s heart lever. The bathing fluid was the same as used for Langendorff preparation – pH 7.4; and bath temperature 37°C. The effects of various doses of noradrenaline (N.A.), isoprenaline (I.S.O.), acetylcholine (Ach, histamine and calcium chloride (CaCl2) were recorded. Besides control rabbits, animals treated with Abana, isoprenaline, isoprenaline + Abana, atenolol and atenolol + Abana were used to obtain preparations.

COMPOSITION
Each Abana tablet contains:
Exts.:  
- Terminalia arjuna (Arjun) 30 mg  
- Withania somnifera (Ashwagandha) 20 mg  
- Tinospora cordifolia (Giloe) 10 mg  
- Nepeta hindoostana (Billilotan) 20 mg  
- Phyllanthus emblica (Amla) 10 mg  
- Terminalia chebula (Hilda) 10 mg  
- Dashamoola 20 mg  
- Eclipta alba (Bhrangraj) 10 mg  
- Glycyrrhiza glabra (Yashtimadhu) 10 mg  
- Asparagus racemosus (Shatavar) 10 mg  
- Boerhaavia diffusa (Purnarunava) 10 mg  
- Centella asiatica (Brahmi) 10 mg  
- Convolvulus pluricaulis (Shankhpushpi) 10 mg  
- Ocimum sanctum (Tulsi) 10 mg  
- Nardostachys jatamansi (Jatamansi) 10 mg  
- Cyperus rotundus (Motha) 5 mg  
- Acorus calamus (Vach) 5 mg  
- Embelia ribes (Vidang) 5 mg  
- Piper longum (Pipali) 10 mg  
- Carum copticum (Ajwain) 10 mg

Processed in Abresham, Onosma bracteatum (Gaozoban), Phyllanthus emblica (Amla), Centella asiatica (Brahmi), Rosa damascena (Gulab ka phool), Nelumbium speciosum (Kamal ka phool), Punica granatum (Anar), Pyrus malus (Seb), Convolvulus pluricaulis (Shankhpushpi), Asparagus racemosus (Shatavar), Aloe vera (Ghikanwar), Nepeta
hindostana (Billilotan), Ocimum sanctum (Tulsi), Foeniculum vulvare (Sonf), Vetiveria zizaniodes (Khas), Daucus carota (Gajar).

Doses and duration of pre-treatment

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose (mg/kg)</th>
<th>Route</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abana</td>
<td>1</td>
<td>orally</td>
<td>twice a day</td>
</tr>
<tr>
<td>Isoprenaline</td>
<td>1</td>
<td>i.p.</td>
<td>twice a day</td>
</tr>
<tr>
<td>Atenolol</td>
<td>3</td>
<td>orally</td>
<td>once a day</td>
</tr>
</tbody>
</table>

RESULTS

The amplitudes of contraction of atria derived from control rabbits, rabbits receiving various pre-treatments and the effects of noradrenaline, isoprenaline, acetylcholine and histamine added in vitro are shown in Figure 1. The basal amplitude of atria removed from rabbits pre-treated with Abana for 3 days was markedly higher than the basal amplitude in untreated animals or those pre-treated with other drugs. The effect of noradrenaline added in concentrations of 0.01 and 0.1 µg/ml was abolished by pre-treatment with Abana for 3 days. Though the increase in amplitude was discernible even after cessation of pre-treatment for 48 hours, the inhibitory effect of Abana on the positive inotropic action of noradrenaline was waning at this time. The effect of isoprenaline, 0.01 µg and 0.1 µg, was also abolished by pre-treatment with Abana. The actions of acetylcholine and histamine were not affected by Abana pre-treatment. Chronic pre-treatment with isoprenaline or Abana and isoprenaline reduce the basal amplitude but did not affect responses to isoprenaline in vitro. Atenolol and atenolol plus Abana reduced basal amplitude and the in vitro responses to isoprenaline were also abolished.

The effect of noradrenaline (N.A.) on the rate of atria obtained from untreated rabbits and rabbits treated with Abana, phenoxybenzamine (P.B.Z.) or Abana plus phenoxybenzamine are depicted in Fig. 1. Abana pre-treatment reduced the responses to lower doses of noradrenaline whereas responses to higher doses were not affected. The basal heart rate was not different in the two groups. Phenoxybenzamine did not alter the response to lower doses but reduced the response to higher doses.

Fig. 2 shows the effect of isoprenaline (I.S.O.) on the rate of atria obtained from untreated rabbits and rabbits pre-treated with Abana, atenolol or Abana plus atenolol. Abana pre-treatment did not affect the response to lower doses but reduced the responses to higher doses of isoprenaline. Atenolol pre-treatment reduced the response to all doses of isoprenaline. Abana plus atenolol did not show an additive effect.

The effect of isoprenaline (I.S.O.) on the rate of atria obtained from untreated rabbits and rabbits pre-treated with isoprenaline or Abana with isoprenaline and Abana are depicted in Fig. 3. In both pre-treated groups the response to higher doses of isoprenaline was reduced.
Fig. 4 depicts the effect of calcium chloride (CaCl$_2$) in partially depolarized atria obtained from untreated and Abana-treated rabbits. The effect of calcium in increasing the amplitude was enhanced by Abana pre-treatment.

DISCUSSION
The experiments demonstrate a marked effect of Abana in increasing the amplitude of contraction of the atrium. This effect was negligible with Abana treatment for one day (not reported) but became appreciable with treatment for 3 days. The effect on basal amplitude was evident 48 hours after discontinuation of Abana treatment. The decrease in amplitude caused by partial depolarization was also less in Abana-treated animals. The positive ionotropic effects of noradrenaline and isoprenaline were abolished by Abana. As expected, atenolol pre-treatment markedly reduced the basal amplitude and abolished the effects of isoprenaline. Abana and atenolol pre-treatment produced effects like atenolol pre-treatment. The positive ionotropic effects of histamine and negative ionotropic effects of acetylcholine were not affected by pre-treatment with Abana.

Abana pre-treatment did not alter the basal rate of atria. However, pre-treatment with Abana significantly reduced the responses to lower doses of noradrenaline and higher doses of isoprenaline. Phenoxybenzamine, an alpha-blocker, reduced responses to higher doses of noradrenaline and, therefore, it appears that in the rabbit atrium, alpha-agonistic activity at higher doses is also responsible for positive chronotropic action. Chronic isoprenaline pre-treatment reduced chronotropic responses of atria to higher doses of isoprenaline added in vitro in the same way as pre-treatment with Abana did. Isoprenaline pre-treatment causes down-regulation of beta-receptors (Nomura et al., 1980) and probably chronic pre-treatment with Abana also caused similar down-regulation of beta-receptors. The action of Abana in increasing the amplitude of contraction was remarkable. This appears to be independent of beta-receptors, since with increased amplitude the effect of noradrenaline and isoprenaline on the amplitude were abolished. Also atenolol – a beta-blocker – had quite an opposite effect on amplitude, namely reduction in basal amplitude. Thus Abana is quite distinct from beta-blockers, i.e. while causing down-regulation of beta-receptors it increased the amplitude of contraction (See Table 1).
## Table 1: Percentage change in amplitude from basal values

<table>
<thead>
<tr>
<th></th>
<th>Control (Pre-treatment)</th>
<th>Abana</th>
<th>Abana 48 hr. after discon.</th>
<th>ISO</th>
<th>Abana + ISO</th>
<th>Atenolol</th>
<th>Abana + Atenolol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal Amplitude (mm)</td>
<td>3.90</td>
<td>7.8</td>
<td>7.3</td>
<td>2.5</td>
<td>1.37</td>
<td>1.66</td>
<td>1.66</td>
</tr>
<tr>
<td>NA (µg/ml)</td>
<td>0.01 + 15</td>
<td>–13%</td>
<td>–13%</td>
<td>+18%</td>
<td>+25%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ISO (µg/ml)</td>
<td>0.01 + 86</td>
<td>–32</td>
<td>+100</td>
<td>+118</td>
<td>+20</td>
<td>+60</td>
<td></td>
</tr>
<tr>
<td>Ach (µg/ml)</td>
<td>0.1 – 53</td>
<td>–45</td>
<td>+128</td>
<td>+182</td>
<td>–10</td>
<td>–20</td>
<td></td>
</tr>
<tr>
<td>Histamine (µg/ml)</td>
<td>0.1 + 12</td>
<td>+11</td>
<td>+182</td>
<td>+20</td>
<td>–10</td>
<td>–20</td>
<td></td>
</tr>
</tbody>
</table>

Both these effects could be very beneficial. By down-regulation of beta-receptors it protects the heart against excessive sympathetic out-bursts and by increasing the amplitude of contraction it maintains cardiac output without producing basal bradycardia. The drug appears to have novel pharmacological effects and deserves critical evaluation.

**REFERENCES**


