SOME PHARMACOLOGICAL ACTIONS OF THE ESSENTIAL OIL OF *BLUMEA MEMBRANACEA*

S. C. MEHTA, H. VARDHAN AND S. P. SAXENA

*Department of Pharmacology,*
*G. R. Medical, College Gwalior - 472 002*

(Received on October 27, 1985)

**Summary:** The essential oil obtained from the plant *Blumea membranacea* produced a marked and long lasting fall in blood pressure in anaesthetized dogs. The oil exerted a direct depressant action on frog heart and spasmolytic effect on rabbit ileum. It also depressed the conditioned avoidance response, adversely affected rotarod performance and potentiated the pentobarbitone induced hypnosis in rats.

**Key words:** *blumea membranacea* hypotensive essential oil CNS depression

**INTRODUCTION**

*Blumea membranacea* (Vern.: Mharbir; Family: Compositae) is a common and variable weed. It flowers during February to March and bears violet pink coloured flowers. The leaves are advocated as stomachic, antispasmodic and diaphoretic (5, 7). As no work seems to have been carried out on the pharmacological actions of the essential oil of *Blumea membranacea*, some actions are described in this report.

**MATERIAL AND METHODS**

**Extraction:** The whole plant at its full flowering stage was subjected to steam distillation. It yielded 0.03% of a reddish brown essential oil having a specific gravity of 0.9870.

**Pharmacological studies:** The essential oil was employed in the form of an emulsion in 2% Polysorbate 80. Control animals received the vehicle alone.
Intact animal experiments:

Dogs:

Dogs of either sex (4-7 kg) were anaesthetized with chloralose (80 mg/kg, iv). Blood pressure was recorded with a mercury manometer from a common carotid artery. Respiration was recorded by a tambour connected to tracheal cannula.

Rats:

Colony bred Swiss adult albino rats of either sex weighing 150-180 g were used.

Preliminary screening: Effects of vehicle and of the essential oil (100 and 200 mg/kg) given ip was screened as described by Turner (9).

Spontaneous motor activity: Pairs of rats (drug or vehicle treated) were placed in Photoactmeter (6) for 15 min and then their activity was recorded for next 20 min. Mean of 5 such observations were computed as mean activity.

Forced motor activity: This was carried out using a rotarod test (3).

Barbiturate sleep: Effect of vehicle and the drug (100 and 200 mg/kg) given ip on sleep induced by pentobarbitone sodium (30 mg/kg) given 30 min later was studied in groups of 10 rats each.

Conditioned avoidance response: (CAR): Vehicle and drug treated rats trained for CAR in pole climbing apparatus (4) were tested for abolition of CAR 30 min after ip injections.

Hypothermia: Rectal temperature of rats was recorded by a thermometer before, 30 min after and later on, every 1 hr after ip injection of vehicle or the drug.

Isolated tissue experiments: Preparations used were isolated perfused hearts of frog, Rana tigrina (1), rabbit ileum (1), guineapig tracheal chain (2) and frog rectus abdominis muscle (1).

RESULTS

Intact animal experiments:

Dogs:

The essential oil (25 mg/kg, iv) produced a marked fall in blood pressure (52±4.2 mm of Hg, n=6). Though slow in onset (Onset time 4 to 5 min), the effect lasted for more than 4 hr. Pretreatment with atropine (2 mg/kg, iv) and pheniramine (10 mg/kg, iv) failed to block the hypotensive effect. The responses of nicotine and adrenaline remained
unaltered after the oil. Hypotension was associated with increase in the rate of respiration.

Rats:

Preliminary screening: The signs of central nervous system depression were observed in rats treated with essential oil in doses of 100 mg/kg and 200 mg/kg. The later dose produced marked decrease in spontaneous motor activity. The response to pain and touch stimuli was slightly reduced. There were no tremors, convulsions or cataleptic state. Gait remained normal and ptosis was not present.

Spontaneous motor activity: 100 mg and 200 mg/kg of the oil significantly decreased the coordinated motor activity of rats (Table I).

<table>
<thead>
<tr>
<th>Treatment group</th>
<th>Dose (mg/kg, ip)</th>
<th>Activity counts (Mean ± SEM)</th>
<th>Sleeping time in minutes (Mean ± SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (Vehicle only)</td>
<td>-</td>
<td>149.6 ± 4.319</td>
<td>64.9 ± 2.49</td>
</tr>
<tr>
<td>Essential oil 100</td>
<td>100</td>
<td>60.6 ± 3.66*</td>
<td>74.5 ± 2.13**</td>
</tr>
<tr>
<td>Essential oil 200</td>
<td>200</td>
<td>31.4 ± 3.30*</td>
<td>81.4 ± 1.69*</td>
</tr>
</tbody>
</table>

Values differ significantly from the control. *P < 0.001  **P < 0.01

There were 10 rats in each group.

Forced motor activity: The oil also affected the rotarod performance of the trained rats (Table III).

Barbiturate sleep: 100 mg/kg and above, dose of the oil produced a significant prolongation of the sleeping time (Table I).

Conditioned avoidance response: Table II shows that the oil was effective in blocking the CAR of trained rats without affecting the unconditioned response.

Body temperature: The oil did not significantly alter the body temperature of rats in the doses of 100 mg/kg and 200 mg/kg.
**TABLE II**: Effect of the essential oil of *Blumea membranacea* on the conditioned avoidance response (CAR) in trained rats.

<table>
<thead>
<tr>
<th>Treatment group</th>
<th>Dose in mg/kg, ip</th>
<th>No. of animals showing blockade of CAR after drug treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30 min</td>
<td>1 hr</td>
</tr>
<tr>
<td>Control (Vehicle)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Essential oil</td>
<td>100</td>
<td>4*</td>
</tr>
<tr>
<td></td>
<td>200</td>
<td>6**</td>
</tr>
<tr>
<td>Chlorpromazine</td>
<td>5</td>
<td>7</td>
</tr>
</tbody>
</table>

* n = 10. The statistical significance of difference was calculated by 'Z' test.
  
  *Z = 2.2 (Significant at P = 0.05); **Z = 2.94 (Significant at P = 0.05)*

**Isolated tissue experiments:**

*Frog's heart*: Doses from 2 to 10 mg produced negative inotropic and chronotropic effect on frog's isolated perfused heart which was slow in onset, dose related and lasted for about 3 min (n=5). Atropine (4-10 mg) failed to block this effect.

*Isolated rabbit ileum*: $1 \times 10^{-4}$ g/ml of the oil reduced markedly rhythmic movement of rabbit's isolated intestine (n=6). The spasms produced by acetylcholine ($15 \times 10^{-7}$ g/ml) and barium ($5 \times 10^{-5}$ g/ml, n=5) were promptly relieved. Prior exposure to the oil effectively prevented acetylcholine, histamine and barium chloride to stimulate the ileum (n=3).

*Guinea pig tracheal chain*: There was no significant effect on isolated tracheal chain of guineapig; however $5 \times 10^{-4}$ g/ml of the oil effectively antagonized the contractions produced by histamine ($5 \times 10^{-5}$ g/ml, n=3) and acetylcholine ($15 \times 10^{-7}$ g/ml, n=2).

*Frog rectus muscle*: 20$\times 10^{-4}$ g/ml of the oil did not exert any effect on rectus muscle. It also did not antagonise acetylcholine induced spasms (n=5).
**DISCUSSION**

Failure of atropine and pheniramine to block hypotensive effect of the oil rules out its mediation through cholinergic or histaminergic mechanisms. Further, the absence of reversal of the effect of adrenaline and nicotine suggests that the oil did not exert hypotensive action through adrenergic or ganglionic blockade. The hypotensive action could thus be due to a direct vasodilator effect, and partly, to a cardiac depressant action which was also manifest in frog heart experiments.

Smooth muscle relaxation of the ileum and complete antagonism of acetylcholine, histamine and barium chloride induced spasms indicates a direct spasmolytic and antispasmodic action.

The effect of the oil in potentiating the pentobarbitone induced hypnosis, decrease in spontaneous motor activity and blockade of CAR in rats indicates that the oil has a CNS depressant action.

The drug did not produce any hypothermia and the potentiation of pentobarbitone hypnosis seems to be the specific action of the oil and not due to hypothermia (8).

The results of present investigation suggest some interesting pharmacological effects which need to be further elaborated, preferably using fractionated components of the essential oil.
ACKNOWLEDGEMENTS

The authors express their sincere thanks to Dr. S.D. Tonpay, Lecturer Department of Pharmacology, G.R. Medical College, Gwalior for keen interest and help.

REFERENCES