Table I  Effect of sublingual nifedipine and sublingual captopril in patients with hypertensive urgencies

<table>
<thead>
<tr>
<th>Time (minutes)</th>
<th>Nifedipine group (n = 25)</th>
<th>Captopril group (n = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SBP (mean ± SD) (mmHg)</td>
<td>DBP (mean ± SD) (mmHg)</td>
</tr>
<tr>
<td>0</td>
<td>88 ± 10</td>
<td>220 ± 10</td>
</tr>
<tr>
<td>5</td>
<td>90 ± 8</td>
<td>211 ± 7</td>
</tr>
<tr>
<td>15</td>
<td>96 ± 6*</td>
<td>176 ± 14</td>
</tr>
<tr>
<td>30</td>
<td>100 ± 8*</td>
<td>164 ± 13</td>
</tr>
<tr>
<td>60</td>
<td>98 ± 5*</td>
<td>166 ± 15</td>
</tr>
<tr>
<td>120</td>
<td>96 ± 7*</td>
<td>168 ± 12</td>
</tr>
<tr>
<td>240</td>
<td>92 ± 8</td>
<td>182 ± 9</td>
</tr>
<tr>
<td>360</td>
<td>90 ± 6</td>
<td>205 ± 10</td>
</tr>
</tbody>
</table>

*P < 0.001 (comparing nifedipine and captopril groups); SBP = systolic blood pressure; DBP = diastolic blood pressure.

References

Primary anaerobic bacterial meningitis caused by Propionibacterium acnes

Sir,
The association of acute bacterial meningitis and Propionibacterium acnes is a rare event. We would like to report a case with complete recovery on chloramphenicol therapy.

A 4 year old female child presented with high grade fever associated with vomiting of 24 hours duration before admission to the hospital. Her past medical history included tuberculosis of bone (right foot) for which she had received anti-tubercular therapy for one and half years, stopped one year before the current illness. The child was fully immunized. On examination, her temperature was 102°F. She was drowsy, responding only to painful stimuli but not to verbal commands. Neck stiffness and Kernig’s signs were positive. There was no neurological deficit. Total leucocyte count was 16.8 x 10^9/l with 90% polymorphs. Lumbar puncture yielded opalescent cerebrospinal fluid (CSF) under normal pressure with 750 cells/mm^3 95% polymorphs. The glucose was 1.6 mmHg against blood glucose 5.04 mmHg and CSF protein was 108 mg/dl. No organisms were seen on Gram staining, simple methylene blue and Ziehl–Neelsen staining of the centrifuged deposit of CSF. Latex agglutination test for N. meningitidis Gr A and C. H. influenzae type b and St. pneumoniae by using ‘slide meningite kit’ (Bioierieux, France) was negative. Aerobic culture was done on chocolate and blood agar plates and incubated at 37°C for 48 hours. Supplemented brain heart infusion agar (SBHIA) and Robertson’s cooked meat (RCM) broth were used for isolation of anaerobic bacteria. These cultures were incubated at 37°C under appropriate gaseous atmosphere for 48 hours to 7 days.

Treatment was instituted by chloramphenicol in the dose of 100 mg/kg/day in four divided doses intravenously. No bacterial growth was observed in any agar plates after 48 hours except RCM which was incubated further. On Gram staining and wet mount preparation, it revealed numerous slender non-motile, non-sporing pleomorphic (diphtheroid like) Gram-positive bacilli. Anaerobic subculture of the isolate showed initially a small white-coloured colony which became larger with more or less yellow. Aerobic subculture was negative. Biochemically the isolate was consistent with P. acnes. However, gas-liquid chromatography was negative. Chloramphenicol was given orally after 5 days and administered for a duration of 14 days. Repeat CSF culture was sterile and the patient had complete clinical recovery.

P. acnes is considered to be a commensal of the normal skin, upper respiratory tract and intestinal tract. It is also a common contaminant of CSF specimens. However, when isolated from patients with signs and symptoms of central nervous system infection, the organism should not be automatically disregarded as a contaminant. It is recognized that anaerobic infections of the central nervous system including meningitis are most likely to occur in the setting of preceding chronic infections of head and neck and often part of a more extensive intracranial infection. There have also been reports of clinically significant infections related to prosthetic devices such as indwelling intravascular catheters and prosthetic heart valves. In the present case the significant past medical history was tuberculosis of bone, fully treated and asymptomatic. However, this is very unlikely to bear any correlation with the current illness. Anaerobic meningitis
is more apt to be a mono-microbial infection and less likely to be a mixed anaerobic–aerobic.

The isolation of *P. acnes* from the CSF concurrently with clinical manifestation of disease should not be routinely dismissed as a contaminant and the possibility of its underdiagnosis needs greater awareness of the possible role of anaerobes as aetiological agents in meningitis.

Chloramphenicol therapy was quite effective because of its excellent CSF penetrations. It is also regarded as one of the drugs of choice in anaerobic infections especially of central nervous system which are not yet defined bacteriologically.  

Bijay R. Mirdha
Praveen Kumar
Department of Microbiology
and Department of Paediatrics,
All India Institute of Medical Sciences,
New Delhi – 110 029, India.

**References**

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**Syncope caused by ranitidine-induced increase in A–V block**

Sir,

The H2-blockers used widely in the treatment of peptic ulcer disease have a good safety profile. However, they rarely produce serious cardiac side effects including bradycardia, asystole, ventricular and atrial arrhythmias, prolonged Q–T interval and hypotension.  

A case of syncope caused by ranitidine-induced increase in atrio-
ventricular block is described here.

An 81 year old male was admitted for prostatectomy. A routine preoperative electrocardiogram (ECG) revealed atrial flutter with ventricular rate of 50/minute. He had experienced no symptoms from atrial flutter but on systemic inquiry gave a vague history of passing dark stools. He was haemodynamically stable and the blood count, blood urea, electrolytes, glucose and thyroid stimulating hormone were all normal.

The operation was postponed and he was discharged home on ranitidine 300 mg at night in addition to his usual medication. Three months later he was admitted to another hospital with a history of fall. The diagnoses were ‘accidental fall and atrial flutter’. He was discharged home on his usual insulin, bronchodilator and 150 mg a day of ranitidine.

Five months later, he was admitted to our unit with a history of dizziness and fainting. The cardiac rate was 35/minute; blood pressure was 140/80 mmHg. The ECG showed atrial flutter and an R–R interval of 1.7 seconds. Ranitidine was suspected as a contributory cause of the bradycardia and was stopped. The ECGs done on his second admission were obtained and these showed atrial flutter with periods of markedly prolonged R–R intervals of 4.2 seconds. A careful inquiry into his symptoms on his second admission revealed that he had dizziness and syncope culminating in a fall but not an accidental fall as thought.

After stopping ranitidine the ventricular rate improved gradually, R–R interval decreased to 0.9 seconds by the 8th day and pulse rate returned to 70/minute by the 11th day. At the time of writing this paper, for 7 months after stopping ranitidine he remained symptom-free in sinus rhythm.

Markedly increased R–R interval (4.2 seconds) after instituting ranitidine therapy, improvement in ventricular rate on reduced dose and reverting to normal rhythm after its discontinuation, in this case is highly suggestive of this drug’s causal relation. The pre-operative ECG should have alerted the physicians that this patient had some sino-atrial disease. The mechanism of cardiac arrhythmias caused by H2-blockers has been discussed previously and is thought to be related to the blockade of histamine receptors in cardiac tissue or to increased prolactin release. This case illustrates the potential danger of aggravating asymptomatic cardiac arrhythmias when H2-blockers are prescribed. It is therefore advisable to keep a close watch with careful clinical examination when patients with cardiac arrhythmias are started on H2-blockers.

Macherla Radhanamanohar
Department of Medicine for the Elderly,
Medway Hospital,
Gillingham,
Kent, UK.

**References**

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B. R. Mirdha and P. Kumar

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