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MILES INTRODUCES

AN AGENT THAT IMPROVES NEUROLOGIC OUTCOME ASSOCIATED WITH VASOSPASM IN ANEURYSMAL SUBARACHNOID HEMORRHAGE
(Hunt and Hess Grades I-III Patients)

Color enhanced digital subtraction angiographic depiction of cerebral aneurysm.

New CEREBROSELECTIVE NIMOTOP nimodipine/Miles CAPSULES 30 mg

Please see references and full prescribing information on last pages of this advertisement.
IMPROVES NEUROLOGIC OUTCOME ASSOCIATED WITH VASOSPASM BY REDUCING ISCHEMIC NEUROLOGIC DEFICITS IN ANEURYSMAL SUBARACHNOID HEMORRHAGE (Hunt and Hess Grades I-III Patients)

SIGNIFICANT REDUCTION IN THE SEVERITY OF DELAYED ISCHEMIC DEFICITS ASSOCIATED WITH VASOSPASM IN GOOD GRADE PATIENTS

* As demonstrated in four double-blind, randomized clinical studies involving 823 evaluable patients

* Treatment initiated within 96 hours of the event

* Two clinical trials also included patients with Hunt and Hess Grades IV-V.

IMPROVEMENT IN NEUROLOGIC OUTCOME OCCURS REGARDLESS OF CONTINUED PRESENCE OF VASOSPASM

* "Nimodipine... has been shown to improve the outcome of the patient with a ruptured aneurysm and specifically to reduce delayed cerebral ischemic dysfunction in such a patient. However the drug does not seem to alter the appearance of arterial narrowing visualized on cerebral arteriograms and so must have its effect by some mechanism other than by alteration of arterial caliber.”

—Wilkins RH: Neurosurgery 1986

SIGNIFICANT INCREASE IN NUMBER OF PATIENTS WITH GOOD RECOVERY AND SIGNIFICANT DECREASE IN NUMBER OF PATIENTS WITH SEVERE DISABILITY IN NIMOTOP-TREATED PATIENTS VS. PLACEBO

<table>
<thead>
<tr>
<th></th>
<th>NIMOTOP</th>
<th>PLACEBO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total patients</td>
<td>278</td>
<td>276</td>
</tr>
<tr>
<td>Good recovery</td>
<td>199†</td>
<td>169</td>
</tr>
<tr>
<td>Moderate disability</td>
<td>24</td>
<td>16</td>
</tr>
<tr>
<td>Severe disability</td>
<td>12†</td>
<td>31</td>
</tr>
<tr>
<td>Death</td>
<td>43§</td>
<td>60</td>
</tr>
</tbody>
</table>

*p = 0.0444 good and moderate vs. severe and death.

*p = 0.001 severe disability.

*p = 0.056 death. Effect on mortality is not yet clear.

Dosage: 60 mg (two 30 mg capsules) every 4 hours within 96 hours of SAH and for 21 days.

*90% of patients in this study were Hunt and Hess Grades I-III.
TWO CLINICAL STUDIES DEMONSTRATE A SIGNIFICANT REDUCTION IN THE NUMBER OF PATIENTS WITH SEVERE DEFICITS

*p = 0.03
NIMOTOP pts. = 56
Placebo pts. = 60
Dose: 20-30 mg q4h for 21 days
Outcome assessed:
21 days.
*Recommended dosage is 60 mg (two 30 mg capsules) every 4 hours within 96 hours of SAH and for 21 days.

Philippon, et al study.
*p = 0.03
NIMOTOP pts. = 31
Placebo pts. = 39
Dose: 60 mg q4h for 21 days.
Outcome assessed:
21 days.

SIGNIFICANT REDUCTION IN INCIDENCE OF DELAYED ISCHEMIC DEFICITS AND PERMANENT DEFICITS DUE TO SPASM

* p = 0.001; nimodipine vs. placebo.

NIMOTOP pts. = 72
Placebo pts. = 82
Dose: 90 mg every 4 hours within 96 hours of SAH and for 21 days.

*Recommended dosage is 60 mg (two 30 mg capsules) every 4 hours within 96 hours of SAH and for 21 days.

Please see references and full prescribing information on last pages of this advertisement.
CEREBROSELECTIVE CALCIUM CHANNEL BLOCKING ACTION AND A LOW SIDE-EFFECT PROFILE

CEREBROSELECTIVE — ACTS PRIMARILY IN THE BRAIN

- Lipophilic — readily crosses the blood brain barrier
- Has special affinity for calcium channels in the cerebral vasculature
- Attaches to specific binding sites in neurons

POSTULATED MECHANISM OF ACTION

- May promote cerebral perfusion by dilating microvasculature not visible on angiography
- May protect neurons by preventing the massive calcium influx that may occur in ischemic cell damage

LOW INCIDENCE OF SIDE EFFECTS AT RECOMMENDED DOSAGE REGIMEN

- Decreased blood pressure is most common side effect, occurring in 3.8% (19/494) of patients. Carefully monitor blood pressure during therapy — see Precautions section in the prescribing information
- Other side effects occurring at a frequency of ≥1.0% include headache, nausea, and bradycardia
- No clinically significant effects on hematologic factors, renal or hepatic function, or carbohydrate metabolism have been causally associated with NIMOTOP

HEMODYNAMIC EFFECTS COMPATIBLE WITH SURGERY

- Minimal decreases in arterial blood pressure
- Minimal changes in heart rate

*For the side effects please see full prescribing information.
DOSAGE RECOMMENDATION*

- NIMOTOP therapy should begin within 96 hours of the aneurysmal subarachnoid hemorrhage regardless of angiographic evidence of vasospasm

- Recommended dosage regimen is 60 mg (two 30 mg liquid-filled capsules) every 4 hours for 21 consecutive days in Hunt and Hess Grades I-III patients

- NIMOTOP can be given orally or can be administered via nasogastric tubing as described in the prescribing information

*Please see references and full prescribing information on last pages of this advertisement.
**Clinical Trials:** Nimodipine has been shown, in 4 randomized, placebo-controlled trials, to reduce the severity of neurologic deficits resulting from vasospasm in SAH patients. The trials used doses ranging from 20-30 mg to 90 mg every 4 hours, with drug given for 21 days in 3 studies, and for at least 18 days in the other. Three of the trials studied relatively well patients, with all or most patients in Hunt and Hess Grades I-II (essentially free of local deficits after the initial bleed), the fourth studied much sicker patients, Hunt and Hess Grades III-V. Two studies, one domestic, one French, were similar in design, with relatively well patients, with all or most patients in Grades I-II. Outcomes were reported to have had lowering of the blood pressure and about 1% left the study because of this (not all could be attributed to nimodipine). Nevertheless, blood pressure should be carefully monitored during treatment with Nimotop® based on its known pharmacology and the known effects of calcium channel blockers.

**Precautions:**
- **General:** Blood Pressure: Nimodipine has the hemodynamic effects expected of a calcium channel blocker, although they are generally not marked. In patients with subarachnoid hemorrhage patients, 5% were reported to have had lowering of the blood pressure and about 1% left the study because of this (not all could be attributed to nimodipine). Nevertheless, blood pressure should be carefully monitored during treatment with Nimotop® based on its known pharmacology and the known effects of calcium channel blockers.
- **Hepatic Disease:** The metabolism of Nimotop® is decreased in patients with hepatic disease, with Cmax approximately double that in normals which necessitates lowering the dose in this group of patients (see Dosage and Administration). The bioavailability of nimodipine averages 13% after oral administration. The bioavailability is significantly increased in patients with hepatic disease, with Cmax approximately double that in normals which necessitates lowering the dose in this group of patients (see Dosage and Administration).

**Clinical Pharmacology:**
- **Mechanism of Action:** Nimodipine is a calcium channel blocker. The mechanisms of actions of these arteries are dependent upon calcium ions, which enter these cells during depolarization as slow ionic transmembrane currents. Nimodipine inhibits calcium ion transfer into these cells and thus inhibits contractions of vascular smooth muscle. In animal experiments, nimodipine had a greater effect on cerebral arteries than on arteries elsewhere in the body perhaps because it is highly lipophilic, allowing it to cross the blood-brain barrier. Concentrations of nimodipine as high as 12.5 ng/mL have been detected in the cerebrospinal fluid of nimodipine treated subarachnoid hemorrhage (SAH) patients.
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not statistically significant; however, the higher rates were well within historical control range for these tumors in the Wistar strain. Nimodipine was found not to be carcinogenic in a 91-week mouse study but the high dose of 1800 rpm nimodipine to feed (1540 to 774 mg/kg/day) shortened the life expectancy of the animals. Mutagenic studies, including the Ames, micronucleus and dominant lethal tests were negative.

Nimodipine did not impair the fertility and general reproductive performance of male and female Wistar rats following oral doses of up to 30 mg/kg/day when administered daily for more than 10 weeks in the males and 3 weeks in the females prior to mating and continued to day 7 of pregnancy. This dose in a rat is about 4 times the equivalent clinical dose of 60 mg 4th in a 50 kg patient.

**Pregnancy:** Pregnancy Category C. Nimodipine has shown to have a teratogenic effect in Hamalayan rabbits. Incidences of malformations and stunned fetuses were increased at oral (by gavage) doses of 1 and 10 mg/kg/day administered from day 6 through day 18 of pregnancy but not at 3.0 mg/kg/day in one of two identical rabbit studies. In the second study an increased incidence of stunned fetuses was seen at 1.0 mg/kg/day but not at higher doses. Nimodipine was embryotoxic, causing resorption and stunned growth of fetuses, in Long Evans rats at 100 mg/kg/day administered by gavage from day 6 through day 15 of pregnancy. In two other rat studies, doses of 30 mg/kg/day nimodipine administered by gavage from day 16 to gestation and continued until sacrifice (day 20 of pregnancy or day 21 post partum) were associated with higher incidences of skeletal variation, stunned fetuses and stillbirths but not malformations. There are no adequate and well controlled studies in pregnant women to directly assess the effect on human fetuses. Nimodipine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

**Nursing Mothers:** Nimodipine and/or its metabolites have been shown to appear in rat milk at concentrations much higher than in maternal plasma. It is not known whether the drug is excreted in human milk. Because many drugs are excreted in human milk, nursing mothers are advised not to breast feed their babies when taking the drug.

**Pediatric Use:** Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS**

Adverse experiences were reported by 92 of 823 patients with subarachnoid hemorrhage (11.2%) who were given nimodipine. The most frequently reported adverse experience was decreased blood pressure in 4.4% of these patients. Twenty-nine of 479 (6.1%) placebo treated patients also reported adverse experiences. The events reported with a frequency greater than 1% are displayed below by dose.

**DOSE q4h Number of Patients (%)**

<table>
<thead>
<tr>
<th>Sign/Symptom</th>
<th>Nimodipine</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Pressure</td>
<td>0.35 mg/kg</td>
<td>30 mg</td>
</tr>
<tr>
<td>Abnormal Liver Function Test</td>
<td>1 (1.2)</td>
<td>0</td>
</tr>
<tr>
<td>Edema</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Rash</td>
<td>2 (2.4)</td>
<td>0</td>
</tr>
<tr>
<td>Headache</td>
<td>1 (1.4)</td>
<td>6 (1.2)</td>
</tr>
<tr>
<td>Gastrointestinal Symptoms</td>
<td>2 (2.4)</td>
<td>0</td>
</tr>
<tr>
<td>Nausea</td>
<td>1 (1.2)</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Diaphoresis</td>
<td>1 (1.2)</td>
<td>0</td>
</tr>
<tr>
<td>EKG</td>
<td>0</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Abnormalities</td>
<td>0</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Muscle Pain/CRamp</td>
<td>0</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Acne</td>
<td>0</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Depression</td>
<td>0</td>
<td>1 (1.4)</td>
</tr>
</tbody>
</table>

There were no other adverse experiences reported by the patients who were given 0.35 mg/kg q4h, 30 mg q4h or 120 mg q4h. Adverse experiences with an incidence rate of less than 1% in the 60 mg q4h dose group were: hepatitis; itching; gastrointestinal hemorrhage; thromboctopenia; anemia; palpitations; vomiting; flushing; dysphonia; wheezing; gastrointestinal toxemia; lightheadedness; dizziness; rebound vasoconstriction; jaundice; hypertension; hematomas.

Adverse experiences with an incidence rate less than 1% in the 90 mg q4h dose group were: itching; gastrointestinal hemorrhage; thromboctopenia; neurological disturbances; vomiting; diaphoresis; congestive heart failure; hypotension; decreasing platelet count; disseminated intravascular coagulation; deep vein thrombosis.

As can be seen from the table, side effects that appear related to nimodipine use based on increased incidence with higher dose or a higher rate compared to placebo control, included decreased blood pressure, edema and headaches which are known pharmacologic actions of calcium channel blockers. It must be noted, however, that SAH is frequently accompanied by alterations in consciousness which lead to an under reporting of adverse experiences. Patients who received nimodipine in clinical trials for other indications reported flushing (2.1%), headache (4.1%) and fluid retention (0.3%), typical responses to calcium channel blockers. As a calcium channel blocker, nimodipine may have the potential to exacerbate heart failure in susceptible patients or to interfere with AV-conduction, but these events were not observed.

Nimodipine and/or its metabolites have been shown to appear in human milk. Because many drugs are excreted in human milk, nimodipine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. This oral dose is 60 mg (two 30 mg capsules) every 4 hours for 21 consecutive days. Oral Nimotop® therapy should commence within 96 hours of the subarachnoid hemorrhage. If the capsule cannot be swallowed, e.g., at the time of surgery, or if the patient is unconscious, a hole should be made in both ends of the capsule with an 18 gauge needle, and the contents of the capsule extracted into a syringe. The contents should then be emptied into the patient's naso-gastric tube and washed down the tube with 30 mL of normal saline (0.9%).

Patients with hepatic failure have substantially reduced clearance and approximately doubled Cmax. Dosage should be reduced to 30 mg every 4 hours, with close monitoring of blood pressure and heart rate.

**Htow Supplied**

Each ivory colored, soft gelatin Nimotop® capsule is imprinted with the word Miles and the number 855 and contains 30 mg of nimodipine. The 30 mg capsules are packaged in unit dose foil pouches and supplied in cartons containing 100 capsules. The capsules should be stored in the manufacturer's original foil package at a controlled room temperature of 95°F to 130°F (35°C to 30°C).
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