

## VICON FORTE®

### Therapeutic vitamin-minerals

**DESCRIPTION:** Each black and orange Vicon Forte® capsule for oral administration contains:

Vitamin A	8000 I.U.
Vitamin E	50 I.U.
Ascorbic Acid	150 mg.
Zinc Sulfate, USP*	80 mg.
Magnesium Sulfate, USP**	70 mg.
Niacinamide	25 mg.
Thiamine Mononitrate	10 mg.
d-Calcium Pantothenate	10 mg.
Riboflavin	5 mg.
Manganese Chloride	4 mg.
Pyridoxine Hydrochloride	2 mg.
Folic Acid	1 mg.
Vitamin B <sub>12</sub> (Cyanocobalamin)	10 mcg.

\*As 50 mg of dried zinc sulfate

\*\*As 50 mg of dried magnesium sulfate

VICON FORTE® is a therapeutic vitamin-mineral preparation.

**INDICATIONS AND USAGE:** VICON FORTE® is indicated for the treatment and/or prevention of vitamin and mineral deficiencies associated with restricted diets, improper food intake, alcoholism and decreased absorption. VICON FORTE® is also indicated in patients with increased requirements for vitamins and minerals due to chronic disease, infection, and burns and in persons using alcohol to excess. Pre- and post-operative use of VICON FORTE® can provide the increased amounts of vitamins and minerals necessary for optimal recovery from the stress of surgery.

**CONTRAINDICATIONS:** None known.

**PRECAUTIONS:** General—Folic acid in doses above 0.1 mg daily may obscure pernicious anemia in that hematologic remission can occur while neurological manifestations remain progressive.

**DOSAGE AND ADMINISTRATION:** One capsule daily or as directed by the physician.

**HOW SUPPLIED:** Capsules, orange and black imprinted with "Glaxo" and "316" in bottles of 60 (NDC 0173-0316-22) and 500 (NDC 0173-0316-24) capsules each and in unit dose packs of 100 (NDC 0173-0316-27) capsules.

Dispense in tight, light-resistant containers as defined in the National Formulary.

For your patients' other vitamin needs, recommend **VICON-C** the original multivitamin with zinc.

#### VICON-C® Capsules

(Therapeutic Vitamins and Minerals)

**Description:** Each yellow and orange capsule contains:

Ascorbic Acid	300 mg
Niacinamide	100 mg
Thiamine Mononitrate	20 mg
d-Calcium Pantothenate	20 mg
Riboflavin	10 mg
Pyridoxine Hydrochloride	5 mg
Magnesium Sulfate, USP*	70 mg
Zinc Sulfate, USP**	80 mg

\*As 50 mg of dried Magnesium Sulfate

\*\*As 50 mg of dried Zinc Sulfate

## Letters to the Editor



The Journal welcomes Letters to the Editor; if found suitable, they will be published as space allows. Letters should be typed double-spaced, should not exceed 400 words, and are subject to abridgment and other editorial changes in accordance with journal style.

### Vasectomy

To the Editor:

I wish to comment about the article entitled "Vasectomy" (*Brownlee HJ, Tibbels CK: Vasectomy. J Fam Pract 16:379, 1983*), which appeared in your February 1983 issue.

In addition to its use in family planning, vasectomy is of established value in the prevention of recurrent epididymitis, since this infection ascends the vas from the prostatic urethra.

In the article the authors discuss preoperative counseling at length, and I completely endorse this. They mention using a booklet, which is a most important tool. I send one to the patient prior to the interview. It explains the procedure and its complications, and gives further information about what to expect. It has one highly important feature that I recommend to everyone who performs vasectomies: The last page of the booklet consists of a consent form, which is to be signed by the patient and his wife. The booklet then becomes part of the patient's records. The following points are stressed: (1) that the patient and wife *request* the vasectomy, (2) that the operation is intended to produce permanent sterility, (3) that they have read the booklet and fully under-

stand the points therein and that all of their questions have been satisfactorily answered, and (4) that they agree to submit postoperative semen specimens for testing and will continue to use birth control methods until told that it is safe to discontinue. The legal profession continues to attempt to make physicians guarantee vasectomies, to pay for raising children born after failed vasectomies, and so on. This consent form affords protection against such suits.

The authors also discuss preoperative medication but fail to stress the necessity of then having the patient bring someone to drive him home after the procedure. A sedated patient could be a dangerous driver. I have never used preoperative medication, relying instead upon "verbal analgesia" and a gentle touch to relax the patient.

Brownlee and Tibbels misunderstand when they state that I advocate "excision of a small specimen for pathologic identification purposes."<sup>1</sup> I do not remove any of the vas, since it is of no value in guaranteeing success of the procedure. Rather, I stated that if the surgeon feels that he *must* do so, the segment should be the shortest possible. The vas deferens has a totally

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Manufactured for:

**Glaxo**

Glaxo Inc., Research Triangle Park, NC 27709

Announcing newly formulated

# Equagesic<sup>®</sup>

(meprobamate with aspirin) © Wyeth

(BRIEF SUMMARY)

**DESCRIPTION:** Each tablet contains 200 mg meprobamate and 325 mg aspirin.

**INDICATIONS:** Adjuvant in short-term treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease. Clinical trials demonstrated that in these situations relief of pain is somewhat greater than with aspirin alone. Effectiveness in long-term use, i.e. over 4 months, has not been assessed by systematic clinical studies. Physicians should periodically reassess usefulness of drug for individual patients.

**CONTRAINDICATIONS:** **ASPIRIN:** Allergic or idiosyncratic reactions to aspirin or related compounds. **MEPROBAMATE:** Acute intermittent porphyria, allergic or idiosyncratic reactions to meprobamate or related compounds, e.g. carisoprodol, meprobamate, or carbromal.

**WARNINGS:** **ASPIRIN:** Use salicylates with extreme caution in patients with peptic ulcer, asthma, coagulation abnormalities, hypoprothrombemia, vitamin K deficiency, or those on anticoagulants. In rare instances, aspirin in persons allergic to salicylates may result in life-threatening allergic episodes.

**MEPROBAMATE DRUG DEPENDENCE:** Physical and psychological dependence, and abuse have occurred. Chronic intoxication from prolonged ingestion of, usually, greater than recommended doses is manifested by ataxia, slurred speech, and vertigo. Therefore, carefully supervise dose and amounts prescribed and avoid prolonged use, especially in alcoholics and others with known propensity for taking excessive quantities of drugs. Sudden withdrawal after prolonged and excessive use may precipitate recurrence of preexisting symptoms, e.g. anxiety, anorexia, or insomnia, or withdrawal reactions, e.g. vomiting, ataxia, tremors, muscle twitching, confusional states, hallucinations, and rarely, convulsive seizures. Such seizures are more likely in persons with CNS damage or preexistent or latent convulsive disorders. Onset of withdrawal symptoms occurs usually within 12 to 48 hours after discontinuation, symptoms usually cease within next 12- to 48-hour period. When excessive dosage has continued for weeks or months, reduce dosage gradually over 1 to 2 weeks rather than stop abruptly. Alternatively, a short-acting barbiturate may be substituted, then gradually withdrawn.

**POTENTIALLY HAZARDOUS TASKS:** Warn patients meprobamate may impair mental or physical abilities required for potentially hazardous tasks, e.g. driving or operating machinery.

**ADDITIVE EFFECTS:** Since CNS-suppressant effects of meprobamate and alcohol or meprobamate and other psychotropic drugs may be additive, exercise caution with patients taking more than one of these agents simultaneously.

**USAGE IN PREGNANCY AND LACTATION:** An increased risk of congenital malformations associated with minor tranquilizers (meprobamate, chlorthalidone, and diazepam) during first trimester of pregnancy, has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at time of institution of therapy should be considered. Advise patients if they become pregnant during therapy or intend to become pregnant to communicate with their physicians about desirability of discontinuing the drug.

**Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breastfeeding patients, consider the drug's higher concentrations in breast milk as compared to maternal plasma levels.**

**USAGE IN CHILDREN:** Keep preparations with aspirin out of reach of children. Equagesic<sup>®</sup> (meprobamate with aspirin) is not recommended for patients 12 years of age and under.

**PRECAUTIONS:** **ASPIRIN:** Salicylates antagonize uricosuric activity of probenecid and sulfipyrazone. Salicylates are reported to enhance hypoglycemic effect of sulfonylurea antidiabetics.

**MEPROBAMATE:** Use lowest effective dose, particularly in elderly and/or debilitated, to preclude over-sedation. Meprobamate is metabolized in the liver and excreted by the kidney. To avoid excess accumulation exercise caution in its use in patients with compromised liver or kidney function. Meprobamate occasionally may precipitate seizures in epileptic patients. It should be prescribed cautiously and in small quantities to patients with suicidal tendencies.

**ADVERSE REACTIONS:** **ASPIRIN:** May cause epigastric discomfort, nausea, and vomiting. Hypersensitivity reactions, including urticaria, angioneurotic edema, purpura, asthma, and anaphylaxis may rarely occur. Patients receiving large doses of salicylates may develop tinnitus.

**MEPROBAMATE CNS:** Drowsiness, ataxia, dizziness, slurred speech, headache, vertigo, weakness, paresthesias, impairment of visual accommodation, euphoria, overstimulation, paradoxical excitement, fast EEG activity.

GI: Nausea, vomiting, diarrhea.

**CARDIOVASCULAR:** Palpitation, tachycardia, various forms of arrhythmia, transient ECG changes, syncope, hypotensive crisis.

**ALLERGIC OR IDIOSYNCRATIC:** Milder reactions are characterized by itchy, urticarial, or erythematous maculopapular rash, generalized or confined to the groin. Other reactions include leukopenia, acute nonthrombocytopenic purpura, petechiae, ecchymoses, exanthema, peripheral edema, adenopathy, fever, fixed drug eruption, with cross-reaction to carisoprodol, and cross-sensitivity between meprobamate-mebutamate and meprobamate-carbromal. Rare, more severe hypersensitivity reactions include hyperpyrexia, chills, angioneurotic edema, bronchospasm, oliguria, and anuria. Also, anaphylaxis, exfoliative dermatitis, stomatitis and proctitis, Stevens-Johnson syndrome and bullous dermatitis have occurred.

**HEMATOLOGIC (SEE ALSO "ALLERGIC OR IDIOSYNCRATIC"):** Agnuculothymic aplasia, anemia have been reported, although no causal relationship has been established and thrombocytopenic purpura.

**OTHER:** Exacerbation of porphyric symptoms.

**DOSSAGE AND ADMINISTRATION:** Usual dose is one or two tablets, 3 to 4 times daily as needed for relief of pain when tension or anxiety is present. Not recommended for patients 12 years of age and under.

**OVERDOSAGE:** Treatment is essentially symptomatic and supportive. Any drug remaining in the stomach should be removed. Induction of vomiting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both aspirin and meprobamate. Aspirin overdosage produces usual symptoms and signs of salicylate intoxication. Observation and treatment should include management of hyperthermia, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombemia which, if it occurs, usually requires whole-blood transfusions. Suicidal attempts with meprobamate have resulted in drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse. Some suicidal attempts have been fatal. The following data, reported in the literature and from other sources, are not expected to correlate with each case (consider also individual susceptibility and length of time from ingestion to treatment), but represent usual ranges reported. Acute simple overdose (meprobamate alone). Death has been reported with ingestion of as little as 12 grams meprobamate and survival with as much as 40 grams.

**BLOOD LEVELS:** 0.5-2.0 mg percent represents usual blood-level range of meprobamate after therapeutic doses. The level may occasionally be as high as 3.0 mg percent. 3-10 mg percent usually corresponds to findings of mild-to-moderate symptoms of overdosage, such as stupor or light coma. 10-20 mg percent usually corresponds to deeper coma, requiring more intensive treatment. Some fatalities occur.

At levels greater than 20 mg percent, more fatalities than survivals can be expected. Acute combined overdose (meprobamate with other psychotropic drugs or alcohol). Since effects can be additive, history of ingestion of a low dose of meprobamate plus any of these compounds (or of a relatively low blood or tissue level) cannot be used as a prognostic indicator.

In cases of excessive doses, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Any drug remaining in stomach should be removed and symptomatic treatment given. Should respiration or blood pressure become compromised, respiratory assistance, CNS stimulants, and pressor agents should be administered cautiously as indicated. Diuresis, osmotic (mannitol) diuresis, peritoneal dialysis, and hemodialysis have been used successfully in removing both aspirin and meprobamate. Alkalinization of the urine increases excretion of salicylates. Careful monitoring of urinary output is necessary, and caution should be taken to avoid overhydration. Relapse and death, after initial recovery, have been attributed to incomplete gastric emptying and delayed absorption.

**HOW SUPPLIED:** Scored tablets, bottles of 100. Redipak<sup>®</sup> strip pack 25's; Redipak<sup>®</sup> unit dose 100's, individually wrapped.

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## LETTERS TO THE EDITOR

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characteristic feel. There is no other white, thick-walled scrotal structure whose lumen does not bleed. Proof of satisfactory surgery lies in the disappearance of sperm postoperatively and not in the trophy sent to the pathologist, or preserved in a bottle in the back room of the office. I have never had a failure using the technique the authors cite, but without excising any of the vas.

The simplest postoperative test is to set a goal of no sperm in the postoperative semen specimen. This permits examination of condom specimens and of specimens that must be transported long distances for analysis. Also, since the reservoirs of sperm may empty only to have a spontaneous anastomosis of the vas follow, I recommend that two negative samples be secured, with a one-month interval, before the patient is told that he is sterile.

It is unnecessary to excise a painful spermatic granuloma of the vas, and indeed this is a difficult task. If the granuloma is cystic it should be evacuated. The vas should then be transected and sealed proximal to the granuloma. Once the flow of sperm into the granuloma ceases, the granuloma will disappear.

The authors discuss postoperative epididymitis. I have never seen postvasectomy bacterial epididymitis. What is seen is an engorgement of the epididymis with spermatozoa, a temporary condition that subsides spontaneously, or a spermatic granuloma of the epididymis wherein the swelling is limited to one portion of the epididymis and subsides slowly. Antibacterials are without value in these conditions.

Having extensive experience in vasectomy reversal, I feel that its increased incidence (now 1 percent of my vasectomy patients) is due

not to a failure of counseling, but rather to the public's awareness that reversals can be performed and that they often succeed. Most reversals are done during a subsequent marriage, years after the vasectomy, a matter that counseling might not have prevented.

Stanwood S. Schmidt, MD  
Research Associate in Urology  
University of California  
Medical School  
San Francisco, California

## Reference

1. Schmidt SS: Technique of vasectomy. Br Med J 2:524, 1974

## Screening for Gestational Diabetes

To the Editor:

I would like to take issue with a recommendation by Swinker in her recent paper in this journal, "Routine Screening for Gestational Diabetes in a Family Practice Center."<sup>1</sup> I take exception to the statement, "routine testing for carbohydrate intolerance in pregnancy is recommended for all patients because of the increased fetal morbidity and mortality in women with gestational diabetes. . . ." This is not consistent with current literature. Swinker cites the 1973 article by O'Sullivan et al<sup>2</sup> to support a 6.4 percent perinatal mortality in gestational diabetes compared with 1.5 percent in controls. However, this group also showed no increased fetal wastage in gestational diabetics less than 25 years of age. Swinker did not refer to the 1977 article by Gabbe et al,<sup>3</sup> which did not show increased fetal mortality with gestational diabetes. They did not support the finding of O'Sullivan et al that patients aged over 25 years are at greatest risk and therefore did not even support evaluating all patients over 25 years of age. Gabbe

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et al did suggest that screening be done after the clinician seeks out certain historical and clinical clues including previous stillbirth, macrosomic infant, family history of diabetes, obesity, hypertension, and glycosuria.

The experience of Swinker does not prove the value of routine screening. It is unclear whether the three women found to have gestational diabetes were truly helped by the discovery. It is also unclear whether they would not also have been found by a routine prenatal urinalysis showing glycosuria. To spend resources to discover a condition that may not truly be associated with increased perinatal mortality and for which outcomes may not be improved as a result seems totally unjustified.

Until a study comparing outcomes in populations screened vs not screened has been undertaken, it is premature to recommend universal screening. The data are not convincing that one should change the approach of selective screening based on historical and clinical factors.

*Sam C. Eggertsen, MD*

*Department of Family Medicine  
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#### References

1. Swinker M: Routine screening for gestational diabetes mellitus in a family practice center. *J Fam Pract* 17:611, 1983
2. O'Sullivan JB, Charles D, Mahan CM, Dandrow R: Gestational diabetes and perinatal mortality rate. *Am J Obstet Gynecol* 116:901, 1973
3. Gabbe SG, Mestman JH, Freeman RK: Management and outcome of class A diabetes mellitus. *Am J Obstet Gynecol* 127:465, 1977

*The preceding letter was referred to Dr. Swinker, who responds as follows:*

In response to Dr. Eggertsen's letter, I would like to refer to

Gabbe's 1978 article<sup>1</sup> in which he states, "Recent investigations would indicate that all patients should be screened no later than 28-30 weeks' gestation. In the past, screening was begun with a search for historical and/or clinical clues." To justify this position, Gabbe cites O'Sullivan's 1973 article<sup>2</sup> in which it was found that "over 50% of patients who subsequently developed diabetes in pregnancy failed to show the historical and clinical associations described above."

There is some disagreement in the literature about whether gestational-diabetic women have a higher rate of perinatal mortality. For example, Mestman,<sup>3</sup> Gabbe's colleague from Los Angeles, does not feel there is an increase in perinatal mortality in gestational diabetics who do not develop fasting hyperglycemia. Nevertheless, he advocates universal screening in all age groups. He notes that although the incidence of positive screening tests may be higher in patients with a positive history or increasing age, a significant number of patients will have abnormal tests without any historical medical clues, even in the younger age groups.

Thus, the rationale for making the diagnosis of gestational diabetes need not be based on increased mortality. The gestational diabetic is managed to avert hyperglycemia, and monitored for the development of fasting hyperglycemia (which would place the pregnancy in the risk category of an overt diabetic with potential for increased perinatal mortality). There is no question that infants of gestational diabetics experience increased perinatal morbidity, eg, hypoglycemia, hyperbilirubinemia. Awareness of possible neonatal problems may aid the physician so that hypoglycemia, for example, may be promptly recognized and treated.

*Marian Swinker, MD*

*University Health Service  
West Virginia University  
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#### References

1. Gabbe S: Application of scientific rationale to the management of the pregnant diabetic. *Semin Perinatol* 2:361, 1978
2. O'Sullivan J, Mahan C, Charles D, Dandrow R: Screening criteria for high risk gestational diabetic patients. *Am J Obstet Gynecol* 116:895, 1973
3. Mestman J: Outcome of diabetes screening in pregnancy and perinatal morbidity in infants of mothers with mild impairment in glucose tolerance. *Diabetes Care* 3:447, 1980

### Insulin-Pump Therapy

To the Editor:

Recently, the use of insulin-pump therapy to improve diabetic control has become more popular, exemplified in recent reports from the Mason Clinic,<sup>1</sup> where experience with 100 patients on insulin pumps has been described. All of these patients were begun on insulin-pump therapy with a period of inpatient instruction.

Over the past year we have had the opportunity to begin 16 patients on insulin-pump therapy on an outpatient basis. All 16 patients had brittle diabetes and all had inadequate control with intensive diabetic treatment and home glucose monitoring.

All patients were first seen in a glucose-monitoring program two times a week in groups supervised by a nurse practitioner and an endocrinologist. The patients were initially instructed in nutrition and CHEMSTRIP use, and they kept daily graphs of their activity, insulin dosage, carbohydrate intake, insulin administration, and blood glucose by CHEMSTRIP seven times a day. All 16 patients had brittle diabetes (15 type I, 1 type II), unable to be controlled satisfactorily on this program and often involving

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multiple doses of insulin; therefore, they were begun on insulin-pump therapy.

The patients were introduced to the use of an insulin pump during an office visit with a nurse practitioner in a 40-minute session. The mechanics of the pump were carefully explained, and the patient performed all procedures including mixing of insulins (for older model pumps) under the supervision of the nurse practitioner before going home. Insulin dosage was generally reduced by approximately 30 percent from the total dose taken by injection, and morning and overnight basal rates and premeal bolus amounts were determined. Possible problems with catheters, batteries, tape allergies, and so on, were discussed. Patients were given the telephone numbers of other patients on the pump, and 24-hour telephone access to a nurse practitioner or physician by beeper was available. These patients continued to be seen in biweekly home glucose-monitoring group sessions where serial clinical observations were made of the patient's course. In addition, an anonymous questionnaire was given to each patient to assess satisfaction with this outpatient program. Of interest is that some of these patients have been instructed on more than one type of pump by this method, and one patient has used successively the Millhill, Autosyringe, and CPI pumps, with each instruction occurring by the above method.

All patients rapidly achieved a high level of understanding and competence with the insulin pump. There were no untoward mishaps during the initial phase of insulin-pump therapy. After several patients were wearing a pump, they became a resource of information and support for additional patients being initiated in insulin-pump

therapy during home glucose-monitoring group sessions. A number of patients (8 of 16) experienced initial apprehension upon taking the pump home, a problem that could have been eliminated by inpatient instruction. One patient specifically stated that 24 to 48 hours of inpatient instruction should be recommended.

Insulin pump therapy is being used with increasing frequency. One obstacle to such therapy is its expense. By initiating therapy on an outpatient basis with frequent peer-group follow-up visits, we have attempted to reduce the cost of initiating such therapy in 16 patients.

Of all our brittle diabetics, with excellent knowledge of home glucose monitoring, 50 percent felt satisfied with this approach. The other 50 percent experienced anxiety when confronted with the need to initiate pump therapy at home. There were no untoward initial problems experienced by these patients aside from their anxiety.

It is our conclusion that insulin-pump therapy can be safely begun on an outpatient basis, but that it is associated with some patient anxiety. A possible future approach to alleviate that anxiety while continuing to attempt outpatient initiation of therapy might be to see each patient on several successive days before or after the patient begins using the pump.

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#### Reference

1. Meckelburg RS, Benson JW Jr, Becker NM, et al: Clinical use of the insulin infusion pump in 100 patients with type I diabetes. *N Engl J Med* 307:513, 1982

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**Nursing Mothers:** Captopril is secreted in human milk. Exercise caution when administering captopril to a nursing woman, and, in general, nursing should be interrupted.

**Pediatric Use:** Safety and effectiveness in children have not been established although there is limited experience with use of captopril in children from 2 months to 15 years of age. Dosage, on a weight basis, was comparable to that used in adults. Captopril should be used in children only if other measures for controlling blood pressure have not been effective.

**ADVERSE REACTIONS:** Reported incidences are based on clinical trials involving about 4000 patients.

**Renal**—One to 2 of 100 patients developed proteinuria (see WARNINGS). Renal insufficiency, renal failure, polyuria, oliguria, and urinary frequency in 1 to 2 of 1000 patients.

**Hematologic**—Neutropenia/agranulocytosis occurred in about 0.3% of captopril treated patients (see WARNINGS). Two of these patients developed sepsis and died.

**Dermatologic**—Rash (usually mild, maculopapular, rarely urticarial), often with pruritus and sometimes with fever and eosinophilia, in about 10 of 100 patients, usually during the 1st 4 weeks of therapy. Pruritus, without rash, in about 2 of 100 patients. A reversible associated pemphigoid-like lesion, and photosensitivity have also been reported. Angioedema of the face, mucous membranes of the mouth, or of the extremities in about 1 of 100 patients—reversible on discontinuance of captopril therapy. One case of laryngeal edema reported. Flushing or pallor in 2 to 5 of 1000 patients.

**Cardiovascular**—Hypotension in about 2 of 100 patients. See WARNINGS (Hypotension) and PRECAUTIONS (Drug Interactions) for discussion of hypotension on initiation of captopril therapy. Tachycardia, chest pain, and palpitations each in about 1 of 100 patients. Angina pectoris, myocardial infarction, Raynaud's syndrome, and congestive heart failure each in 2 to 3 of 1000 patients.

**Dysgeusia**—About 7 of 100 patients developed a diminution or loss of taste perception; taste impairment is reversible and usually self-limited even with continued drug use (2 to 3 months). Gastric irritation, abdominal pain, nausea, vomiting, diarrhea, anorexia, constipation, aphthous ulcers, peptic ulcer, dizziness, headache, malaise, fatigue, insomnia, dry mouth, dyspnea, and paresthesias reported in about 0.5 to 2% of patients but did not appear at increased frequency compared to placebo or other treatments used in controlled trials.

**Altered Laboratory Findings:** Elevations of liver enzymes in a few patients although no causal relationship has been established. Rarely cholestatic jaundice and hepatocellular injury with secondary cholestasis have been reported. A transient elevation of BUN and serum creatinine may occur, especially in volume-depleted or renovascular hypertensive patients. In instances of rapid reduction of longstanding or severely elevated blood pressure, the glomerular filtration rate may decrease transiently, also resulting in transient rises in serum creatinine and BUN. Small increases in serum potassium concentration frequently occur, especially in patients with renal impairment (see PRECAUTIONS).

**OVERDOSAGE:** Primary concern in correction of hypotension. Volume expansion with an I.V. infusion of normal saline is the treatment of choice for restoration of blood pressure. Captopril may be removed from the general circulation by hemodialysis.

**DOSAGE AND ADMINISTRATION:** CAPOTEN should be taken one hour before meals. Dosage must be individualized; see DOSAGE AND ADMINISTRATION section of package insert for detailed information regarding dosage in hypertension and in heart failure. Because CAPOTEN (captopril) is excreted primarily by the kidneys, dosage adjustments are recommended for patients with impaired renal function. **Consult package insert before prescribing CAPOTEN (captopril).**

**HOW SUPPLIED:** Available in tablets of 25, 50, and 100 mg in bottles of 100, and in UNIMATIC® unit-dose packs of 100 tablets.



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