

Usefulness of three additional electrocardiographic chest leads (V_7 , V_8 and V_9) in the diagnosis of acute myocardial infarction

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Additional electrocardiographic chest leads (V_7 , V_8 and V_9) were used in 117 persons consecutively admitted to a coronary care unit. Among the 46 (39%) with a proven acute myocardial infarction the electrocardiograms (ECGs) of 9 (20%) showed ST-segment elevation or abnormal Q-waves, or both, in the three additional leads. In six of the nine, such changes were associated with signs of anterolateral or inferior wall infarction (in three each) on the standard 12-lead ECG, but in the other three (7% of the 46) electrocardiographic changes diagnostic of acute myocardial infarction were found only on the additional chest leads; the last three had characteristic changes in serum enzyme concentrations.

This study showed that additional chest leads are helpful in detecting myocardial injury or necrosis in areas of the heart not properly reflected on the standard 12-lead ECG.

Des dérivations électrocardiographiques thoraciques additionnelles (V_7 , V_8 et V_9) ont été utilisées chez 117 personnes hospitalisées consécutivement dans une unité de soins coronariens. Parmi les 46 (39%) ayant un infarctus aigu du myocarde reconnu les électrocardiogrammes (ECG) de 9 (20%) ont montré une élévation du segment ST ou des ondes Q anormales, ou les deux, dans les trois dérivations additionnelles. Chez six des neuf ces changements ont été associés à des signes d'infarctus antérolatéral ou d'infarctus de la paroi inférieure (dans trois cas chacun) sur l'ECG standard à 12 dérivations, mais dans les trois autres (7% des 46) les changements électrocardiographiques de l'infarctus aigu du myocarde n'ont été retrouvés que sur les dérivations thoraciques additionnelles; les trois derniers présentaient les changements caractéristiques des concentrations enzymatiques du sérum.

Cette étude a démontré que des dérivations thoraciques additionnelles sont utiles pour détecter une lésion

ou une nécrose myocardique survenant dans des régions du coeur qui ne sont pas bien représentées sur l'ECG standard à 12 dérivations.

The introduction of the unipolar lead concept by Wilson, Johnston and Rosenbaum¹ and its application in classic electrocardiography with six precordial leads^{1,2} proved to be major steps in the development of clinical electrocardiography.³ Although the concept has been disputed over the years, the practical use of unipolar lead recordings at various sites on the body surface has gained wide acceptability. More recently this concept has been applied in precordial mapping techniques that attempt quantification of myocardial injury or necrosis.^{4,5}

Experience has shown that each of the six classic chest leads (V_1 to V_6) provides useful information for detecting and localizing myocardial injury or necrosis affecting the interventricular septum or the anterolateral wall of the heart, or both. On the basis of the postulates that under-

lie the use of these leads, one may speculate whether recordings from chest leads in positions V_7 , V_8 and V_9 might not yield diagnostic information in patients suspected of having an acute myocardial infarction, particularly if the affected area was the far lateral portion of the left ventricle (Fig. 1). The purpose of the study described below was to determine the usefulness of these additional leads as part of the routine electrocardiographic examination of patients admitted to a coronary care unit.

Methods

Included in this study were 117 persons admitted consecutively to the coronary care unit of Victoria Hospital, London, Ont. A 15-lead electrocardiogram (ECG) was obtained, either in the emergency room or at the time of arrival at the coronary care unit. For the three additional leads the electrode was placed over the posterior axillary line (V_7), over the midscapular line (V_8) and half-

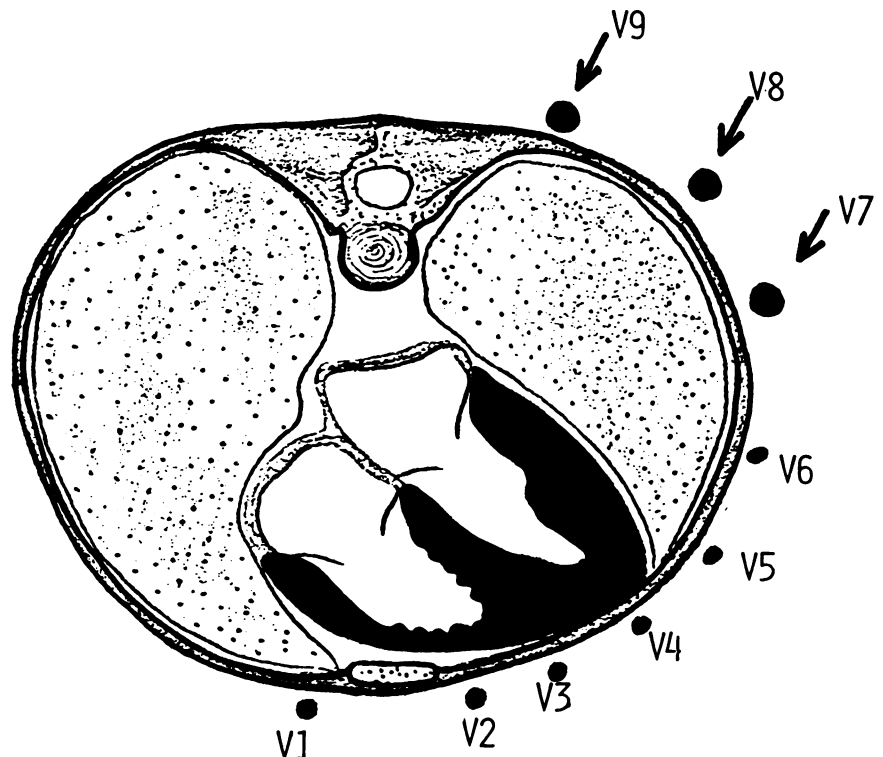


FIG. 1—Cross-sectional view of thorax showing relation of electrocardiographic chest leads to heart.

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way between the midscapular line and the spine (V_9), all at the same level as V_6 . Skin markings for the electrode locations of all nine precordial leads were made at the time the first ECG was done to ensure that the same position was used for subsequent recordings. In a few instances double standardization (2 cm = 1 mV) was used for recording the additional chest leads, but in no instances were voltage criteria considered for diagnostic purposes. A similar ECG was made daily for 3 consecutive days, and concentrations of serum enzymes (serum glutamic oxaloacetic transaminase [SGOT], lactate dehydrogenase [LDH], LDH, and creatine phosphokinase [CPK]) were determined daily for 3 days.

A diagnosis of myocardial infarction was established on the basis of a suggestive history (severe anterior chest pain lasting longer than half an hour) plus a typical 12-lead ECG (with an evolving current of injury pattern or the development of abnormal Q-waves) or characteristic changes in serum enzyme concentrations (rise and fall in the concentrations of SGOT, LDH or CPK for which no other cause was evident) or both.⁶ Electrocardiographic localization of myocardial injury or infarction was established according to the leads that showed characteristic changes, as follows: V_1 to V_3 , antero-septal; V_4 to V_6 , anterolateral; and II, III and aVF, inferior.

Twenty individuals (mean age 51 years) without evidence of heart disease were studied by means of a similar 15-lead ECG so that the normal morphology for leads V_7 , V_8 and V_9 could be defined.

Observations

None of the ECGs in the 20 healthy individuals showed Q-waves lasting at least 40 ms on the additional chest leads. The T-wave was upright in V_7 in all the ECGs, flat in V_8 in one individual and flat in V_9 in three individuals. Inversion of the T-wave was never seen in the additional chest leads.

Among the 117 persons admitted to the coronary care unit acute myocardial infarction was eventually diagnosed with the use of the above-mentioned criteria in 46 (39%). The diagnosis was established from the history plus a typical 12-lead ECG

or characteristic changes in serum enzyme concentrations, or both, in 36 (78%) of the 46; from the history plus characteristic changes in serum enzyme concentrations with a non-specific ECG in 7 (15%); and from the history plus typical acute and evolving electrocardiographic changes without diagnostic changes in serum enzyme concentrations in 3 (7%).

When leads V_7 , V_8 and V_9 were analysed, a current of injury pattern, ST-segment elevation or the development of Q-waves lasting at least 40 ms, or a combination of these features, was found in nine persons. In six individuals these changes were associated with signs of acute myocardial infarction (anterolateral in three and of the inferior wall in the other three) in the standard 12-lead ECG, but in three individuals they were the only definite electrocardiographic evidence of myocardial injury or necrosis. In Fig. 2 is the 15-lead ECG of a person with a typical history and serum enzyme changes diagnostic of acute myocardial infarction, in whom only the chest leads V_7 , V_8 and V_9 show elec-

trocardiographic changes characteristic of acute injury or infarction. Prominent R-waves in leads V_1 to V_3 were not seen in the ECGs of the three individuals in whom changes of myocardial infarction were noted in the additional chest leads as well as in the standard 12 leads in association with acute (two patients) or old (one patient) antero-septal infarction. In one patient with diagnostic changes in the additional chest leads but no evidence of acute or old antero-septal infarction in the standard 12-lead ECG the R-waves were prominent in leads V_1 and V_2 (Fig. 3), a change that has been considered highly suggestive of "true posterior" or "dorsal" wall infarction.⁷

Discussion

These observations suggested the following conclusions:

1. The QRS-ST-T pattern of leads V_7 , V_8 and V_9 in healthy individuals is qualitatively similar to that of leads V_5 and V_6 (Q-waves lasting less than 40 ms, isoelectric ST-segments and upright T-waves).
2. Persons with diagnostic changes

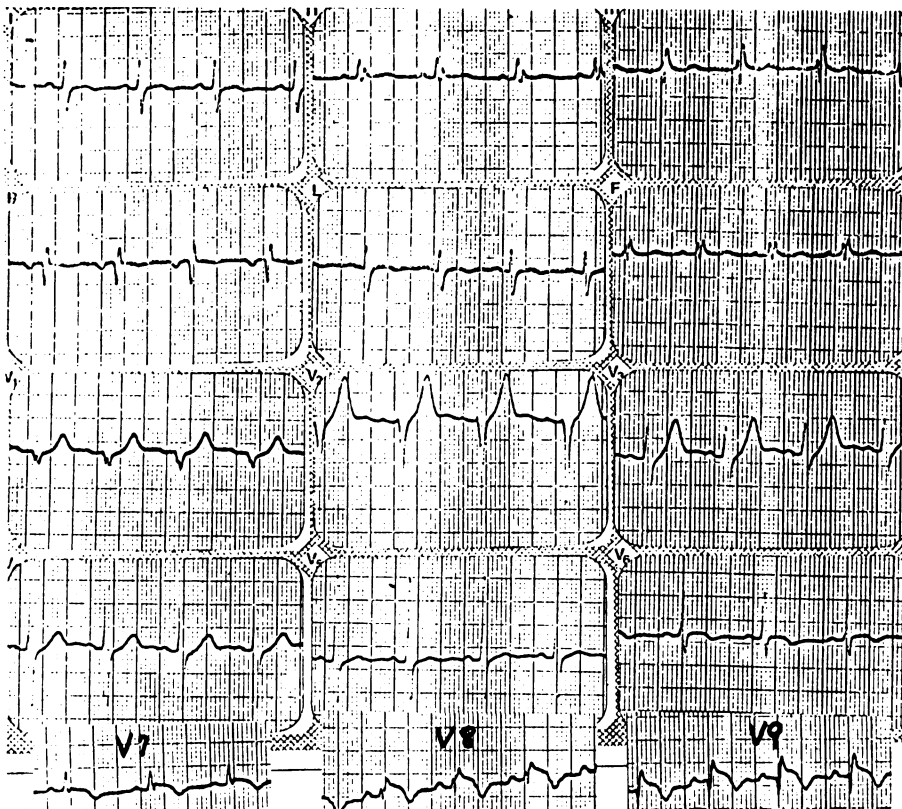


FIG. 2—Evidence of old antero-septal infarction in 12-lead electrocardiogram. While V_6 pattern suggests myocardial ischemia, clearer injury pattern is seen in leads V_8 and V_9 . Serial changes in serum enzyme concentrations were characteristic of acute myocardial infarction.

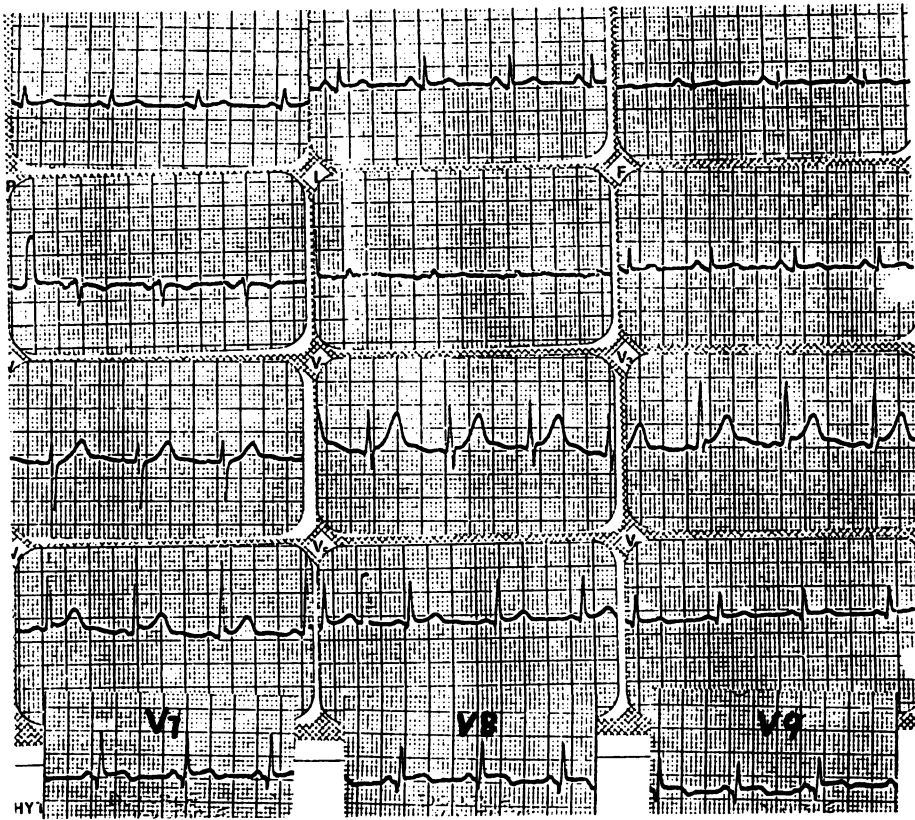


FIG. 3—Abnormal Q-waves and ST-segment elevation in leads V₇, V₈ and V₉, and conspicuous R-waves in leads V₂ and V₃. Patient presented with severe retro-sternal chest pain, and serial determinations of serum enzyme concentrations showed changes characteristic of acute myocardial infarction.

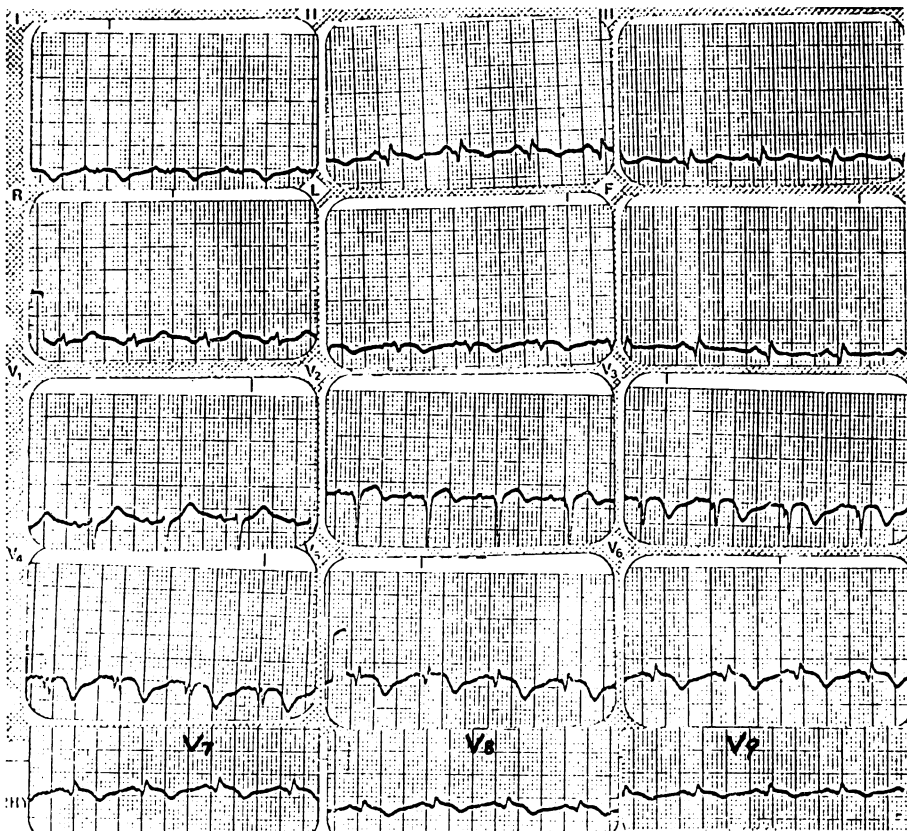


FIG. 4—Extensive acute myocardial infarction. True posterior wall involvement is suggested by appearance of leads V₇, V₈ and V₉. Simultaneous occurrence of anteroseptal infarction may abolish the prominent R-waves in leads V₁ and V₂, which are usually associated with true posterior wall necrosis.

of acute myocardial infarction in the 12-lead ECG may or may not show similar changes in leads V₇, V₈ and V₉. As a corollary, the presence of changes of myocardial injury or infarction in leads V₇, V₈ and V₉ may suggest a larger area of involvement than the classic 12-lead ECG appears to indicate.

3. In some patients with acute myocardial infarction diagnosed from a typical history and characteristic changes in serum enzyme concentrations who have a normal 12-lead ECG, changes indicative of myocardial injury or necrosis may be found in the additional chest leads. The fact that in three of our patients with acute myocardial infarction the only diagnostic electrocardiographic changes were seen in the additional chest leads appears to indicate that these leads reflect areas of the heart that are not properly explored by any of the standard 12 leads.

For leads V₇, V₈ and V₉ the electrodes seem to face the far lateral portion of the left ventricular wall, which is very likely the portion that has been referred to as the true posterior or dorsal wall of the heart. This suggestion is supported by the observation of the development of changes of acute infarction in leads V₇, V₈ and V₉ in a person whose ECG developed prominent R-waves in leads V₁ and V₂ (Fig. 3), a change that has been considered highly suggestive of infarction of the true posterior wall of the heart.⁷ However, the failure of R-waves to become prominent in lead V₁ or V₂ in the presence of infarction of the dorsal wall of the heart may be expected when anteriorly directed vectors are abolished because of the previous or simultaneous occurrence of an anteroseptal infarction, as is illustrated in Fig. 4; the features of this ECG suggest that the patient's extensive involvement included the far lateral or dorsal wall of the heart, but that the old anteroseptal wall damage prevented the development of prominent R-waves in leads V₁ and V₂.

At the time of this study we did not have facilities for recording from orthogonal lead systems. Although such systems may reflect these changes in a simpler form, they are not yet available in many hospitals. We have shown that recording from three additional chest leads, V₇, V₈ and V₉, which can be done with little

additional expenditure or effort, is helpful for electrocardiographic disclosure of myocardial injury or necrosis involving portions of the myocardium that may not be clearly reflected on the routine 12-lead ECG. These additional chest leads may show changes of injury or necrosis associated with infarction of the anterolateral or inferior wall, perhaps reflecting a greater extension of damage, or may, in about 7% of all cases of infarction, reveal the only electrocardiographic abnormality indicative of myocardial injury or necrosis.

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Ludiomil® maprotiline hydrochloride

Brief prescribing information. Indications Endogenous depressive illness, including the depressed phase of manic-depressive illness (bipolar depression) and involuntarily melancholia. Selected patients suffering severe depressive neurosis. **Contraindications** Ludiomil (maprotiline) should not be used concomitantly with monoamine oxidase inhibitors; at least fourteen days should elapse between discontinuing one of the interacting drugs and replacing it with the other. Ludiomil is contraindicated in patients with existing severe hepatic or renal damage, a history of severe blood dyscrasias, narrow angle glaucoma, convulsive disorders and during the acute recovery phase following myocardial infarction. Not recommended for use in children. **Use in Pregnancy** Safe use of Ludiomil during pregnancy and lactation has not been established, therefore, it should not be administered to women of childbearing potential or nursing mothers unless the benefits outweigh the possible hazards. **Warnings** Extreme caution should be used when Ludiomil (maprotiline) is given to patients with known cardiovascular disease including a history of myocardial infarction, arrhythmias and/or ischemic heart disease. Use with caution in hyperthyroid patients or those on thyroid medication, and in patients with a history of urinary retention, particularly in the presence of prostatic hypertrophy. Close supervision and careful adjustment of dosage is required when administering Ludiomil with anticholinergic or sympathomimetic drugs. Patients requiring concomitant treatment for hypertension should not be given antihypertensives of the adrenergic-neurone inhibitor type, such as guanethidine. Activation of psychosis in schizophrenic patients, hypomanic or manic episodes in patients with cyclic disorders have occurred with tricyclic antidepressants. The use of an antipsychotic drug in these latter two conditions is recommended should they occur in the course of Ludiomil administration. **Precautions** Seriously depressed patients must be carefully supervised due to the possibility of suicide. Patients should be warned that their responses to alcoholic beverages or other CNS depressants may be exaggerated. Patients should also be cautioned against performing potentially dangerous tasks that require mental alertness and good physical coordination. Periodic blood cell counts and liver function tests are recommended with prolonged therapy. Prior to elective surgery, Ludiomil should be discontinued for as long a period as clinically feasible. **Adverse reactions** The following adverse reactions have been reported either with Ludiomil or the tricyclic antidepressant drugs: **Neurological:** numbness, tingling, paresthesias of extremities, incoordination, ataxia, tremors, peripheral neuropathy, extrapyramidal symptoms, seizures, alteration in EEG patterns, tinnitus. **Behavioral:** confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation, insomnia and nightmares, hypomania, mania, exacerbation of psychosis, decrease in memory, feelings of unreality, weakness and fatigue, drowsiness, dizziness, urinary frequency. **Autonomic:** dry mouth and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation; paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract, perspiration, flushing. **Cardiovascular:** hypotension, hypertension, congestive heart failure, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke and syncope. **Hematologic:** bone marrow depression including agranulocytosis, eosinophilia, purpura and thrombocytopenia may occur as an idiosyncratic response. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during the therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression. **Gastrointestinal:** nausea or vomiting, anorexia, epigastric distress, diarrhea, bitter taste, stomatitis, abdominal cramps, black tongue, dysphagia, increased salivation, altered liver function. **Endocrine:** gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, impotence, testicular swelling, elevation or depression of blood sugar levels, weight gain or loss. **Allergic or toxic:** skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever, obstructive jaundice, nasal congestion. **Dosage** Adults **Outpatients:** initially 25 mg t.i.d., may be increased in increments of 25-50 mg to a maximum of 50 mg q.i.d. **Hospital patients:** initially 50 mg b.i.d. or t.i.d., may be increased in increments of 25-50 mg to a maximum of 300 mg daily. **Elderly patients:** generally 25 mg t.i.d. or q.i.d. **Maintenance:** dosage may be gradually reduced to 75 mg daily or less. A single daily dose of 75 mg to 150 mg at bedtime can be given by virtue of the long half-life of Ludiomil. This would ensure better patient compliance and may obviate the need for hypnotics. **Supplied** Tablets containing maprotiline hydrochloride, film coated, slightly biconvex: 25 mg, light orange marked CIBA one side and DP on other; 50 mg, orange marked CIBA on one side and ER on other; 75 mg, coral, scored, marked CIBA on one side and FS on other. Bottles of 50 and 500 tablets. Product Monograph supplied on request. **References** 1. Trick, K.L.K.: Double-Blind Comparison of Maprotiline (Ludiomil®) with Amitriptyline in the Treatment of Depressive Illness. *J. Int. Med. Research*, Vol. 3, Suppl. 2, 1975. Edited by J.E. Murphy, Cambridge Medical Publications Ltd., England. pp. 67-70 2. Mathur, G.N.: A Double-Blind Comparative Clinical Trial of Maprotiline (Ludiomil®) and Amitriptyline. *J. Int. Med. Research*, Vol. 3, Suppl. 2, 1975. Edited by J.E. 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Slow-Fe folic® (ferrous sulfate-folic acid) hematinic with folic acid

Indications

Prophylaxis of iron and folic acid deficiencies and treatment of megaloblastic anemia, during pregnancy, puerperium and lactation.

Contraindications

Hemochromatosis, hemosiderosis and hemolytic anemia.

Warnings

Keep out of reach of children.

Adverse Reactions

The following adverse reactions have been reported:

Nausea, diarrhea, constipation, vomiting, dizziness, abdominal pain, skin rash and headache.

Precautions

The use of folic acid in the treatment of pernicious (Addisonian) anemia, in which Vitamin B₁₂ is deficient, may return the peripheral blood picture to normal while neurological manifestations remain progressive.

Oral iron preparations may aggravate existing peptic ulcer, regional enteritis and ulcerative colitis.

Iron, when given with tetracyclines, binds in equimolecular ratio thus lowering the absorption of tetracyclines.

Dosage

Prophylaxis: One tablet daily throughout pregnancy, puerperium and lactation. To be swallowed whole at any time of the day regardless of meal times.

Treatment of megaloblastic anemia: During pregnancy, puerperium and lactation; and in multiple pregnancy: two tablets, in a single dose, should be taken daily.

Supplied

SLOW-Fe folic tablets have an off-white colour and are supplied in push-through foil packs of 30; available in units of 30 and 120 tablets.

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