from a decreased rate of oxidative metabolism, thereby decreasing utilization through the GABA shunt.[11] Increased C.S.F. GABA levels during a migraine headache may therefore reflect an increase in tissue GABA levels occurring coincidentally with the anoxic ischaemic phase of the attack. To support this, increased C.S.F. GABA levels have also been found in patients with cerebral ischaemia and infarction due to cerebrovascular disease.[7]

An increase of GABA in the C.S.F. is most likely the result of increased GABA concentrations in brain tissue extracellular fluid. Whether this is entirely secondary to a rise in intracellular GABA content or due to anoxic ischaemic conditions that encourage neuronal GABA release cannot be established by this study. Extraneuronal release and eventual intraneuronal depletion of dopamine, noradrenaline, and serotonin in ischaemic brain has been shown in other studies,[12 13] so that GABA could be similarly affected.

GABA now seems well established as a major inhibitory neurotransmitter in the C.N.S.[14] Indeed, it has been strongly identified with the natural inhibitory neurotransmitter producing hyperpolarization after neuronal depolarization.[15] Its action is brief and rapidly reversed, mainly by avid cellular reuptake.[3] If excess GABA release is assumed to take place during the prodromal ischaemia of migraine it seems not unreasonable to relate this in part to depression of cortical neuronal function clinically manifest by the rapidly and spontaneously reversible types of neurological deficit that frequently occur in this phase of the attack.[16] In view of the frequency of migraine prodromes affecting visual function is should be pointed out that GABA is also present in large concentration in the retina,[17] where it also functions as an inhibitory neurotransmitter.[18]

In conclusion, the surprisingly clear-cut results in our small series seem to indicate the need for a larger study to confirm the disorder of C.N.S. GABA metabolism during migraine. What pathogenetic role such a disorder has in the syndrome, however, can be only speculative.

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# Value of Infarct-specific Isotope ($^{99m}$Tc-labelled Stannous Pyrophosphate) in Myocardial Scanning

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## Summary

**With the use of $^{99m}$Tc-labelled stannous pyrophosphate scans positive for myocardial infarction were obtained from 28 patients in the acute stage of the disease. In some cases the scan was positive when the initial electrocardiogram gave equivocal results. Negative scans were obtained from a control group of patients and from eight patients in hospital with chest pain but with no other evidence of recent myocardial infarction.**


## Introduction

The diagnosis of myocardial infarction is based on clinical, electrocardiographic, and enzymatic abnormalities. Some or all of these indices may be equivocal with a resultant delay in giving treatment. The use of myocardial scanning as an aid to establishing an earlier diagnosis has been limited mainly by the lack of an infarct-specific isotope suitable for routine use; $^{43}$K (ref.[1]) and $^{131}$I-labelled oleic acid,[2] used to show the infarct as a void against a background of normal activity, are unsatisfactory.[3] Though $^{99m}$Tc-labelled tetracyclines concentrate specifically in the acutely infarcted myocardium, their value in identifying myocardial infarcts[4] is limited by both the poor target:background ratio and the interference from hepatic uptake.

The observation that calcium ions located in the mitochondria of irreversibly damaged myocardial cells are deposited in a crystalline hydroxyapatite structure[5] resulted in the successful experimental[6] and clinical[7] use of the bone-seeking radionuclide $^{99m}$Tc-labelled stannous pyrophosphate in visualizing acute myocardial infarcts. This added a new dimension to myocardial scanning, and we report here our results with the technique in cases admitted to a coronary care unit.

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## Patients and Methods

Forty patients with no clinical or electrocardiographic evidence of myocardial infarction who had been referred for bone scans were used as controls. After an intravenous injection of 15 mCi $^{99m}$Tc-

labelled stannous pyrophosphate images of the myocardial area were obtained in the anterior, lateral, and left anterior oblique positions. A Nuclear Chicago Pho/Gamma Four scintillation camera linked to a videotape and data store was used and 300 000 counts were collected using a low-energy, high-resolution collimator. The images were recorded initially on Polaroid film and, later, on radiographic film using a Nuclear Chicago Microdot.

Twenty-eight patients (22 male, 6 female) aged 10-71 years with definite myocardial infarction (W.H.O. criteria[8]) were scanned (see table), as were eight patients (six male, two female) with chest pain but no enzymatic or electrocardiographic evidence of the disease. Sequential scans to monitor the temporal resolution of the infarct were obtained from 11 patients.

*Details of Patients with Myocardial Infarction and Results of Investigations*

| Case No. | Age (Years) | Sex | Location of Infarct | | Degree in which Scan was Positive | Time of Scanning after Onset of Chest Pain |
|---|---|---|---|---|---|---|
| | | | E.C.G. | Scan | | |
| 1 | 56 | M. | Inferior | Inferior | Strong | 15 h |
| 2 | 71 | M. | Anterior | Anterior | Faint | 24 h |
| 3 | 58 | M. | Inferior | Inferior | Strong | 24 h |
| 4 | 59 | F. | Inferior | Inferior | Strong | 24 h |
| 5 | 43 | M. | Anterior | Anterior | Faint | 24 h |
| 6 | 56 | F. | Anterior | Anterior | Strong | 36 h |
| 7 | 63 | M. | Anterior | Anterior | Strong | 36 h |
| 8 | 48 | M. | Anterior | Anterior | Strong | 2 d |
| 9 | 52 | M. | Anterior | Anterior | Strong | 3 d |
| 10 | 59 | M. | Anterior | Anterior | Strong | 3 d |
| 11 | 64 | F. | Anterior | Anterior | Strong | 4 d |
| 12 | 53 | M. | Anterior | Anterior | Strong | 4 d |
| 13 | 10 | M. | Anterior/ inferior | Anterior/ inferior | Strong | 4 d |
| 14 | 61 | M. | Anterior | Anterior | Faint | 4 d |
| 15 | 63 | M. | Anterior | Anterior | Strong | 4 d |
| 16 | 63 | M. | Anterior | Anterior | Strong | 4 d |
| 17 | 59 | M. | Anterior | Anterior | Strong | 4 d |
| 18 | 62 | M. | Anterior | Anterior | Faint | 5 d |
| 19 | 54 | M. | Anterior | Anterior | Faint | 5 d |
| 20 | 62 | F. | Anterior | Anterior | Faint | 6 d |
| 21 | 61 | M. | Anterior | Anterior | Faint | 6 d |
| 22 | 62 | M. | Inferior | Inferior | Faint | 6 d |
| 23 | 51 | F. | Inferior | Inferior | Strong | 6 d |
| 24 | 48 | M. | Anterior | Anterior | Faint | 6 d |
| 25 | 57 | F. | Anterior | Anterior | Faint | 7 d |
| 26 | 61 | M. | Anterior | Anterior | Strong | 7 d |
| 27 | 59 | M. | Anterior | Anterior | Faint | 8 d |
| 28 | 63 | M. | Anterior | Anterior | Faint | 10 d |

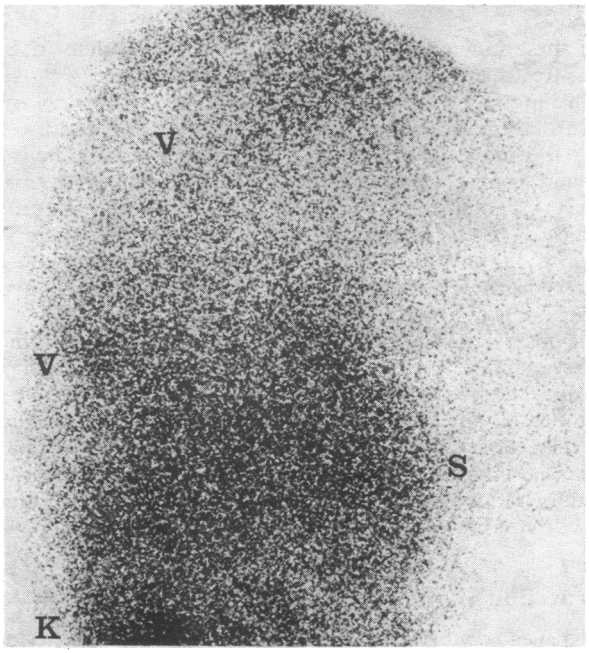


FIG. 1—Normal myocardial scan taken in left lateral position. S = Sternum. V = Vertebral column. K = Kidney.

All the patients were examined under constant electrocardiographic and medical surveillance. The optimum scanning time, found to be 30-45 minutes after the injection, had the effect of reducing interference from bone uptake and allowing for slow blood pool clearance of the radiopharmaceutical.

## Results

Scans negative for myocardial infarction were obtained from the controls and from the eight patients with chest pain but no other evidence of myocardial infarction (fig. 1). A positive scan (one showing increased activity over the myocardium) was obtained from all 28 patients with definite infarction. The location of the infarct as shown by the scan correlated in all cases with the electrocardiographic findings (table). Most of the infarcts were anterior or anterolateral (figs. 2 and 3) but five patients had inferior infarcts (fig. 4). The anterior infarcts could be identified in all three projections but the inferior infarcts were best shown by the lateral scan.

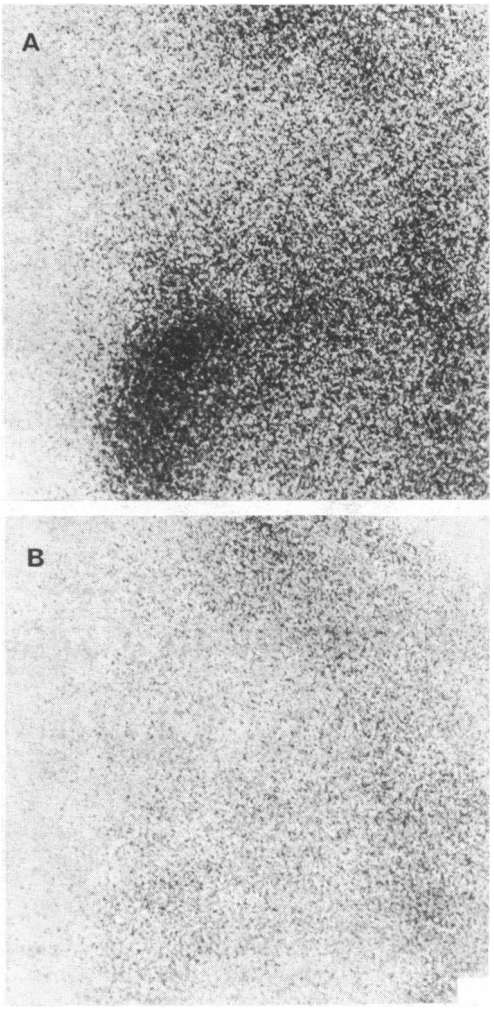


FIG. 2—Case 10. A, scan showing increased uptake anteriorly three days after onset of chest pain; B, normal appearances 10 days later.

The earliest scan was obtained 15 hours after the onset of ischaemic chest pain and was strongly positive. Seven patients had positive scans within 36 hours of the symptoms developing. Most (16) of the positive scans were obtained within three to six days of the ischaemic episode. Three out of four patients scanned 7-10 days after the onset of chest pain gave faintly positive scans (table). Of the 11 scans obtained sequentially nine became negative within 12-21 days (figs. 2, 3, and 4) and activity was greatly reduced in the other two.

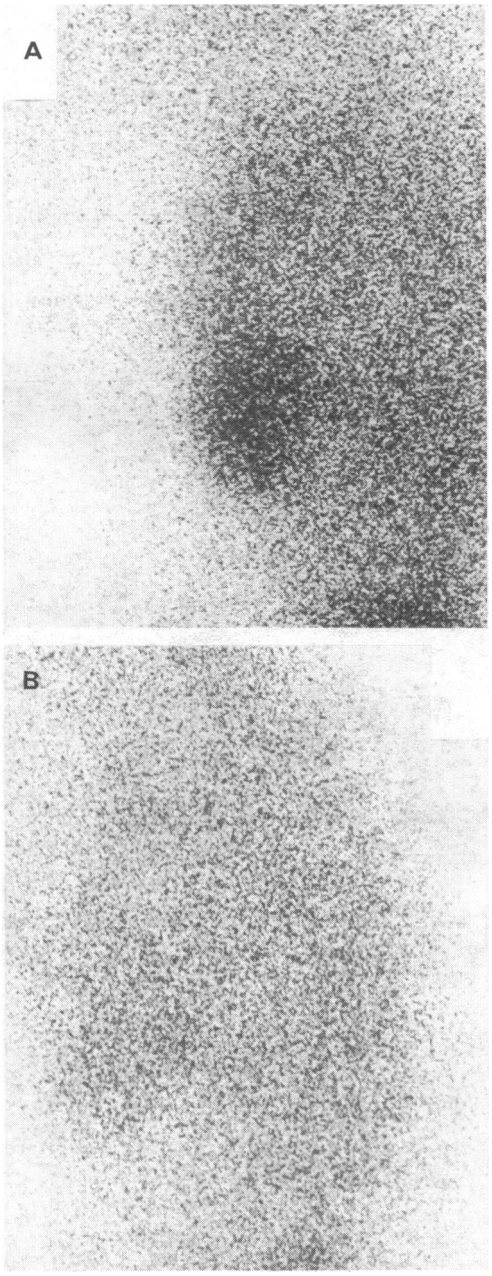


FIG. 3—Case 6. A, positive anterior scan 36 hours after onset of symptoms; B, normal appearances 19 days later.

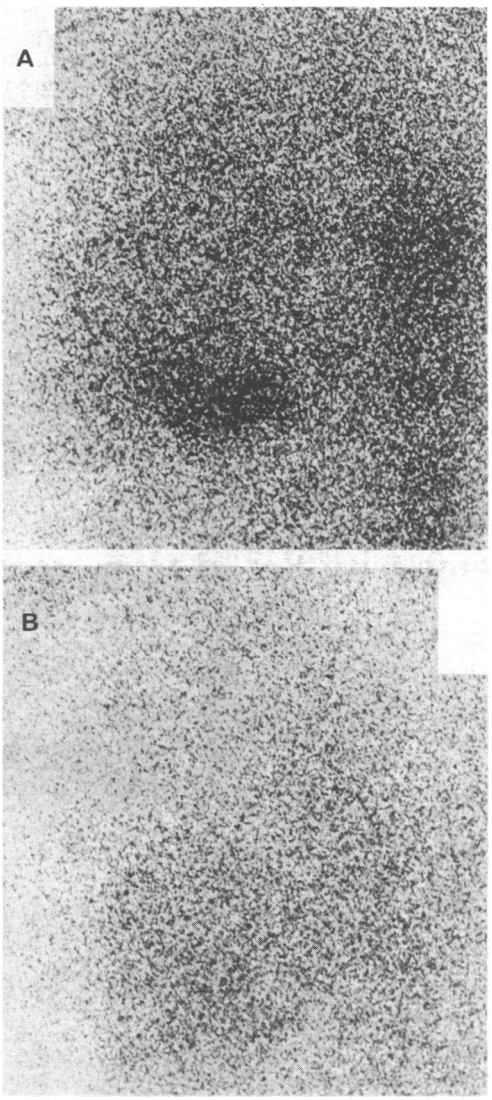


FIG. 4—Case 3. A, positive inferior scan 24 hours after symptoms developed; B, normal appearances 11 days later.

Two of the eight patients with chest pain had positive effort electrocardiograms. Two patients had had documented anterolateral myocardial infarcts six months previously. All eight patients gave negative scans and none developed evidence of myocardial infarction during their stay in hospital.

Several small areas of increased activity in occult rib fractures, not detected radiologically, were seen in three patients who had received external cardiac massage. These did not interfere with interpretation of the scan. The youngest patient in our study, a boy aged 10 years (case 13) admitted to hospital after blunt chest trauma, had electrocardiographic evidence of anterior and inferior myocardial infarction. Myocardial scanning four days after the accident showed an extensive area of increased activity over the anterior and inferior parts of the myocardium.

## Discussion

Most deaths from myocardial infarction occur within a few hours of the onset of symptoms.[9 10] Early initiation of intensive coronary care is imperative. The early positive scans from patients with acute myocardial infarction (table), the negative scans from patients with old infarcts, and the gradual return to normal activity in the healing infarct indicate the value of myocardial scanning in the early assessment of patients admitted with chest pain. Positive scans obtained at an early stage (table), when the electrocardiogram may be equivocal, offer an opportunity for more exact quantification of the infarct within the first 24 hours and serve as a reference against which to compare the subsequent reduction in its size.[11 12]

No patient with a negative scan later developed enzymatic or electrocardiographic evidence of myocardial infarction while in hospital. This is important in relation to the bed occupancy of a coronary care unit. In a study of 32 such units in the United States the proportions of patients with definite myocardial infarction ranged from 28 to 85% (mean 50·4%) and the average length of stay was 4·7 days.[13] A negative myocardial scan may therefore facilitate the early discharge of patients without myocardial infarction, resulting in more effective and more economic coronary care.

The concentration of $^{99m}$Tc-labelled stannous pyrophosphate in rib fractures did not cause problems in interpretation. The concentration of pertechnetate-99m, which has been described in malignant breast lesions, can be clinically excluded.[14] The safety of bone scanning with $^{99m}$Tc-labelled stannous pyrophosphate is not in dispute.[15] Over 500 bone scans have been

performed in this hospital with no untoward effects, and no side effects were recorded during this study.

The practical application of myocardial scanning in the patient with acute myocardial infarction would be enhanced by the addition of portable scanning facilities.

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# SHORT REPORTS

## Splenoma with Portal Hypertension

Splenomas or splenic hamartomas are non-capsulated, single or multiple nodules in the spleen and consist of native elements in gross disproportion.[1] The first case of splenoma was recorded in 1865 by Rokitansky,[1] and until August 1970 only 39 cases had been reported.[2] All but four of these were asymptomatic (excluding abdominal swelling) and discovered accidentally at exploratory laparotomy or necropsy. Of the four symptomatic cases two had pancytopenia,[2 4] one had anaemia,[4 5] and one had thrombocytopenia.[3] We report here the first recorded case of symptomatic splenoma associated with haemodynamically proved portal hypertension but without any of the reported haematological abnormalities.

### Case Report

A 35-year-old Kashmiri Moslem weaver presented with pain in the left abdomen of 11 months' duration. The liver was enlarged (3 cm) and non-tender, and there was a palpable mass in the left hypochondrium, which moved slightly with respiration. The routine haematological measurements showed nothing abnormal except occasional Howell-Jolly bodies in the peripheral smear. The results of urine and stool analysis and liver function tests were normal. Blood sugar, urea, and creatinine were normal. Intravenous pyelography, barium meal examination, and serum electrophoresis for proteins showed nothing abnormal. Immunological studies showed proportions of normal T and B cells in blood, normal T-cell function, poor T-cell response to phytohaemagglutinin, and poor B-cell population in the spleen.

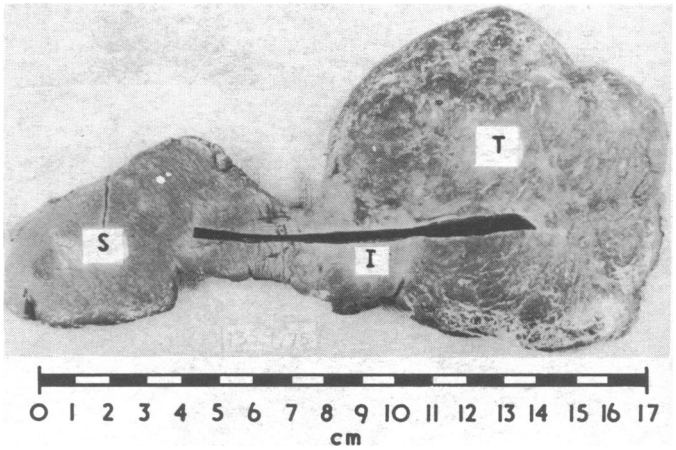
A splenoportogram showed a 5-cm circular dye-opaque area drained by a tortuous aberrant large vein (1·5 cm), joining a small main splenic vein before the formation of the portal vein. The haemodynamic studies showed high intrasplenic pressure (300 mm $H_2O$; normal, 150 mm $H_2O$), slightly raised wedge hepatic venous pressure (7·56 mm Hg; normal 5·4, S.D. 1·8), raised free hepatic venous pressure (6·12 mm Hg; normal 3·1, S.D. 1·6 mm Hg); and raised estimated hepatic blood flow (1·27 l/min; normal 0·8-1·2 l/min). Splenic vein thrombosis was diagnosed preoperatively.

At exploratory laparotomy a vascular tumour of the spleen was found as well as interconnecting vascular channels among the greater omentum, the posterior wall of the stomach, the splenic flexure, and the splenic tumour. The liver seemed to be fatty. Splenectomy and devascularization of the stomach were performed, and a wedge biopsy specimen of the liver taken.

The spleen weighed 290 g (see figure) on the surgical desk. There were three distinct parts; the main spleen, an "isthmus," and the tumour mass. The isthmus connected the lower pole of the spleen to the tumour. Histological examination showed a transition from normal to abnormal splenic architecture. The latter consisted of malformed, ill-defined lymphoid follicles "invading" the red pulp. The red pulp was unremarkable. Splenic vasculature showed perivascular haemorrhages and subintimal fibrous and fatty plaques suggestive of portal hypertension. The liver showed portal fibrosis. Splenoma with predominating abnormal lymphoid components was diagnosed.

### Comment

The high intrasplenic pressure, the drainage of portosystemic collaterals into the azygos system, and the histological character of the splenic vasculature were considered adequate evidence of portal hypertension.




External surface of spleen showing tumour (T) arising from lower pole of spleen (S) with interconnecting "isthmus" (I).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## Ocular Perforating Injury Caused by a Sparrow

Ocular conditions caused by birds are not as rare as might be supposed. Thus a sensitive person may develop acute allergic blepharoconjunctivitis through contact with feathers. The Newcastle virus, which causes fowl-pest, can cause acute follicular conjunctivitis. Psittacosis can cause iritis and subacute focal retinitis leading to macular oedema.[1]