Saravolatz and co-workers documented the lower levels of erythromycin while treating patients with Legionnaires' disease with oral erythromycin and one of their patients developed Legionnaires' disease (LD) while receiving oral erythromycin.

Among the antibiotics, rifampin has been shown in vitro to be more active than erythromycin against the LD bacterium. One should consider its use along with erythromycin in those patients not responding satisfactorily to erythromycin or when pulmonary abscesses are present.

Saravolatz and co-workers also showed that the LD bacterium could be found in sputum and bronchial washings by fluorescent antibody examination. This mode of diagnosis may be useful for early detection of Legionnaires' disease.

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REFERENCES


Exercise Testing Early after Myocardial Infarction

To the Editor:

The article entitled “Exercise Testing Three Weeks after Myocardial Infarction” by Smith and his associates (Chest 75:12-16, 1979) confirms that low-level exercise testing before discharge from the hospital of patients who have suffered a myocardial infarction identifies individuals at high risk of recurrent ischemic events, including sudden death. Furthermore, in other individuals, therapy was altered because of the detection during exercise of arrhythmias or left ventricular dysfunction. This important information was obtained without significant complications.

These data are encouraging, but one must ask whether or not this test might be performed more safely and be equally as useful if carried out three or four weeks later. At present, we do not know. To recommend testing prior to discharge implies that any increased risk which may exist is justified by the desirability of identifying unfavorable responses to exercise testing earlier, rather than later, in convalescence.

My concern with the timing of the procedure derives from the increasing number of patients referred to our laboratory for this examination prior to discharge from the hospital, which now is often no more than 10 to 14 days following infarction. As a consequence of the shortened stay in the hospital, we have been asked to perform these studies as early as nine days after the infarction. Furthermore, I am aware of at least one disaster during such testing in another institution. Fatal left ventricular rupture occurred during exercise on a treadmill 13 days after infarction.

Once it is accepted that timing remains an important issue, several pertinent questions need to be answered: (1) How often do the recurrent coronary events predicted by ischemic ST-segment change occur in the first few weeks after discharge from the hospital? (2) Do exercise-induced arrhythmias predict life-threatening arrhythmias during these weeks? (3) Does exercise testing in the early period after infarction identify arrhythmias predictive of dangerous complications better than does continuous electrocardiographic recording (Holter monitor)? (4) Can left ventricular dysfunction detected during exercise be otherwise identified? (5) If not, is it important to discover the dysfunction prior to discharge?

It may well be that there is information to be learned from exercise testing during the third week following myocardial infarction that is important for the care of the patient during the next few weeks, and it may be that exercise testing in the third week is not more hazardous than in the seventh week, but until this can be established, the procedure should not be advocated for routine use.

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To the Editor:

We appreciate Lindsay's thoughtful letter. His primary concern is with the timing of the exercise stress test after myocardial infarction. He wants to know if the same information can be gained by stress testing later in the convalescent stage of infarction, at a time when it might be safer. There is evidence that changes on exercise testing during the first test are reproducible in subsequent tests at 7 and 11 weeks. Twenty-seven of our first 62 patients had repeat tests three to six months after infarction, and 74 percent (20/27) had similar results. Agreement between functional testing at three weeks and three months after infarction was also reported by Torno Alfonso et al.

A corollary to this first question is whether or not the risk of recurrent events is so high during the first month after discharge that the risk must be identified and intervention instituted before discharge. The risk of dying after discharge is greatest during the first year and particularly within the first six months. Approximately 1.8 percent of the patients who have suffered an infarction die each month for the first six months; thereafter, the risk falls precipitously to about 0.3 percent per month. Six of our 62 exercised patients died within the first year (five within the first six months; two within the first two months). Therefore, the identification of patients early after discharge may indeed make a difference if appropriate therapy could be initiated to prevent these fatalities.

The test has been safe, with no deaths in 628 patients recently studied from five centers. We are also aware of many other centers that are performing the test; and, to our knowledge, the only fatality that has been brought to our attention is that in Lindsay's letter, and full details are not given. It should be emphasized that the test is stopped when a heart rate of 120 beats per minute is achieved. This rate is similar to that obtained during ordinary activity soon after the patient is discharged. The question must be raised, therefore, as to whether it is safer to observe the patient during activity and attempt correction of arrhythmias or ischemia (or both) or to have the patient exert himself without observation. Although the test is reasonably safe, the question of whether it would be safer at seven weeks and still provide the same information is not answered, nor are there...
satisfactory answers to the other cogent questions raised by Lindsay. These pertinent questions should be answered by the Multicenter Postinfarction Program study. This study, funded by the National Heart, Lung, and Blood Institute, was initiated on Jan 1, 1979. Eight hospitals in Rochester, NY, New York City, St. Louis, and Tucson will enroll 1,000 patients with myocardial infarction and follow them for one year. Patients enrolled in the study will have a modified stress test, a 24-hour electrocardiographic recording (Holter monitor), and a radionuclidic left-ventricular ejection fraction determined before discharge from the hospital. The predictability for recurrent coronary events of each of these tests can then be determined. Until the results of that study are known, each physician or institution must decide the risk-benefit ratio of early stress testing after infarction. We continue to find it helpful in making decisions regarding various aspects of the management of patients in the early period after infarction.

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REFERENCES

Aspiration Pneumonia following Gavage Feeding

To the Editor:

With increasing appreciation of the need for maintaining adequate nutrition, hyperalimentation is becoming correspondingly popular. Use of the intravenous route involves risks of infection, fluid overload and maintenance of electrolyte balance. Most physicians agree that enteral alimentation is preferable if possible. For this reason many patients who will not eat are fed by gavage or gastrostomy.

Unfortunately, many of the patients who require alimentation are elderly, debilitated or paralyzed. In our hospital we have observed that gastric feedings are often regurgitated and aspirated into the bronchial tree. This often leads to pneumonia and, if not appreciated early, may be fatal. On my service, we have discontinued gavage and gastrostomy for feeding purposes. If a patient who cannot eat requires prolonged enteral hyperalimentation, we perform jejunostomy at approximately 40 cm distal to the ligament of Treitz. The procedure is well tolerated. Nasogastric suction is maintained postoperatively until intestinal peristalsis has been reestablished. Since instituting this regimen, we have not encountered pneumonia due to aspiration of food.

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Mediastinal Granuloma and Fibrosing Mediastinitis

To the Editor:

In Chest (75:320-324, 1979), Dines et al reviewed their experience at the Mayo Clinic over the past two years with patients with mediastinal granuloma and fibrosing mediastinitis. This was an update of a continuing series of reports from that clinic, as was noted in the article, that now totals 124 patients.1-3 Their conclusion was that in patients with undiagnosed mediastinal granuloma, especially large granuloma, felt to be secondary to histoplasmosis, the mediastinal granuloma should be removed surgically to prevent the potential complication of fibrosing mediastinitis. Their supposition is that the mediastinal fibrosis is caused by rupture of fibrocaseous material from mediastinal lymph nodes into the mediastinum, although they state that there is no proof that this is the cause or that excision will prevent it.

We feel it would be useful if a followup report on these patients were given to show whether they remained symptom-free, or if some may have ultimately developed mediastinal fibrosis. If post surgical mediastinal fibrosis has developed, it casts doubt on the validity of the supposition that removal of the granulomas prevents mediastinal fibrosis and raises the question of whether the surgical procedure itself may cause mediastinal fibrosis in some cases due to spillage of caseous material into the mediastinum.

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REFERENCES

To the Editor:

I appreciate the comments from Drs. Beekman and Weled in regard to our article. We are compiling followups on the patients to determine whether they have indeed remained symptom-free or whether they have ultimately developed mediastinal fibrosis. This type of a followup will take a number of years, but we hope to have the information that Drs. Beekman and Weled are asking for.

We have no proof of our supposition that mediastinal fibrosis is caused by rupture of fibrocaseous material from mediastinal lymph nodes into the mediastinum, but we feel that in the patient who presents with right-sided paratracheal nodes mediastinoscopy should not be carried out, but rather thoracotomy with biopsy of the node to exclude lymphoma and then surgical removal of the mediastinal granuloma to prevent the potential complication of fibrosing mediastinitis.

We hope that in the years to come we will have data to support this.

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