hinges on an effective combined drug regimen administered without the dehumanizing effects of most isolation procedures, with return to normal life as early as possible. This in effect means that tuberculosis is merging into the mainstream of medicine where it now rightly belongs, with most of the treatment given on an ambulatory basis.

Tuberculosis today is preventable. Persons at risk of developing tuberculosis include close contacts of newly diagnosed "transmitters," recently infected persons as indicated by conversion of the tuberculin reaction from negative to positive, and longtime tuberculin positive reactors, especially those with an abnormal shadow on the chest roentgenogram or some medical condition which shifts the balance between the tubercle bacillus and its human host. Treatment with isoniazid for one year can reduce the risk of developing active disease for these people.

The present picture of tuberculosis is thus one of a preventable disease which, when it develops, responds promptly and dramatically to chemotherapy. Sick patients, and smear positive patients who are likely transmitters of tubercle bacilli, generally need a short period of hospital care to establish an effective treatment regimen or to provide temporary isolation. After the diagnosis is made, an acceptable treatment plan must be worked out between the patient and the physician or health agency to assure continued therapy, without interruption, for the full course of treatment as prescribed. This is the key to successful treatment—making it easy for the patient to stay on medications for up to two years. "Local experience," writes Curry, "has been that when 'patient problems' are resolved, 'problem patients' disappear."

Phyllis Q. Edwards, M.D.*
Atlanta

*Chief, Tuberculosis Branch, Center for Disease Control.

"Cine" Qua Non

Coronary arteriography is a valuable and at times indispensable technique for the evaluation of patients with coronary (ischemic) heart disease. Nevertheless, as the number of coronary arteriograms performed increases it would be expected that discrepancies between the clinical and angiographic findings might occur with increasing frequency, and that such discrepancies will be of growing concern to the clinician.

A report in this issue of Chest (see page 618) illustrates a problem that is likely to become increasingly more common. A 52-year-old laborer with unmistakable clinical evidence of acute myocardial infarction applied for disability compensation because he could not find a less strenuous job as advised by his physician. The clinical data were reviewed by the medical director of a large insurance company who refused to award disability compensation until a coronary arteriogram was performed. Thus, in spite of prodromal symptoms of angina pectoris, an abnormal electrocardiogram for four months, the sudden development of left bundle branch block in association with severe retrosternal pain and serial elevations in the serum concentrations of GOT and CPK, the determination of disability depended upon the coronary cineangiogram. In effect, the coronary arteriogram became the sine qua non for determination of coronary heart disease. If such an attitude reflects a trend, it is likely that serious clinical errors will be made in the future.

In a recent report of the coronary arteriogram in patients with left bundle branch block, it was stated that "only four of our 19 patients who complained of chest pain suggestive of angina were found to have coronary artery disease."1 Thus, the diagnosis of coronary artery disease depended entirely on the coronary arteriogram—exclusive of other clinical information. Our purpose in citing the quotation above is not to criticize a particular study but to illustrate a general trend. Many statements of similar content can be found in the current medical literature. Nevertheless, it is disturbing that lessons learned in over a century of clinical and experimental research have been dismissed so quickly in favor of a technique that has only recently been applied to large numbers of patients and has been correlated with autopsy findings in relatively few instances.

Until recently coronary arteriography has been performed primarily in large centers and in patients with clinical findings sufficient to warrant referral to a medical center. During the past several years proliferation of facilities for coronary arteriography, the early encouraging results of aortocoronary bypass surgery, and the introduction of the Judkins technique2 have resulted in an increase in the number of patients undergoing coronary arteriography. It may be no coincidence that both the number of coronary arteriograms performed and concern over normal coronary arteriograms in patients with angina pectoris or myocardial necrosis have increased in parallel.

Whenever the problem of normal coronary arteriograms in patients with angina pectoris or acute myocardial infarction is discussed, the explanation for the normal arteriogram usually in-
Inapparent Signals to Demand Pacemakers

The evaluation of pacemaker function may become quite perplexing when a demand (ventricular-inhibited) pulse generator senses intracorporal voltages invisible on the standard 12-lead electrocardiogram. Such inapparent signals may originate from the following sources: the pacemaker system itself, concealed ventricular extrasystoles, and skeletal muscle potentials.

False signals from the pacemaker system: Abrupt changes in resistance within a pacing system produce corresponding voltage changes between the anode and the cathode and may generate relatively large signals capable of being sensed by a demand pulse generator. Two basic mechanisms are involved: (1) interruption of the polarization voltage or "after potential" that follows delivery of the pacemaker pulse, and (2) disturbance of the small permanent DC voltage across the electrodes in the absence of pacemaker stimuli. These mechanisms are responsible for false signals seen with intermittent derangement of a pacemaker circuit by loose connections, insulation breaks, short circuits and the momentary separation of the well apposed ends of a fractured electrode. The interaction of two pacing catheters within the heart may also generate false signals when functional and inactive electrodes lying side by side make intermittent contact. Moving the test magnet towards most demand pulse generators creates a false signal because closure of the magnetic reed switch shorts the input into the sensing amplifier, thereby causing a sudden change in voltage. Removal of the magnet produces a false signal by the same mechanism.

Sensing of a false signal may stimulate T wave sensing and the following points should be considered in the differential diagnosis: (1) false signals tend to occur at random and often much later than the T wave, (2) T wave sensing by temporary pacemakers can always be eliminated by slight diminution of the input sensitivity while this is often impossible with false signals because of their relatively large magnitude, (3) long pauses without pacemaker stimuli or spontaneous beats excludes T wave sensing. However, when a catheter with a defective tip induces rhythmic recycling of a demand pacemaker because mechanical systole consistently creates a false signal by making and breaking the circuit, the abnormality may be electrocardiographically indistinguishable from T wave sensing because of its timing and regularity.

Concealed ventricular extrasystoles: Massumi et al recently described an unusual demand pacemaker arrhythmia they attributed to sensing of concealed ventricular extrasystoles (depolarization confined to the His Purkinje system invisible on the standard 12-lead electrocardiogram). This attractive and rather intriguing possibility must, however, remain speculative in the absence of direct recordings from the pacing catheter. Until more definitive proof becomes available, sensing of the T wave of paced and spontaneous ventricular beats associated with complete and partial recycling of the demand pulse generator would appear a plausible alternative explanation for the interesting findings of Massumi et al. Thus, in their reported case, the postextrasystolic T wave phenomenon might have been responsible for amplification of the T wave signal of the paced ventricular beats immediately after visible ventricular extrasystoles while the ensuing pacemaker bradycardia probably perpetuated these T wave changes until the occurrence of another visible ventricular extrasystole. Eradication of visible ventricular extrasystoles rather than concealed ones by the administration of lidocaine (Xylocaine) and later quinidine would then simply account for disappearance of the pacemaker arrhythmia in this particular report. A more direct effect of Xylocaine could also be