The sensing circuit can be visualized as shown in Figure 1, where $R_s$ is the source (tissue-electrode) resistance (normally about 500 ohms), $R_L$ is the lead resistance (normally about 50 ohms), $R_b$ is the break resistance, $R_a$ is the pacemaker sense amplifier resistance (normally about 22,000 ohms), $e_s$ is the source (intrinsic cardiac) R-wave voltage (assume 10 mv), and $e_o$ is the R-wave voltage at the sense amplifier.

The resistor chain acts as a voltage divider, as follows:

$$e_o = e_s \left( \frac{R_a}{R_s + R_L + R_b + R_a} \right)$$

Without the break, $R_b$ equals 0 ohms, and the voltage at the pacemaker ($e_o$) is 9.76 mv. With the break, choose $R_b$ equal to 5,000 ohms for the saline bridge. Then the voltage at the pacemaker is only 7.98 mv. Since the usual sensing threshold of pacemakers is about 2 mv, the R-wave would be sensed in any case; however, the statement that the break “should enhance sensing” is clearly not supported.

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To the Editor:

The comments of Reis are appreciated, and we are basically in agreement. Indeed, it is only with one of the presently available pacing system analyzers that measurement of the stimulation impedance can be made, because one needs to know both the current and voltage to make this calculation.

When we stated that there was an “intact pathway within the fractured electrode,” we implied that the pathway for the electrons was comprised of the wire itself and postulated that an electrolytic solution of the body fluids bridged the break in the wire. Indeed, the theoretical calculations by Reis support our hypothesis that the signal registered by the pacemaker will be greater with an increase in resistance between the tip of the electrode and the indifferent electrode on the pacemaker itself.

Some comments ought to be addressed to the sensing characteristics of most pacemakers, as this question is implied by the letter from Reis. While most clinicians think of a pacemaker as sensing a given signal, usually around 2 mv, and, indeed, the manufacturers' specifications record sensitivity in terms of millivolts, this is a vast oversimplification. In order to minimize oversensing problems, such as sensing of T waves or extraneous signals, the manufacturers have incorporated band pass filters to look at only a narrow frequency spectrum and slew-rate-dependent measurements in addition to amplitude criteria. Thus, a 50-Hz signal which might be at the most sensitive area of the band pass filter would require a 1-mv amplitude to activate the sensing circuit; a 10-Hz signal, very similar to most T waves, might require a 12-mv signal in order to be sensed. The manufacturer must use an arbitrary testing signal in order to check their pacemakers, as there is no standard QRS complex available in the laboratory. Their sensitivity refers to the signal used, which is usually indicated at the bottom of the specification sheet that comes with each pacemaker; unfortunately, it is not the same testing signal for each pacemaker. Hence, the sensitivity of one manufacturer's pacemaker cannot be readily compared to that of another by simply comparing the specification sheets, and its relationship to a QRS complex is only an approximation.

Our comment that the fractured electrode with an intact pathway caused by the bridging of the electrolytic solution should enhance sensing refers only to the pacemaker's ability to see a slightly larger signal in this setting. If the signal were of borderline amplitude originally, then sensing would clearly be enhanced. We fully agree that one cannot further improve upon already normal sensing.

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Granular Cell Myoblastoma of the Bronchus
A Benign Lesion with a Potential for Rapid Growth

To the Editor:

Granular cell myoblastoma of the bronchus (GCM) is a relatively rare tumor. Only 46 cases of solitary and 2 cases of multiple lesions of the tracheobronchial tree have been described in literature. We had the opportunity to observe a patient suffering from GCM for approximately one year before she consented to surgery.

Case Report

A 32-year-old obese black woman was admitted to Mary Thompson Hospital because of persistent productive cough and recurrent attacks of lung infections with fever, dyspnea and chest pain. Chest x-ray films revealed right lower lobe atelectasis. Fiberoptic bronchoscopy was performed on October 7, 1976, and revealed a patent but irregular lumen of the right lower lobe bronchus, with hypertrophy of the mucosa in a polypoid cauliflower fashion all around. Biopsy and brushing failed to yield a diagnosis. The patient refused surgery.

On April 24, 1977, she was readmitted because of progression of her symptoms. Plain chest x-ray films and tomograms this time revealed a mass 4.5 × 4 × 3 cm in the right lower