anesthetized dogs that the Aero-Flo design minimizes damage to the bronchial mucosa after repeated suctioning procedures, while the single-eyed and two-eyed types do not.²

I have another objection to the study from a technical standpoint. Jung and Gottlieb state that the catheters were placed into the right or left bronchus; a chest roentgenogram with the catheter at the orifice of the left bronchus is depicted in their report. As previously reported, most of the damage associated with suctioning in intubated patients is found in the right lower bronchus.³ In animal experiments, Sackner et al² simulated this condition by directing the catheters into lobar bronchi. Clearly, if one directs a catheter into a large airway for a brief period with interrupted suction, no damage will be observed with any type of catheter, unless the catheter tip is in close proximity to the mucosa. Regardless of the catheter design, there will be less damage in larger airways as a function of the number of times suctioned, compared to smaller airways. Here again, Jung and Gottlieb have not designed their study to test equivalent clinical conditions.

The comparisons of damage produced by the various catheters, as listed in the "Results" section of the report by Jung and Gottlieb, are sketchy and difficult to assess scientifically. It would have been better to give the site of suctioning, the number of trials, the frequency of mucosa invaginated into the catheters, and the frequency of occurrence of petechial hemorrhages with each catheter. Control studies should have been performed using only the fiberoptic bronchoscope, which itself can cause damage when vacuum is applied to the eyes of this catheter.

We would agree with Jung and Gottlieb that short bursts of interrupted suction are an aid to minimizing mucosal trauma. We believe that additional minimization of such damage is achieved with the Aero-Flo tip, rather than conventional catheters, because the Aero-Flo design tends to prevent contact of the mucosa with the eyes of this catheter.

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REFERENCES

To the Editor:

We welcome the comments of Dr. Sackner in evaluating our article entitled "Comparison of Tracheobronchial Suction Catheters in Humans: Visualization by Fiberoptic Bronchoscopy" (Chest 69:179-181, 1976); however, we believe that some of his comments are inappropriate.

First, in the design of our study, we fully recognized that it would be virtually impossible to duplicate the usual bedside clinical situation, wherein repeated suctioning in patients has occurred over many days and the airway has been violated not only by the use of ventilators but also by normal airway contaminants in a usual hospital environment, all of which would tend to add to the insult of interrupting the natural upper airway defenses with an artificial airway. Therefore, our study was designed only to test the hypothesis that a single suctioning procedure as usually performed is more important in creating damage to the mucosa than the particular design of any currently available catheter.

We fully concur that the administration of atropine reduces mucociliary clearance; however, it is difficult for us to see an association between this effect and damage to the mucosa from tracheobronchial suctioning.

We disagree that the intermittent single suctioning procedure which we employed is any different from that seen in the clinical setting. Certainly, we carefully instruct our nursing and therapy personnel that the suctioning procedure should be no more than 10 or 15 seconds at any time and that it should be intermittent.

We also believe that it would be difficult to fully duplicate the animal study of Sackner et al in a human situation. We only intended to duplicate the current usual practice in the human situation. We take no issue with the findings of Sackner et al in their animal preparations.

Dr. Sackner goes on to criticize the fact that in our Figure 2 the catheter was shown to be in the left main bronchus. This roentgenogram illustrated only one placement of the catheter. We, too, are aware that the majority of damage occurs in the right, rather than the left, bronchus; however, as was carefully pointed out by us, we used one bronchus against the other in the same patient employing two different catheters when we assessed that the gross appearance before suctioning was similar in both bronchi.

We take issue with the animal experiments of Sackner et al where they tested the effects of the catheters in lobar bronchi. It would be exceedingly rare in a bedside situation that a catheter would pass far beyond the main bronchi. Therefore, it seemed to us logical that we should test the effect of the catheters in humans in the right and left main bronchi, rather than passing them deeper into any individual lobar bronchus. Certainly these larger airways are going to be damaged less. This was not an issue with us. What we wanted to assess was how much damage was occurring in the main bronchi.

We also concur that during suctioning through the inner channel of a bronchoscope, very similar results
might occur. We were not interested in the effect of the bronchoscope itself; we were interested in the effects of the catheters.

Finally, we can only reiterate what we saw in these patients via cine as well as still photography; namely, we saw invagination of mucosa into the side holes of all catheters tested. We would only suggest that "a picture is worth a thousand words." Unfortunately, limitations in cost prevented us from illustrating in our article the pictures we recorded with cine and still photography.

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REFERENCE

Pacemaker-Induced Ventricular Fibrillation

To the Editor:

In their communication entitled "Ventricular Fibrillation Induced by a Defective Demand Pacemaker" (Chest 69:247-249, 1976), Chirife et al described a runaway pacemaker which achieved a stimulation rate of "2,100 beats per minute" and finally induced ventricular fibrillation in their patient. I would be reluctant to accept their conclusion without some confirmation of this rate besides their illustration. Was the rate confirmed after the pacemaker had been removed from the patient? Even though the diagnosis of a runaway pacemaker seems incontrovertible in strips 1 and 2 and the first part of strip 3 in the illustration of Chirife et al, thereafter what is alleged to be very rapid pacemaker firing could just as easily be due to artifact. Electrocardiographic artifacts may occur at rapid rates and yet not be due to 60-cycle electrical interference. An example of this phenomenon was recently recorded in the Coronary Care Unit of Pascack Valley Hospital, Westwood, NJ, from a patient in regular sinus rhythm without a pacemaker but with poor electrical contact by one of his monitor electrodes (Fig 1). Our Figure 1 resembles strip 4 in the illustration of Chirife et al. May I suggest that ventricular fibrillation in the patient of Chirife et al may have been independent of stimulation by the runaway pacemaker? For example, their strip 3 shows complete heart block with an unstable escape rhythm and apparently mostly P waves following cessation of capture by the pacemaker. Strip 4 of their illustration again shows complete heart block. The occurrence of ventricular fibrillation in this setting would not be unexpected even in the absence of a runaway pacemaker.

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REFERENCE

To the Editor:

This is in answer to the letter of Leonard J. Lyon, M.D., in reference to our communication entitled, "Ventricular Fibrillation Induced by a Defective Demand Pacemaker" (Chest 69:247-249, 1976). In response to Dr. Lyon's first question, the pacemaker's excessive firing rate was confirmed after removal by oscilloscopic display of the signal, which revealed 40 to 50 impulses per second, slightly faster than the frequencies observed in strip 4 of our Figure 1. This type of pacemaker generator does not have a runaway protection (as new designs do), and in the presence of an electronic component malfunction, they can certainly reach these firing frequencies. Poor electrical contact of the electrocardiographic electrodes in this case was obviously not the cause of the tracings obtained.

In his last statement, Dr. Lyon appears to give more weight to the patient's bradycardia (about 50 beats per minute) than to a very rapid ventricular stimulation rate as the cause of ventricular fibrillation. Since very fast rates were clearly demonstrated in our communication and since a similar condition of pacemaker-induced ventricular fibrillation has been reported in the literature,² we strongly reiterate the high likelihood of a cause-effect relationship in our case.

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REFERENCES

Figure 1. Artifacts caused by loose electrode, resembling runaway pacemaker at 500 impulses per minute.