Three of those cases were operated on to relieve the dyspnœa and distress, where the malignancy and sepsis were so manifest as to forbid hope; three died of sepsis, two died of heart failure, nine died of extension of the exudative disease into the bronchi Of these nine, five gave good promises of recovery for two days after the tube was inserted. The other four were of such malignancy that hope was not entertained.

The doctor dwelt on the impossibility of diagnosing the condition of the lungs in these cases of extreme dyspnœa, the quantity of air entering the lungs is so small, that chest sounds are inappreciable, and therefore it is impossible to know the condition of the lungs before operation, a fact which stands against tracheotomy; for if the lungs were known to be already involved, so serious an operation as tracheotomy would not be performed, whereas the simple bloodless operation of intubation would still be advisable in view of the relief it would afford.

After the tube is introduced the bronchi are found in those cases where extreme dyspnœa had continued for some considerable time, to be more or less obstructed with mucus, due, doubtless, to venous engorgement of lungs and bronchi, from continued stenosis. These mucous rales soon disappear after air is freely admitted and respiration exhibits the true normal vesicular murmur or such gradations from this as furnish fair evidence for prognosis.

In certain cases after the tube was introduced the patient was bright, respiration free, and normal vesicular, with this exception, that it seemed metallic or slightly hard, or had lost some of its soft breezy quality. No rales; no dullness.

In all these cases a fatal result followed in from two to five days. He believed this quality of respiratory murmur, due to the incipient stage of the exudative process in the bronchi. They had not yetthrown out the exudate, but the mucous membrane was swollen, thus lessening the lumen of the tubes and changing the quality of the sounds. With this symptom present, the prognosis is bad, even though the patient is bright and otherwise hopeful.

In another class of cases where the dyspnœa was extreme, the chest walls were found *stationary, no recession, no movement of the larynx* to and fro, as is characteristic of pure laryngeal stenosis. Here the stethoscope reveals, after the tube is introduced, coarse and fine bronchial rales, and a generally emphysematous condition of the lungs.

In these cases where the disease extends to the bronchi the large plugs of mucus formed, and portions of exudation that become more easily detached in the bronchi, prove factors in developing rapidly an acute emphysema. This combination of conditions make the lungs too large for the space they have to occupy, and thus prevent the recession of the chest walls.

This condition being also a cause of part of the dyspnœa, the extensive movements of the larynx, so manifest when the dyspnœa is due to stenosis of the larynx alone, are consequently very much reduced.

When extreme dyspnœa is due to stenosis of the larynx alone, we find movement and recession of chest walls and extensive movements of the larynx up and down. If, therefore, in extreme dyspnœa, the chest walls are more or less *motionless*, with *no recession* and the larynx quiet, we may be sure of extensive exudation into, and engorgement of the lungs, with possibly acute emphysema. This symptom is of value, as the stethoscope, in this extreme dyspnœa reveals nothing. Tracheotomy here would be worse than useless. Intubation would give merely temporary relief.

The sources of danger ascribed to intubation are:

1. Apnœa and laceration of tissue by prolonged efforts at introduction.

2. Forcing down the tube in efforts at removal and injury to the parts. (None of which should ever happen in the hands of an expert.)

3. Interference with deglutition and nourishment. (This may be overcome with jellied foods, soft egg, custards, etc.)

4. Occlusion of the tube and trachea by pushing down the trachea before it. (This is overcome by immediately withdrawing the tube when the false membrane will follow it.)

5. Ulceration of trachea by pressure of the tube and consequent sepsis and necrosis. (This must be very rare indeed, as the tubes do not press upon the trachea.)

6. Traumatic pneumonia from passage of food into respiratory tract. (Avoided by using food in some solid form.)

7. Coughing up the tube and swallowing it. (No objection to this but the temporary loss of the tube.)