

denser condition of that lung. As the case progressed and the condition of the patient improved, this sign also gradually diminished, although it has not entirely disappeared. I regard it as an unequivocal evidence of more or less solidification. Thus, in this case, there have been two signs of pulmonary condensation—one caused by the compression of lung tissue by the organs of circulation, the other by compression of the organs of circulation by the lung tissue.

CASE OF DIPHTHERIA, WITH TRACHEOTOMY. RECOVERY.

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WEDNESDAY, May 27th, 1863, was called, in consultation, to a case of diphtheria. The case presented the usual, ordinary symptoms. The tonsils and posterior portion of the fauces were covered with yellowish-white membrane; the anterior and upper portions of the throat intensely red. A mixture of chlorate of potassa, tincture of muriate of iron and muriatic acid was at once applied, and also administered internally. The voice was nearly gone, and the cough and breathing croupy.

28th, 29th, 30th.—The upper part of the throat appeared to improve, but there was a gradual increase of the croupal symptoms, and from the great obstruction to breathing, shown by the livid appearance, it was evident that the child was dying. There was considerable enlargement of the glands about the neck, a part of which had existed since an attack of scarlet fever, about a year ago. The glandular enlargement finally suppurated and discharged through the mouth. Saturday morning, seeing the child was sinking, I performed the operation of tracheotomy, it being evident that life could not continue much longer, so urgent were the symptoms.

The relief was very marked, and half an hour after the child's countenance assumed quite a rosy appearance, and there was free expectoration through the tube of membranous shreds and mucus. Before the afternoon expired, we felt very hopeful of success. A tonic course of quinine, iron and wine was liberally and regularly administered, with a generous diet. During the night the child slept somewhat. On the morning of the 31st he seemed quite comfortable, breathing entirely through the tube. The throat rapidly improved until the 5th of June, when bloody expectoration and bronchial irritation increased, and an erysipelatous inflammation showed itself and spread nearly to the point of the shoulder and down on the chest.

June 6th.—Fearing the extending irritation, the canula was removed. The parts were moulded to the canula, and a perfect opening was left through which the child breathed and expectorated freely. On covering the opening, symptoms of suffocation were in-

duced; the canula was not replaced, however, to avoid all possible bronchial irritation. Great care was exercised to keep the opening clear of all obstructions until the natural breathing was gradually restored.

The canula was large and double, and kept constantly clean. Anodynes were administered occasionally, to allay restlessness. The case improved, without any serious disturbance, and on the fourteenth day after the operation the patient could speak aloud. On the 17th, the opening had entirely closed, and the patient was constantly gaining in strength and flesh. At present, the 24th, he has entirely recovered.

ON THE DIAGNOSTIC VALUE OF AN ACCENTUATED CARDIAC SECOND SOUND.

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It is now universally admitted that the second sound of the heart is produced during the act of closure of the semilunar valves in the orifices of the aorta and pulmonary artery. The sudden tension of the membranous structure of which these valves are composed is a sufficient, possibly the *only*, cause of the sound. It is, however, probable that, as generally held, the recoil of the blood against the valves contributes to its production.

Careful clinical observation has materially aided the direct experiments which, at a former period, were made regarding the heart's sounds. As respects the second sound, it may indeed be concluded that, by the former means of research, much left unfulfilled by the latter has been supplied. For example, I may refer to one or two particulars of importance, which have a special bearing on the subject of this paper. When the second sound is entirely replaced over the base of the heart by a murmur, it is not audible over the ventricles, and is not to be detected at the apex of the organ. In other words, when the murmur of aortic regurgitation is so loud as to drown all normal second sound, preventing, by its very loudness, the recognition of the pulmonary second sound over the pulmonary valves, or in their immediate neighborhood, where the second sound originating there is most readily heard, it cannot be detected even in small part over any other portion of the heart. Here, however, there are some points worthy of special observation; they concern what may be called exceptional cases of aortic insufficiency. A diastolic murmur may largely obscure, but not obliterate, the second sound over the aortic valves, a portion of it remains; and in such cases, the pulmonary second sound being either readily appreciable or at least audible, the murmur diminishing in loudness as the stethoscope is placed over the ventricles, the normal second sound, pulmonary in origin, or the portion of aortic second sound which remains, is