

Notwithstanding the conviction of the experienced that this hemorrhage rarely proceeds so far as to produce immediate death, yet, remembering that the blood is life, and to see that life, as it were, gushing from both mouth and nostrils, and that, too, from a being already chlorotic from a wasting disease in a vital organ, is sufficient to disturb the equanimity of the most deliberate.

This loss of blood may arise from various causes, and at various periods of health and disease.

The causes may be conveniently, and to a certain extent truly, divided into two classes,—those external to the body, and those internal, or arising as a result of a diseased condition. Of the first, we have mechanical injuries to the chest, such as penetrating wounds. During the late war of the rebellion this was not an uncommon event in the experience of many of us. So also from fracture of the ribs, spiculae of bone penetrating the lung-tissue, the inhalation of caustic fumes and particles of irritating dust, as in the case of file-makers or those employed in grinding and polishing metals. It frequently results from excessive physical exertion, as in straining, or lifting heavy bodies. The forced retention of air in the lungs whilst the abdominal and thoracic muscles are rigidly contracting will, in many cases, produce either an emphysematous condition or a complete laceration of the viscus with its contained plexuses of arteries and veins.

Of the second class, catarrh may be named as occasionally causing hemorrhage; though the uncomplicated attacks usually traceable to this cause are of so slight a character that we are forced to the conclusion that the bleeding is from the vessels on the larger bronchial surfaces, and not from the lungs proper.

Another very fruitful cause of this hemorrhage, and one frequently not recognized, is disease of the left side of the heart. The defective mitral valve, with its indurated, contracted, and weakened segments, no longer protects the delicate lung-structures against the powerful contractions of the left ventricle, and, as a consequence, regurgitation takes place, thus keeping up a constant and forced dilatation of the pulmonary vessels. These sooner or later give way, producing the most annoying and obstinate attacks of hæmoptysis with which we meet. Indeed, there is no more unfortunate complication with which the consumptive can be afflicted than this same disease of the heart.

We have also what are called vicarious hemorrhages,—those following the interruption of habitual discharges, such as hæmorrhoidal or menstrual. They are occasionally serious, yet by that remarkable law of accommodation in nature the vessels soon become reconciled to the excess of blood, and it flows on submissively in the natural channels. Notwithstanding the facility with which we can thus rationally account for almost every form of hæmoptysis, we usually associate its occurrence with an internal and far more grave disorder than any of which we have as yet spoken,—viz. the incipency or full-fledged existence of consumption.

Authorities differ widely in regard to the rela-

tionship existing between this hemorrhage and the presence of tubercles in the lungs. Laenec, Louis, Rokitsansky, Watson, Williams, and many of our own writers, associate the two as almost invariably dependent the one upon the other. Either the hemorrhage, by its subsequent clots, creates a local irritation, congestion, and inflammation, thus clogging the channels of nutrition, the lymphatic glands degenerate and are converted into tubercles, or the whole mass becomes cretaceous phthisis; or, on the other hand, fully-developed and softening tubercles by the ulcerative process open a pulmonary vessel and thus act as a direct cause of hemorrhage. Though there may be valid reasons for rejecting hæmoptysis as pathognomonic of consumption in the early stage, yet in the latter it becomes a direct result, pointing to softening and actual destruction of lung-substance. There are, undoubtedly, numerous instances in which hemorrhage has been frequent and copious without terminating in disease; yet it is a well-established pathological fact that in cases of tubercular cachexia there exists a fragile state of the blood-vessels not usually found in other diseases. This degenerating tendency, so characteristic of consumption, greatly favors, and is the only way of accounting for, this bleeding in the early stage, when the physical signs do not as yet indicate disease. We have often watched, and with a degree of certainty predicted, hemorrhage in the early stages of phthisis. The history and symptoms of such a case would be about as follows. An irritating, short, and dry cough, traceable to a slight cold contracted a few days previously; great oppression, difficulty in breathing, loss of appetite, thirst, continued fever; pulse rarely under 100; temperature from 100° to 104°; expectoration very slight, and of a saltish taste. Percussion-signs at first almost nil. Auscultation would reveal dry tubular sounds over a part of one or both lungs. Respiration rapid, with *prolonged* expiration. These symptoms would gradually become aggravated, continuing for several days, when, after an unusually severe attack of coughing, hemorrhage would commence. The relief following this occurrence sustains our explanation,—viz., the existence in the first place of tubercular deposits to an extent not as yet recognizable by physical signs nor incompatible with the normal functions of the lungs. But the catarrh awakens the fire; the flames spread rapidly to the miliary centres already existing, and upon the principle of *ubi irritatio ibi affluens*, an active congestion results, and the degenerated and enfeebled blood-vessels, unable to endure the pressure, give way.

The main cause, however, in the advanced stage, and the one resulting in the most copious loss of blood, is the dilatation and thinning of the pulmonary blood-vessels. By some authors it has been named ectasis, or aneurism; but to the sight and touch in the cadaver it resembles the varicose condition often found in the veins of the lower extremities. In the process of softening and conversion of the tubercular or adenoid substance into pus, cavities are formed, through or on the walls of which the pulmonary vessels pass; by the loss of structure