one portion of the respiratory centre it is stimulating another; and a similar double character is attributed to the action of venous blood. Thus during the brief time from the beginning of apnœa to the culmination of dyspnœa—a period rarely exceeding one minute—the blood passing to the brain is called upon to exert four different and even diverse effects; first as venous blood stimulating one part of the respiratory centre and paralyzing another portion of the same centre; reverse effects being produced by the same blood on its becoming oxygenated. One is really at a loss to understand how such an explanation could have been admitted to a place in physiological literature. Again it is the exigences of an erroneous theory which have led to such a complicated and unsatisfactory hypothesis. If it be asked how the state of apnœa is induced by forced vigorous respirations, if it be not due to an excess of oxygen introduced into the blood, and how the opposite condition or demand for air by breathing seems to attend the absence of oxygen and the presence of venous blood, I can only answer as to the last that if no better explanation than that venous blood is a stimulant has yet been found, some better explanation is surely to be looked for. And as to the state of apnœa referred to, I find Dr. Austin Flint stating that "according to Hoppe-Seyler, apnœa, in the limited sense above mentioned, is to be attributed, not to an excess of oxygen in the blood, but to fatigue of the respiratory muscles." (a)

A NEW THEORY SUGGESTED.

Dr. Sansom regards the condition of the respiratory centre in this case as one of paresis and direct exhaustion. Hé shows that during the apnœal period "the arteries are strongly contracted." The proof of this is found in the rise of arterial tension in the depression of the "great fontanelle" of the head, and also in the arrest of the process by the inhalation of nitrite of amyl, which dilates the arteries. On the theory of these pages, arterial contraction is due to vasomotor nerve depression or paralysis; and accordingly we find here that the vasomotor centre, as well as the respiratory centre, is depressed in function. It has been amply shown above, that contraction of the arteries occurs in the dying and is complete in death. It is also one of the prominent phenomena during the last stages of asphyxia and is invariably attended by venous fullness. The condition present during the stage of apnœa in the Cheyne-Stokes respiration, with its contracted arteries and dilated veins, appears to correspond very closely to that present as death approaches and in the latter stages of asphyxia. The original paretic and exhausted condition of the respiratory and vasomotor centres is aggravated by the further depression caused by mal-oxygenation of the blood; which, when venous and loaded with carbonic acid, is invariably a depressing, and never a stimulating agent to nerve function. Vasomotor nerve failure induces contraction of the arterioles, systemic emptiness and venous engorgement, as the foregoing examples abundantly prove; and as a consequence, the great mass of blood "becomes lodged and hidden as it were" in the great venous trunks. At that moment death is very near, but as the heart continues to beat, it is fair to assume that a small quantity of blood still finds its way through the lungs, and, from its very scantiness, is capable of being ærated by means of the exchanges of gases still going on in the lungs, owing to the presence of residual air during the temporary, partial or complete arrest of respiration. As a consequence the quantity of blood reaching the nerve centres, though small, is at least partially oxygenated, and serves to revive the function of these centres "imperfectly at first," but with momentary improvement. The effect of this revival on the vasomotor centre, is to facilitate the dilation of the arterioles; in which the pulmonary vessels share, permitting, ere long, the inrush of venous blood from the distended vena cava and portal system, and its transmission onwards through the heart and lungs. This corresponds to the period of increase in respiratory function, in which the laborious efforts of a feeble mechanism have been mistaken for an "exaggerated impulse" from excited and overacting or "exploding" nerve centres. Meanwhile, impure blood from the venous reservoirs (finding an entrance through the now fairly dilated pulmonary vessels) begins to fill the lungs in such a quantity (as it is drawn onwards by an inequality of pressure, towards the as yet unfilled arteries) that the whole mass of blood, failing to be arterialized with sufficient rapidity, again becomes unfit for the

(a) Prac. of Med., 5th Ed., p. 70.