Heart," (*a*) by whom Traube's theory on this subject is said to be "the most plausible." According to Traube, "the first thing which occurs is the establishment of a condition of impaired irritability of the respiratory centre through mal-oxygenation; the long respiratory arrest gives time for the accumulation of carbonic acid in excess in the blood. Arrived at a certain maximum this begins to stimulate, slowly and imperfectly at first and afterwards in increasing degrees, the centre, so that it develops the respiratory efforts till they culminate in dyspnea. Then as the centre ceases to be stimulated or becomes exhausted, dyspnœa again supervenes."

It will be observed that here the *deficiency* of oxygen and subsequently the *presence* of carbonic acid are made to play opposite and antagonistic parts! The lack of oxygen (instead of stimulating the medulla, as supposed by Dr. M. Foster) first enfeebles the respiratory centre, in the medulla, and then the same blood, still deficient in oxygen, but now loaded with carbonic acid, counteracts the previous depression, and tones up the weak nerve centre, so that ere long it displays extraordinary activity. But, unfortunately, this exhilarating pabulum—carbonic acid—is soon exhausted, and the nerve centre resumes its former feebleness till a new supply can be procured. The physiologist is certainly quite impartial, and allows the rivals to have their "innings," turn about. How such nonsense as this "most plausible theory" could find a place in physiological literature seems explicable only on the exigency of the hypothesis so long in vogue.

Filehne's theory in explanation of this state is more complicated, and at least equally absurd. Instead of the respiratory centre being stimulated (as Traube says), it is the vasomotor centre which is excited by the presence of carbonic acid. Arterial contraction follows till "a gradually increasing anemia of the respiratory centre" is brought about. This anemic condition excites the respiratory centre "and inspiration becomes more and more deep," till oxygen is supplied to the blood; "the arterial spasm is thus relieved," owing to the freshly oxygenated blood failing to stimulate the vasomotor centre (so as to contract the arteries), as the carbonic acid had previously done. With the relief of arterial spasm, and a consequent normal dilation of the arteries, "the anemia of the respiratory centre passes off, and with it the exaggerated impulse to respiration, and breathing once more becomes superficial." (*b*) In other words the respiratory centre functionates best when it is supplied not only with non-arterialized blood, but when it has too little even of that; as soon as the anemia passes off, and this nervous centre gets a fair supply of blood, it ceases to act—suspends business—till the better times of bad blood and deficient blood come round again, when it is moved to activity once more!

There is still another explanatory theory to be noticed, which I find referred to editorially in the CANADA LANCET for February, 1886: "Bramwell, who follows the teaching of M. Foster and others, supposes that the respiratory centre consists of two portions, one accelerating (or motor), and one inhibitory. He further believes that these two portions are acted on in opposite directions by the blood, whether arterial or venous. Thus while venous blood stimulates the discharging cells of the centre and depresses the inhibitory portion, arterial blood acts in exactly the opposite direction." At the close of the period of apnœa, the discharging portion of the centre is stimulated by the venous blood," with its excess of carbonic acid, and this same blood, at the same time is depressing the rival, or inhibitorypart of the centre. The motor or discharging portion of the centre triumphs; respiration becomes established and even exaggerated. Unhappily, the victor fails to "hold the fort." As soon as the blood becomes "fully oxygenated," the "inhibitory portion becomes stimulated and gradually overpowers the discharging portion," so that "the respirations grow weaker and weaker until the state of apnœa results." Then the suspension of breathing restores the venous character of the blood and accumulates a store of carbonic acid, the stimulation of which reanimates the centre previously depressed by the presence of oxygen in the blood. Such appears to be the scope of this theory.

In this, as in the previous explanations, arterial blood is made to play the part of a depressor and paralyzer of the respiratory process, which it is constantly tending to arrest; but while paralyzing

(*a*) P. 37.

(*b*) P. 137.