

excess while there is not a corresponding increase in thermolysis, the temperature must rise: if thermogenesis be normal and thermolysis from any cause be inadequate, the temperature must also rise. If the cause disturbing the harmony between thermogenesis and thermolysis be so potent that compensation is no longer possible, fatal hyperpyrexia results. The agent that deranges any or all of these processes may be anything acting directly on the nervous centres that control them, or indirectly on them through the blood. We know of a great variety of poisons, that act in the latter way, notably such as have been found in the eruptive fevers and in malarial fever.

Clinical observation of fever constantly impels one to seek an explanation of its manifestations, and it is more with the view of receiving than of imparting information that I mention some difficulties that have perplexed me. Every physician is familiar with the influence of fever on the respiration, especially in children. Sixty, eighty, or even ninety respirations a minute are not uncommon in children under a year old when suffering from a temperature of 104° or 105° F., and this when the respiratory organs are free from disease. It cannot be due directly to the poison causing the fever because a rapid reduction of the temperature by means of the cold bath promptly reduces the respirations often to the normal rate, and yet the cause of the rise of temperature is not removed by the bath. Doubtless the rapid breathing promotes heat loss, and is in this way conservative. Obstetricians are familiar with the beneficial influence of hot-water in the resuscitation of the new born. Immersion of the child in a hot bath excites the heart very quickly to stronger and more frequent contractions, and I know a skilful physician who never administers an anæsthetic without having hot water at hand to apply over the heart should danger threaten from asthenia, and he assures me that he has seen death averted on more than one occasion by this means.

Lauder Brunton pointed out many years ago what I have repeatedly verified, that the heart of a turtle or of a frog, when removed from the body, will have its beat quickened and slowed by exposure alternately to heat and cold. These observations indicate a stimulating effect of heat on the cardiac sympathetic. Is it not fair to assume that

the hot blood of a fevered child acts in a similar way on the motor nerves of respiration, and that blood cooled by the cold bath inhibits the heart and the respiration through its action on the vagus? The conservative action of the cold bath on the heart of a fevered patient becomes evident in view of this inhibitive power, because, as Gaskell has pointed out, anabolism proceeds during the interval between muscular contractions, and when these are normally slow, or nearly so, nutrition of muscular tissue can be more perfectly maintained than when the period of relaxation is short. In a paper published in the "Transactions of the International Medical Congress," at Philadelphia, in 1876, I pointed out that a rise of temperature, from whatever cause, in a child is frequently accompanied by eclamptic attacks, that so long as the temperature remains at or above the point at which the eclamptic seizures came on the convulsions are liable to continue, and that reduction of the temperature to about the normal invariably arrests the convulsions. So far as I know this plan of treating convulsions accompanying fever was new, and I was led to adopt the cold bath treatment of such cases by the experiments of Brunton, above referred to. It appeared not unlikely that the same agent which acted on the turtle's heart in a manner quite opposite to that of heat, would, by reducing the temperature of the general circulation, and through it the temperature of every tissue in the body, counteract the stimulating effect of the hot blood on the nervous centres, and arrest the convulsions. The correctness of this supposition was fully sustained by subsequent experience, so that I have come to regard the cold bath as an absolute specific for convulsions coming on during a febrile attack.

About five years ago I had occasion to induce labour at the eighth month for threatened eclampsia. All went well until about half an hour after the birth of the child, the temperature then being normal and the pulse 75, when a violent convulsion occurred. Within ten minutes the pulse rose to 140, and the temperature to 104° F. There was no subsequent convulsion and in less than two hours the pulse and temperature became normal and continued so during convalescence, which was uninterrupted. Zinke, in the *American Journal of Obstetrics* for January, 1893, relates a somewhat