

Only a small percentage of the mortality of this disease, however, can be charged to the specific lesions. Tabulating all the statistics to which I have had access, I find that less than six per cent. of the total number of cases have hemorrhage; of these a little less than one-third die, or less than two per cent. of the whole. About one per cent. of the totality of cases has perforation of the bowel, and a small portion of these recover, so that the entire mortality of this disease, arising from the specific lesions, is not far from three per cent.

It is plain, then, that we must look to the group of secondary lesions or those caused by the general disease for the cause of the heavy mortality in typhoid fever. These structural changes do not belong exclusively to this disease, but may occur in any disease characterized by persistent elevation of temperature to which they undoubtedly owe their origin. They consist of congestions, inflammations and degenerations of important organs, and may include any organ or tissue in the body.

Death may result from the sudden arrest of function of some important organ or organs, as the heart, brain, or lungs, without the structural changes just mentioned, but be caused by the effect of elevation of temperature sufficient to produce paralysis of these organs.

Then we may have a fatal result occurring from three different sources in this disease.

1st. From the effect of the primary or specific lesions, ulcerations, hemorrhage and perforation.

2nd. Directly from the effects of hyperpyrexia producing paralysis of heart, lungs or brain.

3rd. Indirectly, by the pathological changes in important organs caused by the persistent elevation of temperature.

It is now a well established fact that the characteristic symptoms of typhoid fever such as low muttering delirium, picking at imaginary objects, sliding down in bed, subsultus tendinum, sordes, etc., are not the direct result of the specific poison of the disease, but rather the effect of the long continued elevation of temperature during which the structure and functions of important organs are effected by the heat, and the circulation is poisoned by the detritus of rapidly oxydized tissue, which accumulates more rapidly than it can be eliminated; this group of symptoms, known as the typhoid condition, occurs in all diseases which are characterized by persistent elevation of temperature, such as

typhus, yellow, and scarlet fevers, small-pox, measles, and even malarial fevers, when they become continued; under any and all circumstances these symptoms owe their origin to continued hyperpyrexia.

The mortality of typhoid fever varies greatly in different epidemics and in different countries. It is exceedingly difficult to arrive at a satisfactory conclusion in regard to the exact death-rate. In the French Army from 1875 to 1880 inclusive, in 26,000 cases the death-rate was over 36 per cent. German statistics under the expectant plan of treatment which was used prior to 1862 gave a mortality of about 28 per cent.; the English and American death-rate is somewhat lower, but it will be safe to state, without wading through long columns of dry figures, that the percentage of deaths tabulated from the statistics of the entire civilized world would be somewhere between 25 and 30 per cent. Less than five per cent. of these deaths are shown to be caused by the specific lesions of the fever, and the remaining 20 or 25 per cent. of deaths are due to the secondary lesions, and are caused by the long continued pyretic condition present in the disease and *can and ought to be prevented by antipyretic treatment.*

The etiology of typhoid fever is imperfectly understood, but modern investigation, however, has a tendency to establish the truth of what has been discussed for ages as the germ theory, and the probabilities are that the causes of all the infectious diseases will be ultimately traced to low living vegetable organisms.

We possess no specifics for the disease in the same sense that quinia is a specific for malarial diseases or that salicylic acid and its salts are specifics for acute articular rheumatism; so we are compelled to adopt a symptomatic treatment, to combat unpleasant and dangerous symptoms and see that the patient does not die from complications, inter-current diseases or sequelæ.

These objects are best subserved by the cooling treatment. The temperature in this disease controls the situation. The danger is proportionate to its height and persistency, and although the hyperpyretic condition is never free from danger, however brief its duration (for death may take place in a few hours from paralysis of heart or brain), it is to its persistency that the danger in this disease owes its origin. A temperature of 103° or 104°, which is persistent for a period of three or four weeks will work more pathological