of gastric disease. The pathology of the dead-house was succeeded by the pathology of the operation theatre. Operation revealed the early stages of the troubles which gave rise to the symptoms for which the patient sought relief, and so by degrees the surgeon was enabled to compose a clinical picture of the symptoms caused by the various organic lesions found.

Modern methods of gastric diagnosis are of composite growth, the clinician, the surgeon, the chemist, the physiologist, the bacteriologist and the radiographer, all have contributed their quota

to the store of knowledge.

We must be satisfied no longer with such vague diagnoses as hyperchlorhydria, acid dyspepsia, nervous dyspepsia, and gastralgia, high-sounding phrases whose only use is as a cloak for lack of knowledge. These terms signify symptoms, not diseases, and as guides to successful treatment are of less value than the old labels on our baggage, for these at least are reminiscences of former trips.

The introduction of the test meal has enabled us to study the gastric functions in health and disease, and incidentally has shown us how misleading such descriptions are. I have seen many patients whose disease has been diagnosed as acid dyspepsia, analysis of whose gastric contents showed not only entire absence of free hydrochloric acid, but also diminished

acidity.

A good many years ago I operated on a man who had gastric symptoms, due to chronic appendicular disease. I removed his appendix, and as he had extreme hyperchlorhydria, I performed, unwisely as I now know, a gastro-jejunostomy. Before operation, with extreme hyperacidity, he did not notice any acidity, after operation he complained bitterly of a "burning acidity," although analysis of his gastric contents showed hypoacidity and absence of free hydrochloric acid. Later, I restored the continuity of the alimentary canal, the acidity increased, the free hydrochloric acid was restored, but his symptoms of acidity disappeared.

In the vast majority of cases acid dyspepsia and the like are the symptoms of definite organic disease. It is perhaps no exaggeration to say that eighty-five per cent. of cases of persistent gastric trouble are due to obvious lesions, not necessarily in the stomach, but somewhere in the abdomen. What about the remaining fif-

teen per cent?

It is remarkable how, within recent years, fresh sources of persistent dyspepsia have been discovered. In the early days if ex-