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Paroxysmal Tachycardia.

H. M. Rich, in an article appearing in the *Journal of the American Medical Association* for June 4, says that the term paroxysmal tachycardia has latterly been confined to cases in which the heart attains a rate of 200 or more, within a short time following the beginning of the attack. The clinical picture is simply one of tachycardia. Irregularities of rhythm are rare. He reports a case and then describes the special symptoms of the disease. The first intimation of the attack is generally described as a "flop" of the heart and the rapid action commences almost immediately. In a few cases three or four extrasystoles have been observed at the onset, but this is not constant. The earlier attacks cause marked anxiety of the patient, but this generally passes off with the later ones, when the patient is assured that he is not going to die. There is a feeling of pressure over the precordial area and the patient has to sit or lie down. The face is usually pale, and profuse sweating sometimes occurs, but is unusual. There is a disinclination to exertion. The end of the attack is usually as abrupt as the onset. There is usually another "flop" and a feeling of goneness and suffocation and of impending death. In long-continued cases signs of stasis may occur. It has not as yet been possible to produce paroxysmal tachycardia by any experimental procedure on the nerve

supply of the heart, and this is described and illustrated. A very important contribution to our knowledge of the subject was made in 1893 by the discovery of the bundle of His and later discoveries of the nodules of Tawara and Keith. These nodes receive special arteries and are now looked on as the seat of the impulses of the heart rhythm. The modern or myogenic theory of the heart rhythm is that the impulses are in and are transmitted by this primitive cardiac tissue known as the bundle of His, which is composed of muscle cells embryonic in character. In particular, it is believed that normally the impulses arise in the node of Keith, but Gaskell's experiments, in which the ventricle started up with an independent rhythm after both nerve and muscle connections were cut, are taken to prove, that Tawara's node alone may also originate impulses. All these are, of course, subject to inhibition and acceleration through the well-known nerve channels. MacKenzie's nodal theory rests on the assumption that for some reason the impulse begins in the node of Tawara instead of that of Keith, reversing the ordinary sequence of contraction. The pathology, according to Keith's findings, is a fibrosis and degeneration of cellular elements in the heart, extending to the bundle of His in such a way as to produce the nodal rhythm, as MacKenzie calls this disorder. The influence of position is