CHAPTER VIII

Intercostal Muscles and Diaphragm

SPINDLES IN RESPIRATORY CONTROL

Introduction

The new ideas on the role of the gamma loop in motor control began to influence the study of respiration some time before anyone had taken the quite considerable trouble of recording from intercostal spindle afferents or their fusimotor neurons. Thus Nathan & Sears (1960) followed Eldred, Granit & Merton (1953) in interpreting their finding in a patient to the effect that the activity of his unilaterally deafferented intercostals was diminished by comparison with the intact side and with the status before the operation. These authors also gave a valuable review of the old literature on the effects of root section (cf. also Euler & Fritts, 1963; Sears, 1964e; Glebovskij, 1965).

Campbell & Howell (1962, 1963), from experimental work on man referred to in their papers, had come to conclusions which could be simplified as follows: in studies of respiration they had found that the effect of a resistive load in breathing through restricted orifices could be detected at a threshold of 0·6 cm H_2O per litre per sec. Hence some kind of sense organ detecting the change in the relation of pressure to volume was required. Now this relation does not change across the lungs with the external load. Therefore volume is assumed to be translated into length in the intercostal muscles whose length receptors over the gamma loop are adjusted to the demanded tidal volume. With breathing hindered, the ventilatory requirements are initially unaltered. Nevertheless the respiratory effort is immediately increased in compensation. This postulate, as we shall see, was proved true by electrophysiological work (see below). In theory this happens because the demand for a given tidal volume resides in a "wanted" length in the biased spindles. The impediment to respiration prevents the wanted length-setting from being realized with the muscular force allotted to a normal breath. The spindles remain extended and therefore continue to discharge impulses which force the alpha motoneurons to increase their