# Neurotransmitters and the Regulation of Food Intake

PHILIP TEITELBAUM AND DAVID L. WOLGIN

*Psychology Department, University of Illinois, Champaign, Ill. 61820 (U.S.A.)*

## INTRODUCTION

Nature's abnormalities often provide the clearest insight into normal functions. Perhaps this is why much of the work on the neural regulation of food intake has focussed around two major syndromes of abnormal feeding: hypothalamic hyperphagia (Brobeck *et al.*, 1943) and lateral hypothalamic aphagia (Anand and Brobeck, 1951). With recent advances in techniques for anatomic visualization of monoamine systems in the brain (Andén *et al.*, 1964, 1966), and with the development of methods for selectively destroying catecholamine systems (Ungerstedt, 1971b), there have been very exciting breakthroughs in our understanding of the neurotransmitters involved in these two syndromes. Since the fundamental work of Grossman (1960, 1962), there has been a great deal of work on intracranial application of neurotransmitters to determine their role in normal feeding. However, many methodological problems with this method still exist (Booth, 1972; Routtenberg, 1972; Baile, 1974). Therefore, we will concentrate on the neurotransmitters involved in hyperphagia and aphagia. In what follows, we will try to do 3 things: (1) highlight some major recent advances in our understanding of these syndromes; (2) pinpoint some unresolved issues; and, (3) present some recent findings from our own laboratory and that of others which may indicate lines of thought and work that will prove fruitful in the next few years.

## HISTORICAL BACKGROUND

When Fröhlich's syndrome began to be studied in humans, it was thought to be related either to pituitary malfunction or damage to hypothalamus (Erdheim, 1904; Fröhlich, 1940). In work on animals, hypophysectomy was shown not to cause obesity, but subsequent ventromedial hypothalamic damage did (Smith, 1927, 1930; Hetherington and Ranson, 1940). Attention and thinking therefore focused on the hypothalamus.

Brobeck *et al.* (1943) emphasized the overeating as a cause for the obesity produced by ventromedial hypothalamic (VMH) damage. Anand and Brobeck (1951) showed that lateral hypothalamic destruction led to aphagia and death from inanition. Therefore, two systems seemed mainly involved in the control of feeding: a medial

*References p. 245–249*