effect? If so, are the benefits great enough to justify their use despite the long-term adverse effects?

There is still no real understanding of how amphetamine reduces appetite. Experiments with animals have demonstrated that it is not a function of local effects on the gastrointestinal tract. There is some evidence that lesions in the hypothalamus may result in a substantial increase in appetite. If some obese people actually have a dysfunction of the hypothalamus, it is possible that amphetamines reduce annetite by their effect on this area of the brain. However, if this mechanism exists at all, it is probably secondary to the central stimulating effect. It has been suggested that diversis may cause some of the weight loss associated with the use of amphetamine. Both divertic and antidivertic effects have been reported, however, and the role of diversis in true weight loss is far from clear.

In 1938 a research group led by Poul Bahnsen compared one hundred normal subjects receiving amphetamine with an enual number receiving placebo. Nineteen of the active drug group and one control reported a reduction in appetite. The first attempts to apply these observations to the clinical management of obesity were made that same year by M. F. Lesses and A. Myerson, and by P. Rosenberg in 1939; both papers reported favorable results. Since then a long series of reports and clinical studies has agreed with them. For example, S. C. Harris, A. C. Ivy, and L. M. Searle found that seven obese patients lost more weight when taking amphetamine than when taking placebo. Those who lost most were the ones who ate least, so the main cause of weight loss was apparently suppression of appetite rather than something like higher activity level. Harris also