HEAD INJURIES IN AMATEUR BOXING

It has been estimated that after five years' fighting as many as 60% of professional boxers have suffered tractable changes, and that 5% have the slow dementia, rigidity, and tremor of parkinsonism. Loss is known about the risk to our 100,000 inactive boxers by Louis and Silberman's 
changes in boxers have been extensively studied. Dow et al. found that such changes amounted to 15% after thirty-three minutes after the injury; but Busser and Silberman reported persistent abnormalities of the left carotid after 30 minutes' exposure, as compared with Gwathmey's figure of 15%, for controls. The relation of these findings is not yet correlated with the length of the boxing career, but the more conspicuous are found in men who have been out of the ring for years. Laceron and associates studied 74 amateur boxers, of whom 44% respectively had some symptoms and signs, and remaining 36% only because they were suspected of having sustained head injuries. An a.m. a.c. read within thirty minutes of the match. In the first day of a year of apparently unimpaired boxers had a proportion close to that found from a month's rest after the fight. The a.m. a.c. read before and after the fights eliminated confusion due to the same stimulus.

An ovarian and Clarke's point out, ideally should be used. They concluded that compensatory recovery is not complete. By comparison of the initial record with the consequent improvement of the same stimulus, it might be made clearer. Meanwhile the risks to life of repeated minor cerebral damage cannot be assessed; but it seems most likely that those exposed to the severe disorders sometimes found in professional boxers.

The body temperature remains constant by balance between heat production and heat loss for slight climatic variations and the limited women, the temperature in health is remarkably normal. We are almost entirely ignorant of the mechanism by which the body temperature is increased in the metabolic effects of pyrexia, and whether pyrexia are advantageous to the organism.

Some stages in the disease response has been experimentally studied. The administration of a pyrogen substance is followed by a latent period, varying in hours but inconstant and lasts for 12-30 minutes, and in man for 48-72 hours. The pyrogen acts by the liberation of leukocytes from the bone marrow. Leukocytes release a pyrogenic substance, which is not the same as the pyrogen. The pyrogen acts directly on the thermoregulating centres of the brain.

The suppression of pyrexia by cortisone studied by Atkins et al. Who found that such suppression was independent of the absence of the normal pyrogen factors but that the action was in the hypothalamic centres. Atkins et al. remark, artificially induced pyrexia is a commonplace phenomenon, but it seems likely that after either its suppression or its appearance on the hypothalamus, and the "serum factors" are of greater importance in its appearance on the hypothalamic centres.