The following article from the 'Medicine in Sports Newsletter' Vol. 4, number 1., is reprinted in full by courtesy of The Rystan Company, Mt. Vernon, New York. Dr. Alan Ryan is an Honorary Member of B.A.S.M. and members present at the Loughborough Conference in June 1963 will remember the tremendous impression Dr. Ryan made both by his expert knowledge and by his warm and friendly personality.

SMOKING AND ATHLETICS

Interest in the effects of long-term smoking of tobacco, particularly of cigarettes, on the human body has focussed principally on its possible role in lung cancer, coronary thrombosis and chronic bronchitis and emphysema. Because its effects on the respiratory passages appear to be related to a chronic irritation, it is only natural to wonder what short-term effects on pulmonary function may result from smoking. Since ability to produce maximum efforts and to build endurance in almost all forms of athletic competition are related to the ability to take in and utilize oxygen, these considerations become very important in athletic training and conditioning.

In fact, it may be stated categorically that in any athletic performance where maximum muscular efforts must be developed and sustained, even for very brief periods, anything which impairs the ventilatory capacity will be bound to affect performance unfavourably. It only remains to be seen, then, if there is sufficient clinical and experimental evidence of acute impairment of this capacity by smoking to determine that athletes should be most strongly advised against it. It is no good simply to forbid them to smoke, since they will do so surreptitiously whether you know it or not. They must be persuaded that it is to their advantage not to smoke, and the following data strongly indicate that this is so:

1) Ten inhalations of cigarette smoke reduce airway conductance to the lungs, the average maximum decrease being 50 per cent and lasting an hour, according to Nadel and Comroe of the University of California Medical Center.¹ This may be attributed to obstruction of the air ducts resulting from muscle contraction, edema and nervous reflex. Their subjects were 36 healthy individuals. The changes were similar in smokers and non-smokers and were reproducible.

2) Wilson, Meador, Jay and Higgins from the University of Texas Southwestern Medical School studied pulmonary function and capacity of 14 young heavy smokers and 14 non-smokers.² They found that the timed one-second forced expiratory capacity, total lung capacity and maximum breathing capacity were significantly decreased in heavy smokers. The ratio of residual volume to total lung volume
was increased in smokers and the diffusibility rate decreased. Decrease in maximum breathing capacity and increase in residual volume are particularly significant to athletes, since both changes would markedly impair the capacity to take in oxygen and make it available in the body for immediate use.

3) Swann of the University of Pittsburgh Graduate School of Public Health reported on studies of pulmonary functions in 13 young non-smokers and 13 smokers at the 12th annual meeting of the American Physiological Society. He found vital capacity for smokers to be 4.0 liters and for non-smokers 5.1 liters. Under conditions of light exercise there was no difference in respiratory rate, tidal volume, minute volume, oxygen consumption, ether and acetylene intake, but carbon monoxide uptake (an index of diffusibility) was lower for smokers. Under moderate exercise the smokers had a higher respiratory rate and minute volume and lower tidal volume, ether, carbon monoxide and acetylene uptake. Oxygen consumption was the same.

4) Shapiro and Patterson of the Medical College of Virginia studied 25 young athlete non-smokers, 11 non-athlete non-smokers and 31 non-athlete smokers. They found that, although the athletes had an increased vital capacity as compared to the non-athletes of both other groups, there was no apparent significant increase in the forced expiratory volume. If the rate of expiratory airflow were compared with the predicted rather than the observed vital capacity in athletes, it was found that there was a difference favoring the athletes. Smoking was also associated with a decreased maximum breathing capacity.

5) In studies of 23 patients with pulmonary disorders and 19 normal subjects before and immediately after smoking two cigarettes, Rothfield, Biber and Bernstein noted a striking increase in the ventilation equivalent in normal subjects. This was attributed to impaired oxygenation caused by smoking.

6) Simonsson reported on dynamic spirometry performed on 32 subjects, some normal and some with pulmonary disease. Tests were conducted before, immediately after and 45 to 60 minutes after inhaling smoke from 1 to 2 cigarettes. In 28 of the 32 cases there was a decrease in the forced expiratory one-second volume immediately after smoking. One hour later 23 of 32 such values had increased but had not reached basal levels. The decrease was attributed to smoke-induced increase of airflow resistance.

7) Martt studied pulmonary diffusing capacity in 79 healthy adults, using the single-breath carbon monoxide technique. One half had smoked a pack or more of cigarettes daily; the remainder had never smoked. The average ages for all subjects was 27.5 years. The mean pulmonary diffusing capacity in smokers was significantly decreased, whereas other pulmonary functions were normal and comparable in both smokers and non-smokers.
Thus we can see that acute impairment of pulmonary function has been demonstrated in young smokers as compared to non-smokers. Significant decreases in the maximum breathing capacity, which is most important to athletes, have been described in many of these studies. This appears to be due in great part to acute obstruction of respiratory inflow. Efficient utilization of oxygen is hampered by decreases in its diffusibility in smokers. There is no longer any room for doubt that athletes should not smoke.

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