BAL KRISHAN ANAND

(19 September 1917 – 02 April 2007)

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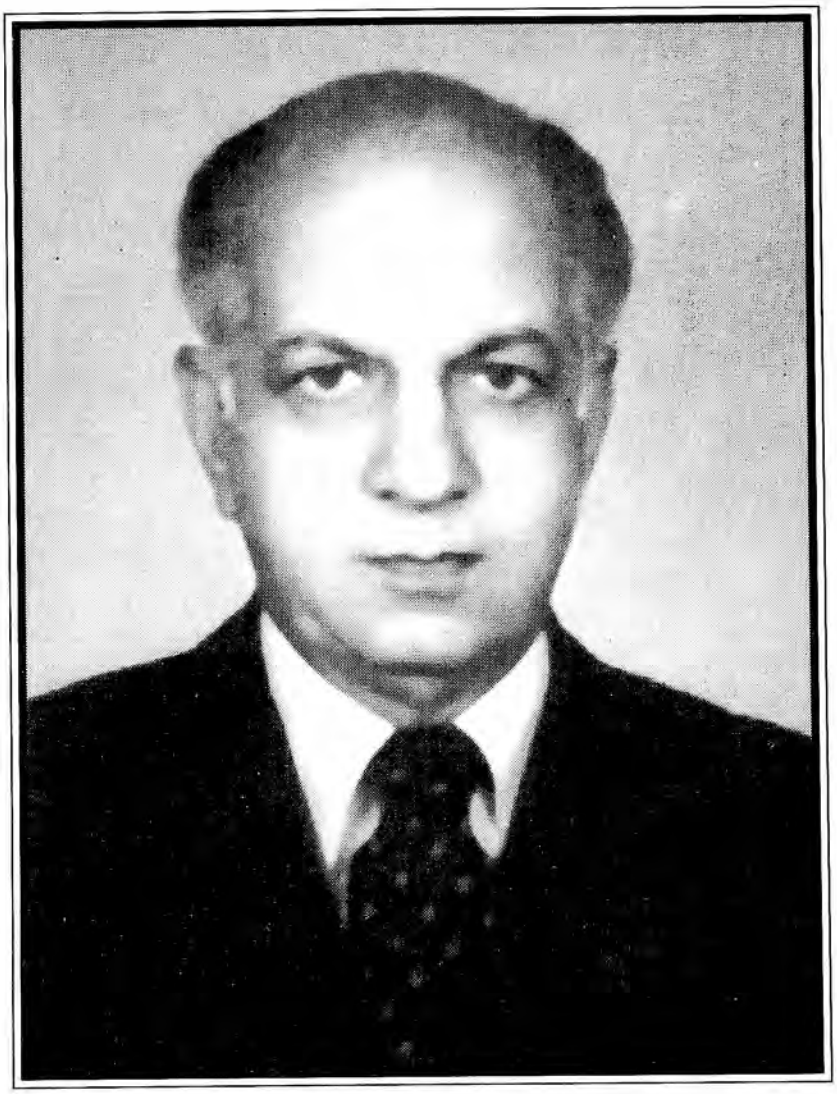

BAL KRISHAN ANAND

(1917-2007)

Elected Fellow 1972

EARLY EDUCATION

BAL KRISHAN ANAND was born on the 19th of September 1917 in Lahore, Punjab then in India. He was the only child of his parents, Saraswati Devi and Visheshar Das Anand. His father, Visveshar, died in 1917 at the age of 25. He spent his early life in Lahore, he had his schooling in Municipal Primary School, (1922-27) and DAV High School (1927-33) and Intermediate Science (medical group) during 1933-35 from the Government College. Anand was a brilliant student and a recipient of several distinctions. He did his MBBS from King Edward Medical College, Lahore during 1935-40. He stood second in final MBBS Examination. He got his MD degree from Punjab University specializing in Medicine and Physiology in 1948. He had his house physicianship in Mayo Hospital, Lahore in 1941. He went to Yale University, New Haven, USA on Rockefeller Foundation fellowship and did post graduate research during 1950-51.

POSITIONS HELD

Dr Anand started her career as a demonstrator in Amritsar Medical College during 1943-49, Professor of Physiology in Lady Hardinge Medical College, New Delhi between 1949 and 57 and finally as a Professor and Head, Department of Physiology, All India Institute of Medical Sciences, New Delhi between 1957 and 74. He was Dean of AIIMS, New Delhi between 1966 and 74. He was Assistant Director, Health and Man power Development, South East Asian Region, WHO, between 1974 and 77. From 1977 till 1991, he was an Emeritus Professor, AIIMS and concurrently Director, Sher-i-Kashmir Institute of Medical Sciences, Jammu and Kashmir till 1982. He was a Commonwealth Visiting Professor, London University, UK in 1966 and a Visiting Professor, Pennsylvania School of Medicine, USA in 1968.

RESEARCH CONTRIBUTION

Professor Anand started his research looking into the nutritional aspects of the etiology of Portal Cirrhosis in Punjab. Studies were conducted on the effects of changes in temperature on the release of acetylcholine at the parasympathetic nerve endings in the heart. His training in Yale University was mostly devoted to the learning of the various techniques used for the study of the functions of the higher

nervous system; Active interest was developed for studying visceral and autonomic activities, with specific reference to food intake.

It was observed that the study could be broadly classified into (i) the more recently evolved neocortical areas, which regulated the conscious mechanisms and control over the somatic activities of the body (ii) the older parts of brain, which s a result of recent studies has been shown to regulate the outflows, the endocrinal activities and the effective behaviour the higher nervous mechanism towards the maintenance of constancy of milieu interior. Professor Anand's studies have been mainly directed towards working out a mechanism of regulating these activities from the hypothalamus and the limbic system of the brain. Regulation of food intake from these higher nervous levels have been of prime interest to him.

(i) Higher Nervous Regulation of Food Intake

Some of the important original contributions have been in this field. It was demonstrated that in the hypothalamus there is a dual mechanism for regulation of food intake. In the lateral hypothalamus is located an area which provides the organism (the urge to eat) and was given the name feeding centre. In the medial hypothalamus is an area which inhibits further eating. This region is activated after feeding and provides the satiety mechanism. This was, therefore, termed satiety centre.

Feeding is the result of flexes operating from lower levels, which are facilitated by feeding centres and inhibited by satiety centres. Satiety centres also inhibit the activity of feeding centres.

Although the basic hunger and satiety mechanisms are located in the hypothalamus, the limbic system also plays in important role in bringing about a discriminative regulation of eating. Thus the mechanism of discriminative appetite is located in the limbic system. In the hypothalamus is also located the regulating mechanism for water intake. Although this drinking area is automatically situated in the same region as the feeding area, the two mechanisms act independently.

In a starving animal, the feeding centre is active, while the satiety centre is non active. After taking a meal, the reverse holds good. It has been clearly demonstrated that it is the increase in glucose utilization which activates the satiety centre. The EEG activity recorded by depth electrodes from these hypothalamic centres shows inverse changes in electrical activity of these two centres when changes in blood sugar concentration are produced hyperglycemia stimulating satiety centre and inhibiting feeding centre, while hypoglycemia producing the reverse picture. Changes in amino acids and fat contents of blood do not produce any change in the activity of hypothalamic centres.

Preliminary studies on the unit activity recoded through microelectrodes have also corroborated these observations. Metabolism of these hypothermic centres have been studied with Warburg manometers, in the fed and starving animals. In fed animals, the satiety centre picks up more oxygen and more glucose than the feeding centres. In the starving animals, the feeding centre picks up more oxygen and glucose than satiety centres. The distension of the stomach also brings about a condition of satiety. When the stomach is distended with balloons, the EEG activity of the satiety centre increases. So there must be afferent projection from stomach to the satiety centre. Effect of some of the pharmacological preparations influencing appetite and hunger on the activity of hypothalamic centres have also been demonstrated. The studies, in this field, are reaching a stage, after some work, it may be possible, to draw a concrete picture regarding the nervous regulation of food intake.

(ii) Higher Nervous Regulation of Autonomic and Visceral Activities

Starting with hypothalamic regions and gradually going over the various limbic lobe structures, the effects of stimulation and ablations of various regions have been mapped out. These studies are by no means complete. Quite extensive further work will be required to complete the picture. It is clear that both the hypothalamus and the limbic system influence the activities of all the viscera, which are under autonomic regulation. It has also been observed that while in the hypothalamic region there is a distinct localisation in terms of sympathetic and parasympathetic controls, the responses of all the visceral systems from the limbic system do not show any localization. It is suggested that hypothalamus is the motor area of this system, while the rest of the limbic system has integrative functions, like the functions of the neo cortical association areas. Some of the original contributions in this area are as follows.

(a) Cardio Vascular System

Frontal lobe limbic structures have a press or effect, while temporal lobe limbic structures produce a depressor response. Response produces a dual effect on the hypothalamus. It inhibits the sympathetic regions and produce active facilitation of parasympathetic regions. After the use of anesthesia, the vascular responses become much varied. It is not advisable to use anaesthetized preparations.

(b) Respiratory System

Frontal lobe structures are facilitatory while temporal lobe structures are inhibitory.

(c) Gastrointestinal System

Lesions in the pre optic region of the hypothalamus produce acute haemorrhagic ulcers in the gastric mucous membrane. Such ulcers can also be produced by lesions of the orbito-mesial cortex and the amygdaloid nuclei. Lesions in the medial

hypothalamus prevent the acute necrosis of the liver produced after heavy malarial infections which nearly 90% RBCs. Lesions in the limbic system affect the liver function tests.

(d) Blood Chemistry

Lesions in the limbic system change the glucose, Na and K contents of the blood, as well as the plasma proteins. It has been show n that the Paleocerebellum projects heavily into the limbic system.

(iii) Nervous Regulation of Endocrinal Activities

Studies conducted showed that the hypothalamic areas controlling ACTH secretion as well as Gonadotropins are the same. This is located in the medial part of the median eminence. By stimulating this area both ovulation and menstrual flow can be produced before they are due. ACTH secretion is not affected from the limbic levels. In hypersexual animals, castration does not abolish this response.

(a) Observations on Yogis

In one yogi, who was kept in an air tight box, for ten hours, O_2 intake and CO_2 output fell 40% below the basal requirement. This suggests decrease of the metabolic activities through the autonomic and endocrinal systems. Another yogi demonstrated complete heart block voluntarily. One yogi could voluntarily bring about restricted to the forehead. In deep meditation the cortex remains in alpha activity and does not pass into the delta activity typical of sleep.

AWARDS AND HONOURS

Dr Anand was elected Fellow of INSA in 1972, Indian Academy of Sciences in 1975 Member, National Academy of Medical Sciences in 1968. He was the President of the National Academy of Medical Sciences in 1982. He received the Amirchand Research Award of ICMR in 1955 and 1962, GJ Watmul Award in Medical Research in 1964, Medical Council of India Silver Jubilee Award in 1969, Padma Shri by the Government of India in 1969.

OTHER CONTRIBUTIONS

Dr Anand was credited with several other contributions. He established the Association of Physiologists and Pharmacologists of India and *the Journal of Physiology and Pharmacology*. This is considered one of the best medical journals in the country. He passed away on the 2nd of April 2007.

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