PREFACE

Data accumulated during the past several years leave no doubt that the neoplastic process eventuating in obvious gross cancer can be initiated by a wide variety of external stimuli. There is also no doubt that the cellular reaction to the carcinogenic stimuli must involve the genetic mechanism of cells in order to endow the cells with properties consistent with the diagnosis of neoplasia. The studies in molecular biology and viral oncology during the past two decades have allowed the development of concepts that merge the viral and genetic theories of yester years. Both theories imply that cancer results from the activation of potentialities inherent in normal cells and that it involves some malfunction of the genetic apparatus of somatic cells. Although an infective agent may be probably responsible for the initiation of the malignant process, establishment of the malignant phenotype will be dependent on an interaction between host factors (immune defect and genetic instability) and tumour factors. This work is an attempt to apply the viral and genetic theories of carcinogenesis in elucidating the etiopathogenesis of HD. HD is a heterogeneous group of lymphoproliferative disorder characterized by the presence of Hodgkin’s and Reed-Sternberg cells in a background of large numbers of immune competent cells. Little is known of the origin of the Hodgkin cell and thus only limited progress has been made in elucidating the pathogenesis of HD. Because Epstein Barr Virus is oncogenic for human B lymphocytes, it was decided to determine its role as a causal agent in the etiopathogenesis of HD which also is a B cell malignancy. Furthermore, it was also decided to study the role of the host’s genomic instability and immune status in the etiopathogenesis of HD.