EXPERIMENTAL
EPILEPSY

ILAE in recent years has nurtured a series of outstanding researchers in the basic sciences, both clinical and non-clinical. A body of excellent work has been produced which has greatly advanced the understanding of the mechanisms and treatment of epilepsy. Experimental epilepsy research in the past half-century has been dominated by cellular and synaptic physiology and by neuropharmacology. The discovery in the 1960s of the paroxysmal depolarization shift (PDS) as the cellular basis of the epileptiform burst discharge was a fundamental advance. The introduction of the brain slice preparation for epilepsy research in the 1970s provided the opportunity for intracellular recording and led to the discoveries of the channel contributions to abnormal discharge, feedback synaptic excitation as a synchronizing mechanism, the modulating roles of glia and other non-synaptic factors, neurotransmitter function and the various inhibitory processes which influence normal and epileptic activities at both systems and molecular levels. In the 1980s, the mechanisms of action of some antiepileptic drugs were first elucidated. From the neurochemical point of view, there was intense interest first in the role of GABA-mediated inhibition and then of glutaminergic excitatory mechanisms. The revolution in molecular genetics in the past two decades has sparked interest in ion channel function and has lead to a detailed understanding of how channel blocking drugs function and the role of a range of neurotransmitters. The importance of neuronal and circuit plasticity as the cellular basis of temporal lobe epilepsies and spike-wave absences was then established. In the 1980's and 90's, paediatric studies have demonstrated in animal models of immature epilepsies that paediatric epilepsies are based on unique molecular properties of the developing brain. In the past decade, there has been an dizzying explosion of molecular genetic techniques leading to insights about an array of molecules and cellular processes involved in idiopathic epilepsies, activity-dependent epileptic plasticities, the role of inflammation and other potential epileptogenic mechanisms.