Science, and he was a member of various scientific, academic, and honorary societies. He served on both state and national committees that were concerned with ecology and its applications. As ecologist with the Oklahoma Biological Survey, he participated with his students in many field excursions, and his own published output of scientific contributions was augmented by their work.

Dr. Weese was the recipient of many distinctions within the university, such as the deanship of the graduate college for a period, the chairmanship of the Committee on Faculty Research for some years, and, during the last years of his life, the David Ross Boyd professorship of zoology. He held elective offices in such local organizations as Sigma Xi, the American Association of University Professors, and others.

His most significant trait was his wide and profound knowledge; his reading and study were extensive and not limited to his field of research. He was an able invertebrate zoologist, he was interested in the quantitative aspects of genetics as well as bioecology, and he developed a course in quantitative biology. He was able to penetrate obscure problems and to give advice and illumination on matters both inside and outside his own fields. He contributed greatly to the development of the departmental library at the university and was responsible for many of its acquisitions. Since his death, this library has been named in his honor.

A kindly, interested, and cooperative scholar, he left a moving and permanent impression on all with whom he came in contact. He bore his own burdens simply and fully shared those of his colleagues and his community, which, in a growing university, were often not light. Like so many whose accomplishments remain long to tell their stories, he will never be replaced, and he will live on in the deeds of his students and associates.

A. Richards

Department of Zoology, University of Oklahoma

H. H. De Jong, Experimental Neurophysiologist

The history of experimental catatonia is the history of the collaboration by investigators from different countries, united in the love of science and in the enjoyment of scientific research, as well as by the synthesis of data from physiology, biology, and clinical experience—a synthesis indispensable to the future progress of neuropsychiatry.

H. Holland De Jong was born in 1895 in Sneeck, Holland. In 1928 at a meeting of the Société de Neurologie de Paris, he presented a paper on the treatment of tremors by bulbocapnine, an alkaloid that he regarded as an "antagonist of tremors." In the course of plethysmographic investigations of many patients, De Jong had discovered a "vascular rigidity" in catatonics that he failed to find in other patients. He thought of the possibility of the experimental reproduction of this illness, and, on the advice of Magnus, became interested in bulbocapnine. But being unacquainted with clinical catatonia, he had not begun this work.

At that time, I had, with others, undertaken a systematic study of Kahlbaum's catatonia, with a physiological exploration of this illness by new processes and a comparison of the physiological data with precise clinical data based on a study of many patients. I had reached the conclusion that catatonia constitutes a psychomotor syndrome of toxic origin. I approached De Jong, and we agreed to collaborate.

Our first joint experiments were undertaken in Paris in the laboratory of Claude. We compared in detail the symptoms that were produced by bulbocapnine in the cat with the symptoms of human catatonia. We found in the animal, as in human beings, the various manifestations of catalepsy: active and passive negativism, barriers, catatonic hyperkinesis, and neurovegetative disturbances, including salivation and respiratory disturbances. The same parallelism was also found in electromyograhic curves and in disturbances in chronaxie, as in the experiments of Bourguignon and De Jong, and in man by Claude, Bourguignon, and Baruk.

Our next experiments were in Amsterdam in the laboratory of Brouwer. We were able to study in detail the action of bulbocapnine for a wide range of doses and for the entire vertebrate series. In this way, we formulated laws describing the stages that follow increasing doses, from sleep to catalepsy, from catalepsy to negativism, from negativism to hyperkinesis, and finally, with still stronger doses, to epilepsy and the rigidity of decerebration. In similar fashion, the study of the animal series, starting with fish through batrachians, reptiles, and birds to mammals, permitted us to verify the role of the cortex. We described this work in our joint book (1930), which brought us both the Herpin prize of the Academy of Medicine.

De Jong and I were then separated. I remained in Paris, and De Jong went to the United States to teaching posts at Duke University and Johns Hopkins University, where he discovered experimental hormonal catatonia (adrenaline, acetylcholine, and so on), surgical catatonia, mescalinic catatonia, and so on. During this period, I discovered, in Paris, experimental catatonia that was induced by the toxin of *Escherichia coli* (1933), biliary experimental catatonia (with Camus, 1934), and other types of experimental catatonia, induced by cerebral edema, ACTH, chlorpromazine, and so on. During his last years, De Jong investigated the role of intestinal and hepatic factors. His research on catatonia by obliteration of the intestinal lumen and by ligature of the biliary ducts must have led him to consider again conceptions of the role of hepatointestinal factors in catatonia and schizophrenia, conceptions that, after the work of Buscaino and our work on this subject had converged toward our present conclusions, have had important therapeutic results.

Recently, De Jong and I began to think about collaborating again in Paris. Alas, his premature death on 16 February 1956, at the age of 61, in Osawatomie, Kansas, where he was director of research and education at Kansas State Hospital, prevented the realization of this plan. This great misfortune abruptly ended a life dedicated to science and deprived medicine of a scientist of exceptional intelligence and perspicacity, a scientist whose goal it was to discover the causes of mental illness.

H. Baruk

Maison Nationale de Charenton, Paris, France