Summary

Several hormones have been found to possess properties that are distinct from their classic endocrine functions. The recent delineation of extensive peptidergic pathways in the brain has been reflected by an accelerated research effort aimed at discovering the precise role that these peptides play in cognitive behaviour.

Vasopressin (VP), a peptide secreted by the posterior pituitary has come under close scrutiny. De Wied and his workers had developed the thesis that VP and some of its analogs influence memory consolidation (de Wied, 1965, 1969). Support for this postulate is provided by a variety of researches in which VP administered either peripherally or centrally (de Wied, et al., 1965; Bohus and de Wied, 1966; van Wimersma, 1975, 1979; Kovacs et al., 1979) was found to effect retention in a dose and time dependent manner while treatment with VP antiserum abolished these effects (van Wimersma and de Wied, 1976). Pretesting administration of VP also facilitated retention indicating that in addition to affecting memory consolidation (de Wied et al., 1976) VP also promotes its retrieval (Rigter et al., 1974, 1978). Further evidence for the memory modulatory role of VP is available from investigations in which
Amnesia induced by Puromycin, Anisomycin, $\text{CO}_2$ or ECS (Rigter et al., 1974, 1978; Judge and Quartermain, 1982), was reversed by the administration of VP.

This hormone also influences the secretion of a number of endogeneous substances such as ACTH, $\beta$-endorphin and NE (de Wied and Gispen, 1976; Weitzman et al., 1977; Vale et al., 1979), and it has been used with success in the treatment of alcoholic, senile and Korsakoff's amnesia and other psychotic disorders such as schizophrenia, manic depressive etc. (Oliveros et al., 1978; Legros et al., 1978; LeBoeuf et al., 1978; Legros et al., 1978; Gold and Goodwin, 1978; Vranck et al., 1978), which are known to occur due to neurohumoral imbalances.

In view of the immense practical importance of this hormone in reverting the amnesia associated with a number of psychological disorders, the present study was designed to investigate whether the amnesia due to depression could be eliminated by administration of VP. Morphine was administered to induce a depressive state in animals.

It was hypothesised that:

1. Immediate post training administration of morphine, an opiate agonist, would have an inhibitory effect on retention of an aversive learning task.
2. Post training administration of VP, would have a facilitative effect on retention of a passive avoidance task.

3. Administration of VP, five minutes after post-training administration of morphine, would antagonize the morphine induced amnesia.

The mean latency score of the morphine injected group was significantly lower than that of the other three groups on all the three retention tests, except the saline group which is not significantly different only on the last retention test. Conversely the latency scores of the VP injected group is significantly higher than the saline group on all the three retention test. These results indicate that morphine is having an inhibitory effect while VP is facilitating retention.

The mean latency score of the morphine plus VP group is significantly different from the morphine injected group indicating that the amnesia induced by morphine has been reversed by administration of VP.

Thus all the three hypotheses are verified. The present investigation indicates that the amnesia induced by a depressive agent is reversed by administration of VP.