evidence given, some of these proposed depressiogenic sleep-related mechanisms may indeed occur during the hSWS period. An obvious possibility for such a mechanism may be the physiologically altered balance of catecholaminergic – cholinergic neurotransmitters with consecutive changes of receptor sensitivities during hSWS. If future research gave more evidence for this hypothesis, it should be asked next whether altered hSWS in depression is a source of primary dysfunction, or whether the decrease is secondary to depression to avoid the depressiogenic effects of 'normal' hSWS.

There is another possible source of hSWS involvement in the origin of depression: hSWS reduces the heat load and dampens hypermetabolic states, and it may be of importance that those depressive patients react best to TSD who have higher nightly temperatures than controls or TSD non-responders (Wehr, 1990).

As mentioned by Dr Horne, it is not necessarily the dorsolateral PFC which is involved in hSWS related depression, but the chain of events leading to depressiogenic hSWS may start in other brain regions, for example the gyrus cingularis and the fronto-orbital cortex. Thus, in previous SPECT and positron emission tomographic studies these two structures were found to be hyperactive in TSD responders before TSD, compared with non-responders and controls, and hypermetabolism was suppressed by TSD (Ebert *et al*, 1991; Wu *et al*, 1992). With the cingulate cortex being also hyperactive during rapid-eye-movement sleep, these findings point to sleep stages other than hSWS as being involved in the origin of depression. However, the possible link between fronto-orbital and cingulate hyperfusion and dorso-lateral prefrontal hyperperfusion demonstrated with SPECT (Ebert *et al*, 1991) still supports Dr Horne's view of hSWS and PFC involvement in depression, and, additionally, both activity patterns are not contradictory to a theory of a catecholaminergic – cholinergic imbalance in a subset of depressed patients.

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D. EBERT

University of Erlangen
Schwabachanlage 6–10
8520 Erlangen
Germany

A HUNDRED YEARS AGO

Broadmoor criminal lunatic asylum

Dr Nicolson's annual report for the year ending 1891 upon the Broadmoor Criminal Lunatic Asylum shows that the asylum population included 336 persons who had committed homicide, 205 of whom were men and 131 women. These cases of homicide consisted of 313 cases of murder and 23 of manslaughter. The admissions to the hospital included 41 men and 15 women during the year. Of these 33 were "Queen's pleasure" inmates and the remaining 23 consisted of insane convicts. The report points out that although there were in all 539 afflicted people in the asylum suffering from various degrees of insanity yet at no period during the whole year had it been found necessary to rebuke any member of the staff for imprudence in dealing with those under his charge. This is a very satisfactory statement. The report also mentions that no inmate had succeeded in effecting an escape, and that it had not been found necessary during the year to have recourse to mechanical restrain in any form. The facts and figures stated above afford ample evidence of the nature of the organisation and of the excellence of the arrangements made at Broadmoor for the care of criminal lunatics. On Friday, the 3rd inst., the staff and attendants of the asylum enjoyed their annual entertainment. In the course of some complimentary remarks to the members of the staff Dr Nicolson expressed himself as being highly pleased with the efficient assistance which they had rendered during the past year. We take the opportunity of congratulating Dr Nicolson himself and his assistant medical officers, Dr Issac, Dr Noott and Mr W. E. Miles, on the able manner in which the onerous and difficult duties appertaining to the management of the institution are carried out.

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Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey

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