

'Kleine-Levin Syndrome' Bibliography

(arranged by year of publication from 1925-2007)

(with "periodic hypersomnia" bibliography supplement)

J Am Acad Child Adolesc Psychiatry. 2007 May;46(5):551-2

Kleine-Levin syndrome mimicking mania.

Masi G, Mucci M, D'Acunto G.

Type: Letter

Orv Hetil. 2007 Apr 22;148(16):723-30.

[Some neurological and psychiatric complications of the disorders of the hypothalamo-hypophyseal system] [Article in Hungarian]

Aszalós Z.

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Connection between the central nervous system and the endocrine system is extremely complex. The hypothalamus serves as a crucial centre for the integration and coordination of autonomic functions by neuronal and hormonal pathways. It plays a central role in the homeostatic regulation of internal physiological conditions. It controls growth and reproduction, stress reactions, and determines rhythmicity, periodicity and timing of physiological processes. Beside its well-known functions, antidiuretic hormone has a role in social behavior as it enhances aggression via vasopressin receptor 1A. Oxitocin is affected in the formation of maternal behavior, and in other social interactions, like the pair bonding, as well as in analgesia and pain modulation. The corticotrop-releasing hormone acts as a neurotransmitter, it has a special role in stress-behavior, anxiety, and depression, and it blocks deep sleeping. Among the neurotransmitters and neuropeptides of the hypothalamus, serotonin, norepinephrine, GABA, cholecystikinin, neuropeptide-Y, Agouti-related protein, alpha-MSH and ghrelin have essential importance in the eating disorders. The levels of leptin and galanin determine whether formation of anabolic or catabolic neurotransmitters should take place. In the thermoregulation the central thermoreceptors play role, and suprachiasmatic nucleus is responsible for circadian rhythm, through "timing genes". The diseases of the hypothalamus cause most frequently bulimia or anorexia, hypersomnia, impotency, and attacks of anxiety. The most common expansive process of the hypothalamus is craniopharyngioma. The lack or diminution of vasopressin causes diabetes insipidus, while inappropriate antidiuretic hormone secretion induces Schwartz-Barter syndrome. Fröhlich-, Kleine-Levin- or Prader-Willi syndromes have characteristic neuropsychiatric features. The main psychiatric symptom of hypopituitarism is a combination of dementia and delirium. The most characteristic neurological sign of pituitary adenoma is the visual field defect. Carpal tunnel syndrome, obstructive sleeping apnoe and headache are typical neurological features in somatotrop adenomas.

Publication Type: Review

Arq Neuropsiquiatr. 2007 Mar;65(1):150-2. [free full text available at SciELO.org]

Kleine-Levin syndrome: interface between neurology and psychiatry.

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We report the first episode of Kleine-Levin (KLS) syndrome in a 17-year-old male. The illness onset, clinical features, neuropsychological evaluation and polysomnographic recording are described. Typical symptoms hypersomnia, hyperphagia and sexual disinhibition were observed besides behavioral disturbances, polysomnographic and neuropsychological alterations. Behavioral disturbances similar to a manic episode including psychotic symptoms were relevant. The pharmacologic treatment included lithium, methylphenidate and risperidone. The introduction of risperidone aimed the control of psychotic symptoms and the persistent manifestations of hypersexuality after sleepiness control and to the best of our knowledge there are no other reports regarding risperidone use for KLS in the literature.

(2006)

Sleep Med. 2006 Dec;7(8):649-51. Epub 2006 Nov 13.

Kleine-Levin syndrome in a 14-year-old girl: CSF hypocretin-1 measurements

Podestá C, Ferreras M, Mozzi M, Bassetti C, Dauvilliers Y, Billiard M.

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CSF hypocretin-1 measurements were performed during a period of hypersomnia and during an asymptomatic interval in a 14-year-old girl affected with severe Kleine-Levin syndrome. A twofold decrease in hypocretin-1 was evidenced during the period of hypersomnia in comparison with the asymptomatic interval. Together with previous data, this result is in favour of recurrent dysfunction at the hypothalamic level in Kleine-Levin syndrome.

Publication Type: Case Report

Curr Opin Pulm Med. 2006 Nov;12(6):383-9.

Gender differences in sleep disorders.

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PURPOSE OF REVIEW: To evaluate recent evidence regarding gender differences in sleep. **RECENT FINDINGS:** Women have better sleep quality compared with men, with longer sleep times, shorter sleep-onset latency and higher sleep efficiency. Despite this, women have more sleep-related complaints than men. The amount of slow-wave sleep decreases with age in men and women. Normal

physiologic periods, including puberty, menstruation, pregnancy, and menopause, are associated with alterations in sleep patterns. Gender differences in normal sleep may underlie the observed differences in risk of sleep disorders. Studies of insomnia support a female predominance, with increased divergence of prevalence between men and women with older age. Recent findings for the gender differences in obstructive sleep apnea have focused on differences in local neuromuscular reflexes and central ventilatory control. Restless legs syndrome has a slight female predominance, whereas rapid eye movement sleep behavior disorder and Kleine-Levin syndrome are more common in men. SUMMARY: Gender differences in sleep become apparent after the onset of puberty. Menstrual cycles, pregnancy, and menopause can alter sleep architecture. Gender-related differences in sleep disorders, such as obstructive sleep apnea, insomnia, and restless legs syndrome, include differences in prevalence, pathophysiology, clinical presentation, and response to therapy.

Publication Type: Review

Gen Hosp Psychiatry. 2006 Sep-Oct;28(5):443-5.

Posttraumatic Kleine-Levin syndrome.

Cheung G.

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Publication Type: Case Reports

Sleep. 2006 Aug 1;29(8):1091-3.

Episodic diencephalic hypoperfusion in Kleine-Levin syndrome.

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A 22 year-old woman suffered from recurrent episodes of hypersomnia, apathy, and hyperphagia. The symptoms occurred 3 to 4 times per year, and each attack lasted 2 to 3 weeks. 99mTc-ethylcysteinate dimer brain single photon emission computed tomography (SPECT) was performed during symptomatic and asymptomatic periods. To localize brain regions with perfusion changes during symptomatic period, asymptomatic SPECT was subtracted from symptomatic SPECT. The subtracted SPECT showed significant hypoperfusion in the left hypothalamus, bilateral thalami, basal ganglia, bilateral medial and dorsolateral frontal regions, and left temporal lobe during the symptomatic period. These cerebral hypoperfusion areas support the diencephalic hypothesis and clinical symptoms of Kleine-Levin syndrome.

Publication Type: Case Report

Nippon Rinsho. 2006 May 28;Suppl 1:19-22.

[Kleine-Levin syndrome] [Article in Japanese]

Shintani M, Nishimura H.

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Publication Type: Review

(2005)

Med J Aust. 2005 Dec 5-19;183(11-12):670-1.

Ailing allegories and sickly stories: the quest for pathology in children's literature.

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Brain. 2005 Dec;128(Pt 12):2763-76. Epub 2005 Oct 17.

Kleine-Levin syndrome: a systematic review of 186 cases in the literature.

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Kleine-Levin syndrome (KLS) is a rare disorder with symptoms that include periodic hypersomnia, cognitive and behavioural disturbances. Large series of patients are lacking. In order to report on various KLS symptoms, identify risk factors and analyse treatment response, we performed a systematic review of 195 articles, written in English and non-English languages, which are available on Medline dating from 1962 to 2004. Doubtful or duplicate cases, case series without individual details and reviews (n = 56 articles) were excluded. In addition, the details of 186 patients from 139 articles were compiled. Primary KLS cases (n = 168) were found mostly in men (68%) and occurred sporadically worldwide. The median age of onset was 15 years (range 4-82 years, 81% during the second decade) and the syndrome lasted 8 years, with seven episodes of 10 days, recurring every 3.5 months (median values) with the disease lasting longer in women and in patients with less frequent episodes during the first year. It was precipitated most frequently by infections (38.2%), head trauma (9%), or alcohol consumption (5.4%). Common symptoms were hypersomnia (100%), cognitive changes (96%, including a specific feeling of derealization), eating disturbances (80%), hypersexuality (43%), compulsions (29%), and depressed mood (48%). In 75 treated patients (213 trials), somnolence decreased using stimulants (mainly amphetamines) in 40% of cases, while neuroleptics and antidepressants were of poor benefit. Only lithium (but not carbamazepine or other antiepileptics) had a higher reported response rate (41%) for stopping relapses when compared to medical abstention (19%). Secondary KLS (n = 18) patients were older and had more frequent and longer episodes, but had clinical symptoms and treatment responses similar to primary cases. In conclusion, KLS is a unique disease which may be more severe in female and secondary cases.

Publication Type: Review

Sleep. 2005 Aug 1;28(8):955-60.

[See comment in: Sleep. 2005 Aug 1;28(8):915-6.]

SPECT findings in the Kleine-Levin syndrome.

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STUDY OBJECTIVES: The Kleine-Levin Syndrome, is a rare disorder with onset during teenage years, but little is known on etiopathogenesis. Seven subjects with Kleine-Levin Syndrome accumulated over time had systematic SPECT studies during (n=5) and out (n=7) of the symptomatic period. **SUBJECTS:** Seven boys with symptom onset between 11 and 17 years of age and at least 2 episodes per year were followed for a mean of 6 years. **METHODS:** Electroencephalogram awake-asleep, computed tomography scan, and magnetic resonance imaging studies were performed before Tc-99m ECD single photon emission tomography (SPECT) obtained during day 4 or 5 (n=5) and at least 1 month away from the symptomatic period (n=7). **RESULTS:** All imaging tests except SPECT were normal. Hypoperfusion of both thalami were seen during the symptomatic period that completely disappeared during the asymptomatic period. Hypoperfusion in other regions were also noted in some, but not all subjects. They persisted during the asymptomatic period in 2 cases over the temporal lobe (2/7 cases), frontal lobe (1/7 cases), and basal ganglia (1/7 cases). The largest amount of persistent hypoperfusion was seen in the subject with longest clinical evolution. **CONCLUSION:** Hypoperfusion of the thalamus is a consistent finding during the symptomatic period, but perfusion abnormalities may persist even during the asymptomatic period. The longer the duration of the syndrome, the more extended the hypoperfusion regions during the asymptomatic period.

Sleep. 2005 Aug 1;28(8):915-6.

[Comment on: Sleep. 2005 Aug 1;28(8):955-60.]

The Kleine-Levin syndrome: a paramedian thalamic dysfunction?

Billiard M.

Publication Type: Comment, Editorial

J Coll Physicians Surg Pak. 2005 Jan;15(1):46-7.

Kleine-Levin syndrome.

Mapari UU, Khealani BA, Ali S, Syed NA.

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Kleine-Levin Syndrome (KLS) is a rare disorder of uncertain etiology, characterized by recurring episodes of undue sleepiness lasting for days. We report a case of young female presenting with episodes of undue sleep along with hypersexuality and excessive food intake, who improved significantly on lithium and valproate. KLS should be considered in young patients, who present with episodes of undue somnolence.

Publication Type: Case Report

Br J Sports Med. 2005 Feb;39(2):e7; discussion e7.

Kleine-Levin syndrome: a unique cause of fatigue in an athlete.

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Kleine-Levin syndrome (KLS) is a rare disorder characterised, most notably, by periodic episodes of hypersomnolence and hyperphagia. Associated features of the disorder include a lack of concentration, mood changes, and anxiety. Laboratory tests may show slight changes in the electroencephalogram. However, clinical presentation and laboratory tests are normal during asymptomatic intervals. KLS most often presents in adolescent males, with complete recovery by the 3rd to 4th decade of life. Possible precipitating factors include excessive workload, febrile illness, and respiratory infections. Presented is a classical case of KLS in an adolescent male athlete. The patient's history, complete laboratory results, and symptoms are discussed. Possible treatments for this disorder are also mentioned, along with diagnostic criteria.

Publication Type: Case Report

(2004)

Eur Psychiatry. 2004 Dec;19(8):521-2.

Posttraumatic Kleine-Levin syndrome: a case report.

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Publication Type: Case Report

Pediatr Clin North Am. 2004 Feb;51(1):89-115.

Neurologic disorders masquerading as pediatric sleep problems.

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Abs: Neurologic disorders may present or masquerade as pediatric sleep problems and fool the pediatrician, which may delay diagnosis and treatment. Many of the sleep problems in children with neurologic disorders arise directly from primary dysfunction or delayed maturation of their sleep-wake regulation systems. It is important to realize that nocturnal frontal lobe seizures or cluster headaches can be mistaken for night terrors, and craniopharyngiomas or myotonic dystrophy may present as narcolepsy-cataplexy. Hypothalamic dysfunction may explain not only the impaired circadian rhythm disorders in children with profound mental retardation but also excessive sleepiness and hyperphagia in Prader-Willi and Kleine-Levin syndromes. Intellectually challenged children perform better, learn more, and are better behaved with sufficient restorative sleep.

Publication Type: Review

(2003)

J Neurol Neurosurg Psychiatry. 2003 Dec;74(12):1667-73.

CSF hypocretin-1 levels in narcolepsy, Kleine-Levin syndrome, and other hypersomnias and neurological conditions.

✓_ Dauvilliers Y, Baumann CR, Carlander B, Bischof M, Blatter T, Lecendreux M, Maly F, Besset A, Touchon J, Billiard M, Tafti M, Bassetti CL.

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OBJECTIVE: To determine the role of CSF hypocretin-1 in narcolepsy with and without cataplexy, Kleine-Levin syndrome (KLS), idiopathic and other hypersomnias, and several neurological conditions. **PATIENTS:** 26 narcoleptic patients with cataplexy, 9 narcoleptic patients without cataplexy, 2 patients with abnormal REM-sleep-associated hypersomnia, 7 patients with idiopathic hypersomnia, 2 patients with post-traumatic hypersomnia, 4 patients with KLS, and 88 patients with other neurological disorders. **RESULTS:** 23 patients with narcolepsy-cataplexy had low CSF hypocretin-1 levels, while one patient had a normal hypocretin level (HLA-DQB1*0602 negative) and the other two had intermediate levels (familial forms). One narcoleptic patient without cataplexy had a low hypocretin level. One patient affected with post-traumatic hypersomnia had intermediate hypocretin levels. The KLS patients had normal hypocretin levels while asymptomatic, but one KLS patient (also affected with Prader-Willi syndrome) showed a twofold decrease in hypocretin levels during a symptomatic episode. Among the patients without hypersomnia, two patients with normal pressure hydrocephalus and one with unclear central vertigo had intermediate levels.

CONCLUSION: Low CSF hypocretin-1 is highly specific (99.1%) and sensitive (88.5%) for narcolepsy with cataplexy. Hypocretin ligand deficiency appears not to be the major cause for other hypersomnias, with a possible continuum in the pathophysiology of narcolepsy without cataplexy and idiopathic hypersomnia. However, partial hypocretin lesions without low CSF hypocretin-1 consequences cannot be definitely excluded in those disorders. The existence of normal hypocretin levels in narcoleptic patients and intermediate levels in other rare aetiologies needs further investigation, especially for KLS, to establish the functional significance of hypocretin neurotransmission alterations.

Acta Neurol Scand. 2003 Nov;108(5):363-7.

Short-term memory dysfunction in Kleine-Levin syndrome.

_ Landtblom AM, Dige N, Schwerdt K, Safstrom P, Granerus G.

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BACKGROUND : A previous investigation at our department of a young man with typical Kleine-Levin syndrome revealed short-term memory dysfunction as well as hypoperfusion of the temporal lobes on single photon emission tomography (SPECT) (CERETEC) examination, 6 and 7 years after recovery, suggesting long lasting or even permanent cerebral dysfunction. **PATIENTS AND METHODS :** We investigated four cases with classical adolescent Kleine-Levin syndrome characterized by hypersomnia and typical associated symptoms. We used neuropsychological testing and SPECT (CERETEC) of the brain. The results from the previous report related to above is included. **RESULTS :** Examination with SPECT (CERETEC) during remission revealed hypoperfusion of the temporal lobes and fronto-temporal region in two of four cases.

There were normal findings in two. Neuropsychological testing performed during remission showed reduction in the short-term memory capacity in all four cases.

CONCLUSION : It is striking that all the cases investigated showed short-term memory dysfunction. One patient who had recovered from paroxysmal symptoms (hypersomnia attacks and bulimia) 6 years earlier showed progress in the short-term memory dysfunction. A pathologic condition in the temporal lobes may be suspected in Kleine-Levin syndrome.

Type: SPECT

Rev Neurol. 2003 Jul 16-31;37(2):200.

Comment on: Rev Neurol. 2002 Sep 16-30;35(6):531-3

[Kleine Levin syndrome and sleep-related eating disorder]

[Article in Spanish]

_ Ortega-Albas JJ, de Entrambasaguas-Barreto M.

Type: Comment Letter

Neuropediatrics. 2003 Jun;34(3):113-9.

The Kleine-Levin syndrome - effects of treatment with lithium

_ Poppe M, Friebel D, Reuner U, Todt H, Koch R, Heubner G.

Department of Neuropediatrics, Technical University Dresden, Dresden, Germany.

Kleine-Levin syndrome (KLS) is a rare disorder which affects mainly adolescents.

Periods of extreme somnolence alternate with megaphagia, psychomental changes and behavioural symptoms. The cause and pathogenesis of KLS remains unknown. Several treatments have been tried and recently lithium has been proposed for a prophylactic use in single cases. In view of the rarity of KLS, long-term results of lithium therapy have not been described yet. We report the clinical course of five adolescents with KLS who were treated with lithium. All patients showed significant EEG and polysomnographic changes during the episodes and had normal results in the interval. All patients had relapses while being treated with lithium. But episodes of hypersomnia under lithium therapy were shorter and monosymptomatic with lack of behavioural symptoms. Statistical modelling showed that the risk for a relapsing episode under maintenance of lithium drops per months of therapy from 100 % to 93 %, and furthermore that the maintenance of lithium shortens the mean duration of episodes to 19 %. No severe side effects were observed. In conclusion, in KLS with a high frequency of episodes and severe behavioural changes lithium may become a treatment option.

Type: Case Report, Review

J Child Neurol. 2003 Jun;18(6):432-3.

Photosensitivity during the hypersomnic phase in a patient with Kleine-Levin syndrome.

_ Papacostas SS.

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The case of an adolescent with Kleine-Levin syndrome is presented who exhibited a consistent and predictable photoparoxysmal self-limited response to intermittent photic

stimulation during all relapses of his hypersomnic phase. A possible cause of this association that has not been previously reported is speculated based on observations concerning the two disorders.

Type: Case Report

Acta Neurol Scand. 2003 Apr;107(4):304-5; author reply 306.

Comment on: Acta Neurol Scand. 2002 Apr;105(4):318-21.

Persistent neuropsychological deficits in the Kleine-Levin syndrome.

_ Fontenelle LF, Mendlowicz MV, Marques C, Mattos P, Versiani M.

Type: Comment Letter

Rev Neurol. 2003 Mar 16-31;36(6):599; author reply 600

Comment on: Rev Neurol. 2002 Sep 16-30;35(6):531-3

[Kleine-Levin syndrome: diagnostic contribution made by brain SPECT]

[Article in Spanish]

_ Peraita-Adrados R.

Type: Comment Letter, SPECT

(2002)

Neurology. 2002 Dec 10;59(11):1739-45

Kleine-Levin syndrome: an autoimmune hypothesis based on clinical and genetic analyses.

✓_ Dauvilliers Y, Mayer G, Lecendreux M, Neidhart E, Peraita-Adrados R, Sonka K, Billiard M, Tafti M.

Neurologie B, Hopital Gui-de-Chauliac, Montpellier, France.

BACKGROUND: Kleine-Levin syndrome (KLS) is a rare disorder of unknown etiology. Pathophysiologic hypotheses include a hypothalamic dysfunction and abnormalities in the central serotonin and dopamine metabolism. Several clinical symptoms also suggest an underlying autoimmune process. OBJECTIVE: To systematically investigate patients with KLS with reference to the available hypotheses. METHODS: The authors collected clinical, polysomnographic, CSF, CT, and MRI records and analyzed gene polymorphisms of HLA-DQB1, tryptophan hydroxylase (TpH), and catechol-O-methyltransferase (COMT) in 30 unrelated patients with KLS and their families. The genotype data were contrasted with data from a normal control population. RESULTS: Only human leukocyte antigen (HLA)-DQB1*0201 allele frequency was significantly increased in patients with KLS. Three patients with KLS but none of the control subjects were DQB1*0201 homozygous. Two affected subjects from the same family were DQB1*0201 homozygous. In 17 DQB1*0201 heterozygous parents, 11 (64.7%) had transmitted this allele, suggesting a preferential transmission. CONCLUSION: These findings, together with the young age at onset, the recurrence of symptoms, and the frequent infectious precipitating factors, suggest an autoimmune etiology for Kleine-Levin syndrome.

Arch Neurol. 2002 Dec;59(12):1959-61

Familial Kleine-Levin syndrome: two siblings with unusually long hypersomnic spells.

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Kleine-Levin syndrome is a rare, sporadic disorder, with discrete spells of hypersomnolence occurring during adolescence, variously accompanied by megaphagia, behavioral changes, psychosis, and mild autonomic symptoms. Familial cases have not previously been reported. We describe 2 siblings who shared uncharacteristically prolonged episodes of hypersomnolence, and the HLA-DR2 haplotype. In one patient, levels of cerebrospinal fluid orexin (hypocretin) during an attack were normal. The presence of an increased sleep drive, despite the occurrence of large amounts of ostensibly restorative sleep, suggests the possible existence of a disorder of sleep satiety.

Type: Case Report

Indian J Pediatr. 2002 Nov;69(11):999-1000

Kleine-Levin syndrome and encephalitis.

_ Sethi S, Bhargava SC.

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Kleine-Levin Syndrome is characterized by hypersomnolence, hyperphagia and sexual disinhibition. The article reported a case of 10-year-old boy with a two-week history of altered sensorium, irrelevant talks, markedly increasing appetite and tendency to sleep most of the times. Immediately preceding to it the child had been an episode of enteric fever confirmed by the serological tests.

Type: Case Report

Rev Neurol. 2002 Sep 16-30;35(6):531-3.

Comment in: Rev Neurol. 2003 Jul 16-31;37(2):200.

Rev Neurol. 2003 Mar 16-31;36(6):599; author reply 600.

[Kleine-Levin syndrome: contribution of brain SPECT in diagnosis]

[Article in Spanish]

_ Arias M, Crespo Iglesias JM, Perez J, Requena- Caballero I, Sesar-Ignacio A, Peleteiro-Fernandez M.

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INTRODUCTION: Kleine Levin syndrome is an infrequent disorder of uncertain aetiopathogenesis that usually affects adolescent males, producing drowsiness and alterations in behaviour, appetite and sexuality. We report a clinically typical case in which the brain SPECT showed right frontal hypoperfusion. CASE REPORT: Male aged 17, right handed, who presented drowsiness, apathy, alterations in his sexual behaviour (masturbations in public and attempted assault of women) and hyperphagia, which coincided with his undergoing an emotionally stressful period in his life. Brain SPECT revealed reduced flow in the right frontal lobe, although MRI and CSF study were

normal. The polysomnographic study revealed a destructured pattern of sleep, with a reduction in phases III and IV and in REM sleep. He was treated with lithium and evolved favourably without any relapses during the 18 month follow up.

CONCLUSIONS: Cases of Kleine Levin syndrome with structural lesions have been reported, but most of them are idiopathic. A disorder in the hypothalamus and the limbic system is suggested. In our case, the findings from the brain SPECT confirmed a non dominant hypoperfusion of the frontal lobe, which could result from a phenomenon of diaschisis brought about by a diencephalic dysfunction.

Type: Case Report, SPECT

Eur Psychiatry. 2002 Jul;17(4):232-3.

Efficacy of lithium treatment in Kleine-Levin syndrome.

_ Muratori F, Bertini N, Masi G.

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The Kleine-Levin syndrome (KLS) is characterized by periodic, sudden-onset episodes of hypersomnia, compulsive hyperphagia, and behavioral-emotional symptoms, lasting from a few days to a few weeks, with complete remission in the intercritical periods. We report on efficacy of lithium treatment in a highly recurring form of the disorder in a 17-year-old male adolescent. The decreasing severity of the disorder paralleled the progressive increase of lithium dosage, up to 0.9 mEq l(-1). Implications regarding the pharmacological treatment of this neglected disorder are discussed.

Type: Case Report

Z Kinder Jugendpsychiatr Psychother. 2002 Aug;30(3):185-98.

[Sleep disorders and child and adolescent psychiatric illnesses]

[Article in German]

_ Hagenah U.

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As a symptom of many psychiatric disorders of childhood and adolescence, sleep disturbances often complicate the course and treatment of the underlying disorder. A somatic aetiology, e.g., as in Kleine-Levin syndrome or narcolepsy, may lead to diagnostic misinterpretations. It is not clear whether specific alterations of sleep architecture already exist in this age group and are thus trait markers for psychiatric disorders. Although it is well-known that sleep problems in adults, especially insomnia, are important in the later development of depressive syndromes, it is not clear whether persistent sleep problems during childhood constitute markers of vulnerability for psychiatric disorders. This review demonstrates interactions between sleep disturbances and psychiatric disorders of childhood and adolescence and their importance for assessment and therapy.

Type: Review

Rev Neurol (Paris). 2002 May;158(5 Pt 1):593-5.

[SPECT-identified hypoperfusion of the left temporomesial structures in a Kleine-Levin syndrome]

[Article in French]

_ Portilla P, Durand E, Chalvon A, Habert M, Navelet Y, Prigent A, Landrieu P.
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France.

A 13-year-old boy developed typical features of Kleine-Levin syndrome. Routine investigations and MRI were normal. SPECT, performed both during an attack and during a symptom-free period, demonstrated clear hypoperfusion of the left mesiotemporal structures. The possible implications of this finding are discussed.

Type: Case Report; SPECT

Indian J Pediatr. 2002 May;69(5):451.

Kleine-Levin syndrome following acute non-specific encephalitis.

_ Sethi S, Bhargava SC.

Type: Case Report Letter

Acta Neurol Scand. 2002 Apr;105(4):318-21.

Comment in: Acta Neurol Scand. 2003 Apr;107(4):304-5; author reply 306.

A case of Kleine-Levin syndrome examined with SPECT and neuropsychological testing.

_ Landtblom AM, Dige N, Schwerdt K, Safstrom P, Granerus G.

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A case of Kleine-Levin syndrome with typical periodic hypersomnia and bulimia was diagnosed. On examination with single photo emission tomography (SPECT) (CERETEC) during a relapse period and 2 weeks later there was marked cortical hypoperfusion of the frontal and temporal lobes, especially on the left side as well as in the right parietal lobe. Neuropsychological testing performed 1 week after a relapse showed a reduction in encoding to memory function of verbal learning indicating neocortical damage of the left fronto-temporal region. A follow-up 2 months later after the patient had spontaneously recovered showed only a slight left fronto-temporal disturbance. CT and MRI of the brain were normal although the MRI showed a large and asymmetric mamillary body. Neuropsychological testing 6 years after recovery showed pronounced reduction in short-time verbal and visual memory. Seven years after recovery SPECT demonstrated a normalized frontal perfusion but still a slight hypoperfusion in the left temporal lobe. Our results correlate to autopsy findings in two cases described previously.

Type: Case Report, SPECT

(2001)

J Sleep Res. 2001 Dec;10(4):337-41.

Clinical and polysomnographic characteristics of 34 patients with Kleine-Levin syndrome.

✓_ Gadoth N, Kesler A, Vainstein G, Peled R, Lavie P.

Department of Neurology, Sapir Medical Center, Meir General Hospital, Kfar Saba and

the Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel.

There is only scant information on sleep characteristics and long-term follow-up in patients with Kleine-Levin syndrome (KLS). This study describes the clinical course, results of polysomnography and long-term follow-up in a relatively large group of patients with KLS. During the years 1982-97, we encountered 34 patients (26 males and eight females) with KLS. We were able to obtain the original polysomnographs from 28 males and four females. In 25 patients, data regarding their present state of health were obtained. Fourteen agreed to be present at a detailed interview and examination while 11 gave the information by phone. The mean age at onset was 15.8 +/- 2.8 years and the mean diagnostic delay, 3.8 +/- 4.2 years. The mean duration of a single hypersomnolent attack was 11.5 +/- 6.6 days. The main abnormal findings extracted out of 35 polysomnographs obtained from 32 patients during and/or in-between attacks included: decreased sleep efficiency, and frequent awakenings from sleep stage 2. All 25 patients reported present perfect health, with no evidence of behavioral or endocrine dysfunction. In adolescents with periodic hypersomnia, the diagnosis of KLS should be explored. Sleep recordings during a hypersomnolent period will often show frequent awakenings from sleep stage 2. The long-term prognosis is excellent.

Sleep Med Rev. 2001 Oct;5(5):349-358.

Idiopathic Hypersomnia.

_ Billiard M, Dauvilliers Y.

Department of Neurology B, Gui de Chauliac Hospital, Montpellier, France

In contrast to narcolepsy and the Kleine-Levin syndrome, idiopathic hypersomnia is a recently described sleep disorder. Absence of associated clinical features such as cataplexy or megaphagia and characteristic polysomnographic features such as sleep-onset REM episodes render positive diagnosis more uncertain in idiopathic hypersomnia than in the two former conditions. Consequently there has been an unfortunate tendency to label all difficult to classify cases of excessive daytime sleepiness as idiopathic hypersomnia. At present due to the description of new disorders such as upper airway resistance syndrome, narcolepsy without cataplexy, delayed sleep phase syndrome, all of which were formerly confused with idiopathic hypersomnia and the clear identification of a "polysymptomatic" or "classic" form of idiopathic hypersomnia, the limits of the disorder become more precise. Still there are a number of cases of isolated excessive daytime sleepiness with no prolonged night sleep, no difficulty waking up, which lay between narcolepsy and genuine idiopathic hypersomnia. Thus there is a definite need to further develop laboratory investigations to help identify and classify these cases. Moreover pathophysiology and pathogenesis are still in their infancy and efforts have to be pursued in this direction. Treatment has not made consistent progress except for the use of a new wake promoting compound, modafinil, which has not yet been evaluated in controlled studies.

Psychosomatics. 2001 Jul-Aug;42(4):350-2.

A case report of Kleine-Levin syndrome in an adolescent girl.

_ Janicki S, Franco K, Zarko R.

Department of Psychiatry, Cleveland Clinic Foundation, OH 44195, USA.

Type: Case Report

Am J Respir Crit Care Med. 2001 May;163(6):1310-3.

"Why am I sleepy?": sorting the somnolent.

_ Douglas NJ.

The University of Edinburgh, Respiratory & Sleep Medicine Unit, Department of Medicine, Royal Infirmary, Edinburgh EH3 9YW, Scotland, UK. n.j.douglas@ed.ac.uk

Type: Review

Rev Neurol (Paris). 2001 Mar;157(3):344-5.

[Kleine-Levin syndrome]

[Article in French]

_ Bouchard C, Levasseur M.

Service de medecine-neurologie, Centre Hospitalier d'Orsay, Orsay, France.

Type: Case Report

(2000)

J Clin Neurophysiol. 2000 Sep;17(5):519-22

Multiple sleep latency test and polysomnography in diagnosing Kleine-Levin syndrome and periodic hypersomnia.

_ Rosenow F, Kotagal P, Cohen BH, Green C, Wyllie E.

Section of Pediatric Epilepsy, Department of Neurology, The Cleveland Clinic Foundation, Ohio, USA.

Kleine-Levin syndrome and periodic hypersomnia are often misdiagnosed initially because there is no objective test for these conditions. To determine the value of the Multiple Sleep Latency Test and polysomnography in this respect, the authors studied four patients with Kleine-Levin syndrome or periodic hypersomnia who had taken the Multiple Sleep Latency Test and undergone polysomnography during the symptomatic episode and/or during the asymptomatic interval. During but not between symptomatic episodes, the Multiple Sleep Latency Test revealed abnormal sleep latencies in all patients, and polysomnography revealed increased rapid eye movement propensity in one patient and a reduction in delta-sleep in two patients. In conclusion, the Multiple Sleep Latency Test and polysomnography are useful in diagnosing Kleine-Levin syndrome and periodic hypersomnia, especially when administered in a standardized fashion during and after the symptomatic period. The authors recommend that polysomnography and the Multiple Sleep Latency Test be performed no earlier than the second night after the onset of a symptomatic episode and the following day to reveal maximal hypersomnolence, and more than 2 weeks after a symptomatic episode to represent the asymptomatic interval.

Type: Case Report

Encephale. 2000 Jul-Aug;26(4):71-4.

[Klein-Levin syndrome: a neurological disease with psychiatric symptoms]

[Article in French]

Minvielle S.

Service de Psychiatrie de l'Hopital d'Instruction des Armees du Val-de-Grace, Paris. Kleine-Levin syndrome belongs to the recurrent hypersomnias group. It is a rare and benignant disease, occurring in young men 10 to 25 years old. The diagnosis is first clinical and the hypersomniac episodes, joined to psychiatric symptoms, are irregularly recurrent during few years. Diagnosis is uneasy during the first episode and in the attenuated forms... 500 cases have been described all around the world but it's highly likely that many patients haven't been listed. This syndrome, just like Gelineau disease, stands in the group of primary pathological hypersomnias. In a clinical point of view, the cardinal and constant symptom is hypersomnia. Psychiatric symptoms can be irregularly joined: megaphagia, sexual behavioural disorders, thymic disorders, personality modifications. The clinical examination is poor and aspecific. During an hypersomniac episode, a polygraphic recording during 24 or 48 hours will give diagnosis informations (fragmented and unstable sleep, reduction in stages 3 and 4 of non-REM sleep, reduction in REM sleep latency) and a biological and radiological evaluation will be necessary to exclude organic etiology (tumoral progres, infectious disease...). In a therapeutic point of view, prescription of psychostimulant drugs is recommended during fits and some treatments are used in a preventive way (lithium and carbamazepine).

Type: Case Report

Eur Psychiatry. 2000 Jun;15(4):231-5.

The Kleine-Levin syndrome. Report of a case and review of the literature.

_ Papacostas SS, Hadjivasilis V.

The Cyprus Institute of Neurology and Genetics, Nicosia, Cyprus.

Kleine-Levin syndrome is a rare self-limited disorder which usually affects adolescent males and is characterized by episodic hypersomnia, increased appetite, and behavioral/psychiatric disturbances. Individuals are normal between the attacks. The case of an adolescent boy is presented who suffered from recurrent sleepiness, hyperphagia, and behavioral disturbances such as rocking, punching and pacing, and was originally misdiagnosed as suffering from encephalitis. Before the diagnosis of Kleine-Levin was given, the patient underwent unnecessary investigations and treatment which, in turn, complicated his clinical condition both physically as well as psychologically. In the course of five years he had four such episodes which appeared to have progressively milder manifestations. Between episodes he was normal. It is important that the diagnosis is suspected early, especially in adolescent males who present with recurrent episodes of somnolence, increased appetite, and abnormal behavior, since it most often represents a benign and self-limited entity and does not warrant extensive investigations or treatment. It is also important to distinguish this syndrome from more serious organic and psychiatric diseases with more serious prognoses. The differential diagnosis of this syndrome is discussed and a review of the literature is presented including evidence and hypotheses regarding its pathophysiology.

Type: Case Report

Arq Neuropsiquiatr. 2000 Jun;58(2B):531-4.

Neuropsychological sequelae in Kleine-Levin syndrome: case report.

_ Fontenelle L, Mendlowicz MV, Gillin JC, Mattos P, Versiani M.
Institute of Psychiatry, Federal University of Rio de Janeiro, Rio de Janeiro, Brazil.
lfontenelle@bigfoot.com

Kleine-Levin syndrome is characterized by periodic hypersomnia, hyperphagia, sexual disinhibitions and behavioral disturbances. The prognosis is generally benign, with normal cognitive and social functions after the episodes. We describe a typical case of Kleine-Levin syndrome associated with apparent academic decline, neuropsychological sequelae and personality alterations after the second episode of the illness. Further research in the natural history of Kleine-Levin syndrome is needed, for example, to determine whether early intervention would improve long-term prognosis.

Type: Case Report

Sleep 2000 Jun 15;23(4):563-7

Kleine Levin syndrome (KLS) in young females.

✓_ Kesler A, Gadoth N, Vainstein G, Peled R, Lavie P

Department of Neurology, Sapir Medical Center, Meir General Hospital, Kfar Saba, the Sackler Faculty of Medicine, Tel-Aviv University, Israel.

REVIEW: During the years 1982-1998, we encountered 7 adolescents and one young woman suffering from KLS. In 4 patients, hypersomnolence was accompanied by hyperphagia and hypersexuality, while in the remaining 4, recurrent hypersomnia was the only symptom. Mean age at onset of hypersomnolent attacks was 15.1+/-3.5 yrs. The mean duration of a hypersomnolent attack was 9.9+/-5.4 days, and the number of attacks per patient was 6.2+/-3.4. Polysomnographic recordings from 3 patients in between attacks, and from one patient during an attack, showed relatively normal sleep structure with decreased sleep efficiency due to numerous awakenings from sleep stage 2. Besides the recurrent hypersomnia, all patients enjoyed good health, with no evidence of behavioral or endocrine dysfunction. Similarly aged males with KLS from our clinic and previously reported females, had similar clinical features.

Psychiatry. 2000 Spring;63(1):101-3.

Comment on: Psychiatry. 2000 Spring;63(1):93-100.

The mind-body harmony.

_ Eth S, Ladds BJ.

Department of Psychiatry, Saint Vincent's Hospital, New York, NY 10011, USA.

Psychiatry 2000 Spring; 63(1):93-100

The Kleine-Levin syndrome as a neuropsychiatric disorder: a case report.

_ Masi G, Favilla L, Millepiedi S

Division of Child Neurology and Psychiatry, University of Pisa, Italy.

masi@inpe.unipi.it

AB: The Kleine-Levin syndrome (KLS) is characterized by periodic, sudden-onset episodes of hypersomnia, compulsive hyperphagia, and behavioral-emotional disorders (typically indiscriminate hypersexuality, irritability, impulsive behaviors), lasting from a few days to a few weeks, with almost complete remission in the intercritical periods. Depression, confusion, and thought disorders are frequently associated with the critical symptomatology, and they may suggest other psychiatric diagnoses (schizophrenia,

mood disorder, conversion disorder) or a substance abuse. A diencephalic-hypothalamic dysfunction is suspected, even if this composite symptomatology cannot easily be linked to a simple mechanism. The aim of this article is to illustrate problems in differential diagnosis, using a case approach. History, course, and therapeutic intervention in a 21-year-old patient with KLS, associated with a clear psychiatric symptomatology and a critical affective pattern, is reported. Psychiatric correlates of KLS are discussed, including the relationship with affective disorders and the possible emotional impact of the attacks. Implications regarding a combined psychological and pharmacological treatment are also discussed.

J Clin Psychiatry 2000 Mar;61(3):215

Treatment of Kleine-Levin syndrome: melatonin on the starting block.

✓_ Kornreich C, Fossion P, Hoffmann G, Baleriaux M, Pelc I
Letter Comment on: J Clin Psychiatry 1997 Sep;58(9):383-8

Neuropsychiatry Neuropsychol Behav Neurol 2000 Apr;13(2):140-2

Kleine-Levin syndrome and psychosis: observation from an unusual case.

__ Lu ML, Liu HC, Chen CH, Sung SM

Department of Adult Psychiatry, Taipei City Psychiatry Center, and Shin Kong Wu Ho-Su Memorial Hospital, Taiwan.

OBJECTIVE: This study evaluated the possible pathologic relation between Kleine-Levin syndrome (KLS) and mood disorders. BACKGROUND: A 28-year-old man with a remote history of KLS had the sudden onset of a manic episode with psychotic features after the end of hypersomnolence. METHOD: The patient received an extensive laboratory examination, including single photon emission computed tomography and magnetic resonance imaging. RESULTS: Single photon emission computed tomography showed decreased tracer perfusion in the basal ganglion, hypothalamus, and right frontotemporal region. Magnetic resonance imaging revealed a cystic lesion in the pineal region. CONCLUSIONS: Hypothalamic dysfunction has been described in KLS and mood disorders, but pineal gland dysfunction has been mentioned only rarely. The clinical and neuroimaging findings suggest the need for further study of KLS.
Type: SPECT

(1999)

J Am Acad Child Adolesc Psychiatry 1999 Jul;38(7):791-2

Carbamazepine for Kleine-Levin syndrome.

__ Mukaddes NM, Kora ME, Bilge S
Type: Letter

Child Psychiatry Hum Dev 1999 Spring;29(3):253-8

The psychiatric symptomatology in Kleine-Levin syndrome.

__ Mukaddes NM, Alyanak B, Kora ME, Polvan O
Istanbul University, Turkey.

AB: The Kleine-Levin syndrome is a rare disorder with its main symptoms being periodic hypersomnolence and excessive eating accompanied by behavioral changes. The dominance of the behavioral and psychological symptoms may obscure the diagnosis. In this article the diagnostic process and the psychiatric symptomatology of two adolescent male patients with Kleine-Levin syndrome is discussed.

Eur J Endocrinol 1999 Feb;140(2):140-2

Kleine-Levin and Munchausen syndromes in a patient with recurrent acromegaly.

__ Jungheim K, Badenhoop K, Ottmann OG, Usadel KH

Department of Medicine I, Klinikum of the J W Goethe University, Frankfurt, Germany.

AB: Hypothalamic disease often affects the patients' personality and this also applies to pituitary tumors with suprasellar extension. We report on a patient with a 12-year history of recurrent acromegaly, treated with three transphenoidal operations, single field radiation therapy and bromocriptine/octreotide administration. During the course of follow-up she presented with self-inflicted anemia and Kleine-Levin syndrome (hypersomnia, hyperphagia and hypersexuality). Furthermore, she developed post-radiation necrosis within the right temporal lobe. Whether her neurological and personality disorders result - at least partially - from the acromegaly or the temporal lobe necrosis remains unclear.

(1998)

J Am Acad Child Adolesc Psychiatry 1998 Dec;37(12):1245

Light therapy for Kleine-Levin syndrome.

__ Crumley FE

Type: Letter

Arq Neuropsiquiatr 1998 Sep;56(3B):650-4

Kleine-Levin syndrome. Clinical course, polysomnography and multiple sleep latency test. Case report.

__ Reimao R, Shimizu MH

CDS-Centro de Disturbios do Sono, Sao Paulo, Brasil. reimaor@apm.org.br

AB: A case of Kleine-Levin syndrome, with chronic severe periodic hypersomnia is described in a 17-year-old female. The first episode started when she was 15 years old. The episodes were characterized by periodic hypersomnia accompanied by hyperphagia, lasting 5 days, and repeating at 28 to 60 day intervals. The severity of hypersomnia prevented her from attending school activities. Outside the hypersomnia periods, she was asymptomatic. EEG, brain computerized tomography and brain nuclear magnetic resonance were normal; all-night polysomnography, Multiple Sleep Latency Test (MSLT) and Epworth Sleepiness Scale (ESS) were within normal limits. During the period of hypersomnolence, polysomnography showed short sleep latency and short REM latency. MSLT mean sleep latency was 1.8 min; and REM period was present in one subtest; the ESS was markedly elevated.

J Neurol Sci 1998 May 7;157(2):214-6

Kleine-Levin syndrome and Parkinsonian symptoms--a case report.

___ Muller T, Kuhn W, Bornke C, Buttner T, Przuntek H

Department of Neurology, St. Josef-Hospital, Ruhr-University of Bochum, Germany.

AB: We report the case of a 54-year-old male patient, who developed the symptoms of Kleine-Levin syndrome (KLS) at the age of fifty. Four years later Parkinsonian symptoms (PS) appeared. The possible relationship between both KLS and PS, e.g. regarding neuroendocrinological similarities, will be discussed to explain the subsequent onset of PS after the manifestation of KLS in this patient.

Sleep 1998 May 1;21(3):278-84

Endocrinological and polysomnographic findings in Kleine-Levin syndrome: no evidence for hypothalamic and circadian dysfunction.

___ Mayer G, Leonhard E, Krieg J, Meier-Ewert K

Hephata Klinik Schwalmstadt-Treysa, Germany.

AB: Five subjects--four men, ages 17-28, and one woman, age 30--with Kleine-Levin syndrome were investigated during symptomatic (SP) and asymptomatic (ASP) periods. Investigations comprised medical history, MRI, polysomnography, 24-hour hormone profile of human growth hormone, melatonin, TSH, cortisol and FSH (in the woman only) assessed every 2 hours, actimetry, and sleep logs. Medical history confirmed presence of the three symptoms diagnostic of typical Kleine-Levin syndrome: hypersomnia, excessive food intake, and psychic alteration. MRIs of the brain were normal in all patients. Symptomatic periods were triggered by unspecific events, such as infection, sleep deprivation, and alcohol. Polysomnography revealed low sleep efficiency during SPs, decreased amount of slow-wave sleep, and high frequency of stage shifts, indicating sleep fragmentation. Mean 24-hour growth hormone levels were reduced during the SPs in only two patients. Their hGH peaks were dissociated from slow-wave sleep during attacks and intervals, often occurring during wake time. Twenty-four-hour melatonin levels were increased during the SPs in all patients, but were lower in two patients during the nocturnal sleep period. Cortisol, TSH and FSH did not reveal important differences between attacks and intervals. Except for hGH, all hormones had normal circadian excretion during symptomatic and asymptomatic periods. Amplitude of nocturnal activity as assessed by actimetry was significantly increased in two patients, whereas amplitude of daytime activity was significantly reduced in three patients. Actimetry and sleep logs demonstrated prolonged sleep phases during SPs. Our investigation could confirm changes of sleep structure described in the literature. The neuroendocrinological findings could not confirm decreased hGH and cortisol and increased TSH levels during SPs, as previously reported in single cases by many authors. Endocrinological findings did not support an underlying circadian disorder in KLS.

Rev Neurol (Paris) 1998 Feb;154(2):111-29

[Wake disorders. I. Primary wake disorders]

LA: FRENCH

___ Billiard M, Carlander B

Service de Neurologie B, Hopital Gui de Chauliac, Montpellier.

REVIEW: Primary wake disorders encompass various conditions of excessive daytime sleepiness and/or increased nighttime sleep, of unknown origin beginning most often in adolescence and of chronic or recurrent natural history. The best known of these conditions is narcolepsy associating two major clinical features, irresistible episodes of sleep, sleep onset REM periods and an almost constant association with HLA DR2-DQ1. The prevalence of the condition is close to the one of multiple sclerosis but positive diagnosis requires most often over 10 years to be made. The treatment of excessive daytime sleepiness has recently benefited from a new non-amphetamine awakening compound, modafinil, active in 60 to 70 p. 100 of the cases. The treatment of cataplexy still relies on antidepressants, tricyclics or selective serotonin reuptake blockers. Major advances in pathophysiology and pathogeny have been obtained through a natural model of the disease, canine narcolepsy. Pharmacological studies point to the importance of alpha-1 b adrenergic mechanisms in cataplexy, while dopaminergic systems seem more involved in excessive daytime sleepiness. As concerns genetics, the HLA DQB1*0602 gene predisposes to narcolepsy. In the canine model it is mirrored by an autosomal recessive gene showing a strong homology with the human immunoglobulin gene mu-switch. Familial studies have shown that besides typical phenotypes, attenuated forms of the condition characterized by isolated recurrent daytime naps and/or lapses into sleep do exist. In addition one or several other genes may be involved. Narcolepsy is multifactorial, including one or several genes as well as environmental factors. Idiopathic hypersomnia is noted for very long night sleep, difficulty waking up and more or less constant excessive daytime sleepiness. In contrast with narcolepsy sleep is not refreshing. There is no polysomnographic or immunogenetic special feature. Idiopathic hypersomnia is 10 times less frequent than narcolepsy. It is often overdiagnosed due to insufficient knowledge of other causes of excessive daytime sleepiness such as the upper airway resistance syndrome. Modafinil is also of great value in the treatment of idiopathic hypersomnia. In the absence of an animal model, pathophysiology and pathogeny are still poorly understood. Even rarer is the Kleine-Levin syndrome which is easily distinguishable through its recurrent character and its tendency to progressively disappear. It mainly occurs in early adolescent males. Its main features are episodes of sleep of a week duration recurring at a several months' interval along with disturbances of alimentary and sexual behavior. There is no satisfactory treatment of hypersomniac episodes. On the other hand a prophylactic treatment with carbamazepine or lithium may be active. Pathophysiology remains unsettled in spite of some evidence of a hypothalamic functional disturbance.

Nippon Rinsho 1998 Feb;56(2):365-70

[Recurrent hypersomnia]

LA: JAPANESE

— Suzuki H

Nippon Medical School, Department of Neuropsychiatry.

REVIEW: The recurrent hypersomnia is a rare disorder and diagnosed by recurrent episodes of hypersomnia more than 18 hours a day lasting from several days to several weeks. The Kleine-Levin syndrome is a subtype of the recurrent hypersomnia and characterized by excessive eating or hypersexuality. I review the clinical features of

recurrent hypersomnia comparing with our own two cases. The slight consciousness disturbance is seemed to cause the hypersomnic state. Several neurophysiological findings also support this hypothesis. Generally, the prognosis of this disorder is good. The psychophysical factor is seemed as a major factor to induce this disorder, therefore to reduce this factor is one of the most important treatment and the lithium carbonate is also useful as a pharmacotherapy. The mechanism of this disorder is still unknown.

East Afr Med J 1998 Jan;75(1):55-6

Kleine-Levin syndrome: case report.

___ Malomo IO, Lawal RA, Orija OB

Psychiatric Hospital, Yaba-Lagos, Nigeria.

AB: A case of Kleine-Levin syndrome in a fourteen year old junior high school female student is described. The episodic disorder was characterised by excessive sleep, voracious appetite, irritability, confusion and electro-encephalographic changes.

Remission was spontaneous. Prospects for organic aetiology, differential diagnosis, and the importance of early detection are discussed.

J Clin Psychiatry 1998 Jan;59(1):14-9

Clinical and polysomnographic features of sleep-related eating disorder.

___ Winkelman JW

Sleep Disorders Center, Brigham and Women's Hospital, Harvard Medical School, Boston, Mass 02115, USA.

BACKGROUND: Sleep-related eating disorder is a recently described clinical syndrome that combines characteristics of both eating and sleep disorders. Nocturnal partial arousals are followed by rapid ingestion of food and subsequent poor memory for the episode. Only two case series examining this disorder have been published, and both are from the same sleep disorders center in a general hospital. METHOD: The author describes 23 consecutive cases of sleep-related eating disorder that presented to the Sleep Disorders Center at McLean Hospital. All patients were administered at a standardized clinical sleep disorders evaluation followed by a semistructured interview to elicit information regarding characteristics of sleep-related eating disorder.

Polysomnographic evaluation was performed on all patients with clinical histories of sleep-related eating disorder. RESULTS: Eighty-three percent (N = 19) of the 23 patients were female. For most of the patients, the disorder had begun in adolescence (mean +/- SD = 21.6 +/- 10.9 years) and had been chronic, with a mean duration of 15.8 +/- 11.2 years. Nearly all patients reported eating on a nightly basis (1-6 times per night), and all episodes followed a period of sleep. All patients described their eating as "out of control," and two thirds stated that they "binged" during the night. Over 90% (21/23) reported their state at the time of nocturnal eating as "half-awake, half-asleep" or "asleep", and over 90% reported "consistent" or "occasional" amnesia for the event. Nearly half (11/23) of the sample were given a polysomnographic diagnosis of somnambulism. Thirty-five percent (8/23) had a lifetime eating disorder diagnosis.

CONCLUSION: Sleep-related eating disorder appears to be a relatively homogeneous syndrome combining features of somnambulism and daytime eating disorders.

However, no current nosology accurately characterizes these patients. Physicians

should be aware of the existence of the disorder and the value of referring patients with sleep-related eating disorder to a sleep disorders center.

J Neurol Neurosurg Psychiatry 1998 Jan;64(1):113-6

Novel chromosomal aberation in a patient with a unique sleep disorder.

___ Hasegawa Y, Morishita M, Suzumura A

Department of Neurology, Prefectural Tajimi Hospital, Maehata, Japan.

AB: A 45 year old woman presenting with periodic hypersomnia for 17 years is reported on. She would sleep for three weeks followed by the same period awake.

Polysomnography in the somnolent period disclosed an excess of total sleeping time with remarkably increased stage 1, 3/4, and REM sleep, without cataplexy or sleep paralysis. HLA typing was incompatible with narcolepsy or REM sleep behavioural disorder. Her chromosomes showed premature centromere division with chromatid puffing in areas of constitutive heterochromatin, which is exclusively found in the syndrome of infants termed Roberts' syndrome/SC phocomelia. Other laboratory findings were not normal. It is suggested that the present case is a novel sleep disorder related to a unique chromosomal aberration.

(1997)

Beratis S, Frimas C, Kyriazopoulou V, Vagenakis AG PSYCHOPATHOLOGY IN THE **KLEINE-LEVIN** SYNDROME The European Journal of Psychiatry, 11:173-178, 1997

Indian J Pediatr 1997 Sep-Oct;64(5):625-38

Sleep disorders in school-age children.

___ Kotagal S

Department of Neurology, Saint Louis University Health Sciences Center, Missouri 63104, USA.

REVIEW: This article on school age children reviews relevant issues in sleep physiology, the classification of sleep disorders, their clinical and laboratory assessment, some common sleep disorders, the sleep-epilepsy relationship, as well as the impact of daytime sleepiness on higher cortical functions.

Rev Neurol 1997 Sep;25(145):1430-1

[Disorders of excessive somnolence in childhood].

LA: SPANISH

___ Espinar J

Unidad de Sueno, Hospital Universitario San Carlos, Madrid, Espana.

Biol Psychiatry 1997 Aug 15;42(4):299-301

A clinical study of Kleine-Levin syndrome with evidence for hypothalamic-pituitary axis dysfunction.

___ Malhotra S, Das MK, Gupta N, Muralidharan R

Department of Psychiatry, PGIMER, Chandigarh, India.

J Am Acad Child Adolesc Psychiatry 1997 Jul;36(7):868-9

Valproic acid for Kleine-Levin syndrome.

✓_ Crumley FE

Type: Letter

Z Kinder Jugendpsychiatr Psychother 1997 May;25(2):117-21

[Kleine-Levin syndrome--diagnostic and therapeutic problems]

LA: GERMAN

___ Pfeiffer E

Psychosomatische Abteilung der Kinderklinik des Charite-Virchow-Klinikums, Medizinische Fakultät der Humboldt-Universität.

AB: An overview of the literature is given and an attempt is made to describe the diagnostic problems associated with this etiologically unclear disorder. The only successful therapy to date is treatment with lithium. A case study is presented of a 14-year-old boy with typical symptoms. Within a period of 12 months the boy had 6 episodes characterized by hypersomnia and hyperphagia, each lasting between 8 and 14 days. The symptom-free intervals lasted from 10 days to 8 months. Extensive medical and neurological evaluation including single-photon emission-computed tomography (SPECT) showed no abnormalities, and no criteria for another psychiatric disorder were met. After the sixth episode we considered treating the patient with lithium, but this option was rejected by his family. The patient has remained asymptomatic (36-month follow-up). A possible relationship to endogenous psychotic disorders and the role of neurotransmitter metabolism are discussed. Computer-assisted analysis of electroencephalographic activity revealed high signal complexity, which we believe suggests a primary cortical regulatory defect.

Type: SPECT

(1996)

Indian J Med Sci 1996 Dec;50(12):342-4

Kleine-Levin syndrome--a case report with a brief review.

___ Bhatia MS, Choudhary S

Department of Psychiatry, University College of Medical Sciences, Shahdara, Delhi.

J Am Acad Child Adolesc Psychiatry 1996 Aug;35(8):1050-4

Case study: hypersomnolence and precocious puberty in a child with pica and chronic lead intoxication.

✓_ Boris NW, Hagino OR, Steiner GP

Department of Psychiatry, Louisiana State University, New Orleans, USA.

AB: The limited literature on hypersomnolence suggests that it is a poorly defined symptom associated with a spectrum of disorders from monosymptomatic hypersomnolence to the Kleine-Levin syndrome. These disorders often herald an organic central nervous system syndrome. Recent evidence suggests a frequent association between these disorders and hypothalamic dysfunction, which itself may be

caused by a variety of factors. This case study of a patient with persistent hypersomnolence, hypothalamic dysfunction (in the form of precocious puberty), pica, and chronic lead intoxication strengthens the association between hypersomnolence and hypothalamic dysfunction and suggests a heretofore unreported cause of hypothalamic dysfunction in humans.

[Note: Clearly not KLS]

European Psychiatry 1996 11(2):104-105

Kleine-Levin syndrome misdiagnosed as schizophrenia

___ Bonnet F, Thibaut F, Levillain D, Petit M

Aviat Space Environ Med 1996 Jan;67(1):61-2

The sleeping aviator--aeromedical disposition of Kleine-Levin syndrome.

✓_ Wygnanski T, Kokia E, Barak P, Terlo L, Caine YG

Israel Air Force Aeromedical Center, Tel-Aviv.

AB: We present the case of an aviator with Kleine Levin syndrome (KLS). History, physical examination, and special studies presented confirm the diagnosis of this syndrome. Our patient presented as an atypical case of KLS with respect to the presenting symptoms and to frequency of the episodes (6 years apart). He exhibited only intense somnolence, easy arousability, photophobia, hyperacusis, and a voracious appetite. Following a complete medical work up we recommended that an applicant with such a classical case of KLS be disqualified as a crewmember; however, in cases such as that presented above a limited waiver may be considered. The aeromedical significance of this case is to reinforce the importance of screening candidates and seeking precise diagnosis of a past illness or hospitalization.

Sleep 1996 Jan;19(1):13-7

Kleine-Levin syndrome in a boy with Prader-Willi syndrome.

✓_ Gau SF, Soong WT, Liu HM, Hou JW, Tsai WY, Chiu YN, Yeh YC, Wang PJ, Wang TR

Department of Psychiatry, National Taiwan University Hospital, Taipei, R.O.C.

AB: A 9 1/2-year-old Taiwanese boy with Prader-Willi syndrome had the following characteristics: difficulties with sucking, feeding and hypotonia during infancy, a dysmorphic face (triangular mouth, high arched palate, almond-shaped eyes and large head circumference with a relatively narrow bifrontal diameter), borderline intelligence, hypogonadism, hyperphagia, skin picking and truncal obesity. The boy experienced two hypersomnia episodes, at age 8 and 9 years, with both episodes lasting for 10 days. During the two episodes, he was found to have an exacerbated case of hyperphagia, pica, poor emotional control, stereotyped speech and agitated behavior upon awakening. After each episode, the boy had complete remission. Our findings show that the two episodes are compatible with Kleine-Levin syndrome. The relationship between the two syndromes, the Prader-Willi syndrome and the Kleine-Levin syndrome, deserves further study.

(1995)

Neurophysiol Clin 1995;25(6):327-8

[Disorders of awakening]

LA: FRENCH

__ Billiard M, Besset A, Carlander B

Type: Editorial

Sleep 1995 Apr;18(3):206-8

Phase advancement of slow-wave sleep in a patient with periodic hypersomnia.

✓_ Romero O, Sagales T, Gimeno V, Cordoba J

Centre d'Estudis del Son, Hospital General Universitari Vall d'Hebron, Barcelona, Spain.

AB: Twenty-four-hour polysomnographic recordings were obtained during asymptomatic (ASP) and symptomatic (SP) periods in a patient with periodic hypersomnia. During both periods, an abnormal sleep architecture was detected, with a greater sleep fragmentation during the SP. An unexpected finding was a shortening of the latency of slow-wave sleep, with a difference of more than 5 hours between the ASP and the SP. The greater fragmentation of rapid eye movement sleep during the SP may suggest a disorganization of the ultradian rhythm of rapid eye movement sleep.

Sleep Research 1995 24:231 (abstract)

Kleine-Levin Syndrome: Psychiatric, Polysomnographic, and Actigraphic Findings

✓_ Feldman R, Dahl R, Guterson J

(1994)

TI: Kleine-Levin syndrome: a cause of diagnostic confusion.

✓AU: Pike-M; Stores-G

SO: Arch-Dis-Child. 1994 Oct; 71(4): 355-7

LA: ENGLISH

AB: The case is described of a boy with the Kleine-Levin syndrome in whom prominent behavioural disturbances and the initial absence of a clear cyclical pattern obscured the diagnosis. Treatment with lithium was effective.

AN: 95070304

TI: Kleine-Levin syndrome: a case report.

✓AU: Baral-A; Shah-N

SO: J-Indian-Med-Assoc. 1994 aug; 92(8): 273-4, 266

LA: ENGLISH

AN: 95052735

TI: Kleine-Levin syndrome [letter]

✓AU: Gordon-N; McKinlay-I

SO: Dev-Med-Child-Neurol. 1994 Jun; 36(6): 560-1

LA: ENGLISH

AN: 94273952

TI: 'A case of fever, attended with inordinate appetite'. A report delivered and recorded in 1815.

_AU: Doyle L

North West Lung Centre, Wythenshawe Hospital, Manchester.

SO: J R Coll Physicians Lond 1994 Mar-Apr;28(2):172-3

Type: Historical article

(1993)

TI: Clinical features of Kleine-Levin syndrome with localized encephalitis.

✓AU: Fenzi-F; Simonati-A; Crosato-F; Ghersini-L; Rizzuto-N

SO: Neuropediatrics. 1993 Oct; 24(5): 292-5

LA: ENGLISH

AB: We report the clinico-pathological findings regarding a 9 year-old girl with some clinical features of Kleine-Levin syndrome who died suddenly as a result of pulmonary embolism in the course of femoro-iliac thrombophlebitis. Neuropathological examination provided evidence of perivascular inflammatory infiltrates and microglial proliferation of nodular type located in the diencephalon and midbrain. These findings suggest that a localized encephalitis may be the underlying condition in Kleine-Levin syndrome.

AN: 94142834

TI: Electrophysiological and immunogenetic findings in recurrent monosymptomatic-type hypersomnia: a study of two unrelated Italian cases.

✓AU: Manni-R; Martinetti-M; Ratti-MT; Tartara-A

SO: Acta-Neurol-Scand. 1993 Oct; 88(4): 293-5

LA: ENGLISH

AB: MSLT and immunogenetic findings in two unrelated Italian subjects with recurrent monosymptomatic hypersomnia are reported. In both patients MSLT documented a markedly increased daytime sleep propensity during the attacks without augmented REM sleep pressure. Both patients share the same HLA haplotype (HLA-DR1, DQ1) which has been found in Kleine-Levin syndrome. This makes these subtypes of recurrent hypersomnia indistinguishable one from the other, under the immunogenetic profile, but permits differentiation from narcolepsy which is HLA-DR2, DQ1 closely linked.

AN: 94078731

TI: A variant of the Kleine-Levin syndrome precipitated by both Epstein-Barr and varicella-zoster virus infections.

✓AU: Salter-MS; White-PD

SO: Biol-Psychiatry. 1993 Mar 1; 33(5): 388-90

LA: ENGLISH

AN: 93229631

TI: Thalamo-frontal psychosis [see comments]

✓AU: McGilchrist-I; Goldstein-LH; Jadresic-D; Fenwick-P

SO: Br-J-Psychiatry. 1993 Jul; 163: 113-5

LA: ENGLISH

AB: A 43-year-old man presented with an 18-month history of acute-onset cyclical behavioural change affecting mood, appetite, sleep, and energy levels. This had followed an initial episode of transient drowsiness which lasted 24 hours. On examination, there was some evidence of visual memory and frontal lobe deficits. A brain CT scan showed bilateral thalamic infarcts and a brain SPECT scan showed bilateral hypoperfusion of the frontal lobes. To our knowledge, this is the first reported case of thalamic infarction associated with acute-onset cyclical affective psychosis with clinical and neurophysiological features of frontal lobe syndrome. The case also highlights the possible role of thalamo-frontal circuits in the pathogenesis of the Kleine-Levin syndrome.

AN: 93358137; TYPE: SPECT

TI: Abnormal fluctuations of acetylcholine and serotonin.

✓AU: Brown-DW

SO: Med-Hypotheses. 1993 May; 40(5): 309-10

LA: ENGLISH

AB: Abnormal fluctuations of acetylcholine and serotonin might cause, or be correlated with, a variety of symptoms. This communication cites evidence which suggests that fluctuations of serotonin might have a causative role in migraine, mania, and depression and that fluctuations of acetylcholine might have a causative role in epilepsy, catalepsy, and Kleine-Levin attacks.

AN: 93354159

TI: [Review of Kleine-Levin syndrome: toward an integrated approach]

✓AU: Lemire-I

SO: Can-J-Psychiatry. 1993 May; 38(4): 277-84

LA: FRENCH

AB: Kleine-Levin syndrome is characterized by a periodic hypersomnia crisis as well as dietary and variable significant psychic symptoms. Its rarity makes its definition and study complex. Many etiologies have been considered, especially regarding a hypothalamic dysfunction. Physiological, radiologic, anatomical and polysomnographic studies have not shown clearly conclusive results, but the syndrome appears to be related to affective disorders. This hypothesis is supported by the findings of a positive therapeutic response to lithium. However, we are interested in the related psychological factors, whether they are primary or secondary. A clinical case illustrates these factors, and a basic neuropsychiatric intervention concept is proposed.

AN: 93299734

(1992)

TI: [Kleine-Levin syndrome]

_AU: Hansen-D; Lonborg-Moller-L

SO: Ugeskr-Laeger (Denmark). 1992 Oct 19; 154(43): 2975-8

LA: DANISH

AB: The Kleine-Levin syndrome is a rare and probably underdiagnosed syndrome. It is characterized by periodic attacks of the triad: hypersomnia, vegetative disturbances such as hyperphagia and hypersexuality, psychopathological changes in the level of consciousness and control of emotions. Boys and young men in the age group 10-20 years are most commonly affected. Spontaneous remission with a tendency to remission is observed and the disease "burns out" after a prolonged period of years. The etiology and pathogenesis are unknown. Theories have been propounded suggesting dysfunction of the hypothalamus. No pathognomonic findings have been observed in the early phase of sleep during the daylight hours. Central stimulating drugs have been reported to have some effect on the hypersomnia. The diagnosis is based on the clinical picture. Frequently, a long period can elapse before the diagnosis is established and some cases are never diagnosed. The literature is reviewed and is illustrated by two case reports.

AN: 93097410

TI: **Clinical use of moclobemide in Kleine-Levin syndrome** [letter]

✓AU: Chaudhry-HR

SO: Br-J-Psychiatry. 1992 Nov; 161: 720

LA: ENGLISH

AN: 93045260

TI: **The more unusual sleep disturbances of childhood.**

✓AU: Gordon-N

SO: Brain & Development (Tokyo). 1992 May; 14(3): 182-4

LA: ENGLISH

AB: The sleep patterns of children often cause anxiety to their parents. Some disturbances are unusual, and therefore may cause diagnostic difficulties. Sleep walking and night terrors can be confused with epileptic seizures. The sudden sleep of narcolepsy can lead to false accusations, when in fact the episodes are beyond the child's control. The associated phenomena of cataplexy, hypnogogic hallucinations and sleep paralysis can be particularly alarming, especially if they occur in the absence of narcolepsy. The overlap between narcolepsy and the Kleine-Levin syndrome is confirmed. Although of a different nature the sleep apnoea syndrome is equally important from the point of view of diagnosis and treatment.

AN: 92384388

TI: **Recurrent hypersomnia in two adolescent males with Asperger's syndrome.**

✓AU: Berthier-ML; Santamaria-J; Encabo-H; Tolosa-ES

SO: J-Am-Acad-Child-Adolesc-Psychiatry. 1992 Jul; 31(4): 735-8

LA: ENGLISH

AB: Two individuals with Asperger's syndrome, a rare pervasive developmental disorder, developed recurrent episodes of hypersomnia and abnormal behavior (Kleine-Levin syndrome) during adolescence. The possible etiological role of developmental

structural brain anomalies and the differential diagnosis of recurrent hypersomnia and abnormal behavior in patients with pervasive developmental disorders are discussed.
AN: 92355480

TI: Triazolam-induced nocturnal bingeing with amnesia.

✓AU: Menkes-DB

SO: Aust-N-Z-J-Psychiatry. 1992 Jun; 26(2): 320-1

LA: ENGLISH

AB: A combination of behavioural and cognitive adverse effects is illustrated in this case report of a recurrent triazolam-induced eating disorder. The co-occurrence of bingeing, irritability and anterograde amnesia is suggestive of a drug-induced Kleine-Levin Syndrome.

AN: 92352437

TI: Kleine-Levin syndrome in an 82 year old man.

_AU: Badino-R; Caja-A; Del-Conte-I; Guida-C; Ivaldi-M

SO: Ital-J-Neurol-Sci. 1992 May; 13(4): 355-6

LA: ENGLISH

AN: 92290778

TI: Kleine-Levin syndrome: a case report.

✓AU: Russell-J; Grunstein-R

SO: Aust-N-Z-J-Psychiatry. 1992 Mar; 26(1): 119-23

LA: ENGLISH

AB: Kleine-Levin Syndrome is an unusual sleep disorder occurring predominantly, but not exclusively, in late adolescent males. A case is described which illustrates some of the difficulties in diagnosis and management.

AN: 92255700

(1991)

TI: Neuroendocrine evaluation in Kleine-Levin syndrome: evidence of reduced dopaminergic tone during periods of hypersomnolence.

✓AU: Chesson-AL Jr; Levine-SN; Kong-LS; Lee-SC

SO: Sleep. 1991 Jun; 14(3): 226-32

LA: ENGLISH

AB: A patient with Kleine-Levin syndrome had polysomnography and neuroendocrinological assays performed during asymptomatic (ASMP) and symptomatic (SMP) 24-hr periods. During the SMP, sleep data revealed poor nocturnal sleep efficiency, increased sleep fragmentation and reduced stages 3, 4 and rapid eye movement (REM). No sleep onset REM episodes were seen. Sleep staging in the ASMP was normal. Blood samples were obtained every 20 min and assayed for thyroid-stimulating hormone (TSH), cortisol (CORT), prolactin (PRL) and growth hormone (GH). Patterns of secretion, 24-hr mean and total integrated concentrations, and mean sleep period time values during the ASMP and SMP were compared. The mean 24-hr level of

TSH was increased and GH decreased in the SMP. Comparing sleep period time in the SMP to the ASMP, values for TSH and PRL were increased and GH and CORT were reduced in the SMP. These hormone changes support the hypothesis that reduced hypothalamic dopaminergic tone is present in the SMP compared to the ASMP in Kleine-Levin patients.

AN: 91376494

TI: **[The Klein-Levin syndrome (a case)]**

_AU: Novitskaia-AK

SO: Zh-Nevropatol-Psikhiatr-Im-S-S-Korsakova. 1991; 91(3): 84-6

LA: RUSSIAN

AB: The author provides a detailed description and a clinical analysis of a case of Kleine-Levin syndrome. Reviews the psychopathological characteristics of the patient with Kleine-Levin syndrome. Hypersomnic attacks occurred in an epileptoid personality at an age of 14 years, each attack was coupled with affective derealization symptomatology. The epileptic nature of the disease is not supported by the EEG data. It is suggested that the psychopathological symptomatology of the hypersomnic attack is consequent on the derangement of mediator regulation of hypothalamic functions.

AN: 91263532

TI: **[Kleine-Levin syndrome. Report of a case]**

_AU: Da-Silveira-Neto-O; Da-Silveira-OA

SO: Arq-Neuropsiquiatr. 1991 Sep; 49(3): 330-2

LA: PORTUGUESE

AB: A case of a 14 year-old boy suffering from Kleine-Levin syndrome is described. The diagnosis was made by symptoms which suggested this disorder: megaphagia, hypersomnia and hypersexuality. A brief review is made emphasizing the pathophysiology, diagnosis and treatment of this syndrome which is frequently misdiagnosed.

AN: 92222392

TI: **[Kleine-Levin syndrome]**

_AU: Wurthmann-C; Klieser-E

SO: Fortschr-Neurol-Psychiatr. 1991 Sep; 59(9): 371-5

LA: GERMAN

AB: The Kleine-Levin syndrome is generally considered to be a benign functional disorder of hypothalamic structures. Its onset is usually in adolescence. The most characteristic symptoms are periodic hypersomnia, excessive eating, hypersexuality, irritability and apathy. Associated features are depressive and schizophrenic symptoms. A biological relationship between the Kleine-Levin syndrome and endogenous psychoses is discussed.

AN: 92064282

TI: **[Kleine-Levin syndrome]** [note same abstract as above, but different citation]

_AU: Wurthmann-C; Klieser-E

SO: Fortschr-Neurol-Psychiatr. 1991 May; 59(5): 190-4

LA: GERMAN

AB: The Kleine-Levin syndrome is generally considered to be a benign functional disorder of hypothalamic structures. Its onset is usually in adolescence. The most characteristic symptoms are periodic hypersomnia, excessive eating, hypersexuality, irritability and apathy. Associated features are depressive and schizophrenic symptoms. A biological relationship between the Klein-Levin syndrome and endogenous psychoses is discussed.

AN: 91331526

TI: **[Sleep polygraphic studies as an objective method for assessing the therapeutic result in a case of periodic hypersomnia (Kleine-Levin syndrome)]**

_AU: Ugoljew-A; Kurella-B; Nickel-B

SO: Nervenarzt. 1991 May; 62(5): 292-7

LA: GERMAN

AN: 91326163

(1990)

TI: **Disturbed hypothalamic-pituitary axis in idiopathic recurring hypersomnia syndrome.**

✓AU: Fernandez-JM; Lara-I; Gila-L; O'Neill-of-Tyrone-A; Tovar-J; Gimeno-A

SO: Acta-Neurol-Scand. 1990 Dec; 82(6): 361-3

LA: ENGLISH

AB: Disturbed function of the hypothalamic-pituitary axis at adrenal and thyroid levels was found during an episode of hypersomnia in the idiopathic recurring hypersomnia syndrome. These endocrinological abnormalities, abolished ACTH and cortisol responses to insulin-induced hypoglycemia and absent TSH response to TRH, normalised thereafter in the symptom-free interval. These data support the hypothesis that Kleine-Levin syndrome is related to an intermittent hypothalamic dysfunction.

AN: 91150436

TI: **A disorder unique to adolescence? The Kleine-Levin syndrome.**

✓AU: Cawthron-P

SO: J-Adolesc. 1990 Dec; 13(4): 401-6

LA: ENGLISH

AB: The Kleine-Levin syndrome is a rare disorder. In the pure form it is a diagnosis that can only be made in adolescent males. The patient described fits this category and can be added to similar case histories reported in the world literature. Evidence is presented to suggest a link between the Kleine-Levin syndrome and the cyclical affective disorders.

AN: 91161746

TI: **[Kleine-Levin syndrome--an unusual behavioral disorder in young men]**

_AU: Holmberg-BH; Kempe-A; Joensen-H; Johansson-F

SO: Lakartidningen. 1990 Oct 24; 87(43): 3514, 3519

LA: SWEDISH
AN: 91041176

Wiad Lek 1990 Sep 1-15;43(17-18):911-3

[A case of periodic hypersomnia in a 14-year-old boy].

LA: POLISH

_AU: Jurynczyk J Oddzialu Neurologicznego Rejonowego Szpitala w Belchatowie.
AB: A 14-years-old boy is reported with paroxysmal sleep disturbances associated with bulimia. Despite absence of EEG verification it seems that it was Kleine-Levine syndrome developing probably after cranial trauma.

TI: HLA-DR antigens in Kleine-Levin syndrome [letter]

✓AU: Visscher-F; van-der-Horst-AR; Smit-LM

SO: Ann-Neurol. 1990 Aug; 28(2): 195

LA: ENGLISH

AN: 91024080

TI: Interepisodic morbidity in Kleine-Levin syndrome.

✓AU: Sagar-RS; Khandelwal-SK; Gupta-S

SO: Br-J-Psychiatry. 1990 Jul; 157: 139-41

LA: ENGLISH

AB: Three patients with Kleine-Levin syndrome had evidence of interepisodic organic brain dysfunction. This is counter to earlier views that the disorder is benign.

AN: 90374060

TI: Autonomic events in Kleine-Levin syndrome [letter]

✓AU: Hegarty-A; Merriam-AE

SO: Am-J-Psychiatry. 1990 Jul; 147(7): 951-2

LA: ENGLISH

AN: 90289848

(1989)

TI: [The Kleine-Levin syndrome]

_AU: Visscher-F; Smit-LM; Smith-F; Boer-F; Njiokiktjien-C

SO: Tijdschr-Kindergeneesk. 1989 Dec; 57(6): 218-21

LA: DUTCH; NON-ENGLISH

AB: Two boys, aged 12 and 13 years, showed relapsing periods of somnolence and excessive eating, starting after a viral illness. One of them also showed periodic disturbance of sexual impulse control. The symptomatic periods were followed by symptom-free intervals in a highly characteristic pattern. This gave the clue to the diagnosis Kleine-Levin syndrome. The cause of this syndrome is unknown, in some cases a relationship between infectious disease or traumatic brain damage has been postulated. A dysfunction of the hypothalamus and associated structures is suspected. The syndrome has a rather favourable prognosis. The symptoms can be relieved by amphetamines, methylphenidate and probably also by lithium carbonate.

AN: 90141627

TI: Kleine-Levin syndrome 15 years later.

_AU: Chiu-HF; Li-SW; Lee-S

SO: Aust-N-Z-J-Psychiatry. 1989 Sep; 23(3): 425-7

LA: ENGLISH

AB: A Chinese man with Kleine-Levin syndrome showed evidence of hypothalamic disturbance and damage to the limbic system. He has been free from symptoms of hypersomnia and hyperphagia for 15 years, but now suffers from ejaculatory impotence which is discussed in the light of aetiological theories of this puzzling disorder.

AN: 90026093

TI: [Kleine-Levin syndrome. The provocation of manic symptoms by an antidepressant and a therapeutic trial of carbamazepine]

_AU: Wurthmann-C; Hartung-HP; Dengler-W; Gerhardt-P

SO: Dtsch-Med-Wochenschr. 1989 Oct 6; 114(40): 1528-31

LA: GERMAN;

AB: A 17-year-old adolescent had a recurrent episode of somnolence and morbid hunger (Kleine-Levin syndrome) three years after a first attack, from which he had spontaneously recovered. He was treated with 50 mg daily of clomipramine for the somnolence accompanied by disturbance of attention and memory. Under this treatment he developed thymoleptic symptoms with polyphagia, logorrhea and hyperactivity. Placed on a trial dose of at first 600 mg, then 400 mg carbamazepine daily the abnormal findings disappeared within a few days, and there has been no recurrence after some months. It is postulated, based on the observations of this case, that the Kleine-Levin syndrome, presumably a functional hypothalamic disorder, is closely related to the endogenous psychoses.

AN: 90004953

TI: Periodic hypersomnia, congenital ectodermal disorders and multiple exostosis.

_AU: Reimao-R; Diament-A

SO: Arq-Neuropsiquiatr. 1989 Mar; 47(1): 76-9

LA: ENGLISH

AB: A case of periodic hypersomnia in an 11-year-old female with the unique features of mental deficiency, incontinentia pigmenti, acanthosis nigricans and hereditary multiple exostosis (diaphysial aclasis) is reported. The clinical, polysomnographic and Multiple Sleep Latency test features of this case with a follow up of seven years are consistent with a diagnosis of periodic (intermittent) excessive somnolence. The unique presentation, however, does differ from Kleine-Levin syndrome and suggests a relationship between the predominantly ectodermal, congenital disorders and the sleep-wake pattern dysfunction.

AN: 89350568

TI: A female case with the Kleine-Levin syndrome and its physiopathologic aspects.

✓AU: Fukunishi-I; Hosokawa-K

SO: Jpn-J-Psychiatry-Neurol. 1989 Mar; 43(1): 45-9

LA: ENGLISH

AB: A female case with the Kleine-Levin syndrome (KLS), which first occurred at the age of 19, was discussed in relation to the following four characteristics: 1) a female case, 2) a loss of memory and the appearance of slow waves in the pathosis, 3) the abnormal pattern of growth hormone (GH) secretion during sleep in the pathosis, and 4) a prolonged latency between the peaks of III and V in the auditory brainstem response (ABR) in both the pathosis and nonpathosis. These characteristics may suggest that there is a slight disturbance of consciousness in the pathosis, and that there is a functional disturbance in the hypothalamus.

AN: 89294239

(1988)

TI: Kleine-Levin syndrome: report of two cases with onset of symptoms precipitated by head trauma.

✓AU: Will-RG; Young-JP; Thomas-DJ

SO: Br-J-Psychiatry. 1988 Mar; 152: 410-2

LA: ENGLISH

AB: Two cases of Kleine-Levin syndrome are described in which the onset of symptoms was temporally related to head injury. The possible aetiological role of trauma and an apparent response to treatment with lithium carbonate are discussed.

AN: 89001717

TI: Kleine-Levin syndrome ethiopathogenesis and treatment.

✓AU: Smolik-P; Roth-B

SO: Acta Universitatis Carolinae-Medica 1988; 128: 5-94 monograph Praha: Univerzita Karlova

LA: ENGLISH

AB: The complex of the symptoms of psychic disorders and of the disorders of sleep, appetite, and food intake often forms the basis of the clinical picture of a mental disease. However, it is only rarely conceived in a complex manner as a set of physiologically interdependent functions. A remarkable proof of the interdependence of these functions is their complex disorder, the Kleine-Levin syndrome. The first descriptions of the symptoms of the Kleine-Levin syndrome can be found in the studies of several authors published as early as at the turn of the century. In 1942, the syndrome was designated by Critchley and Hoffmann after Willi Kleine and Max Levin, who defined it precisely in 1925 and 1929. The syndrome of periodic hypersomnia, megaphagia, and psychic disorders, originally described only in young males, was later found in females as well; the original very strict criteria were gradually broadened and complemented to some extent. At present, the most commonly accepted criterion for the diagnosis of the Kleine-Levin syndrome is the existence of the combined sleep disorder (hypersomnia or insomnia lasting from days to weeks), food intake disorders (megaphagia or anorexia), and various psychic abnormalities accompanying or following the attacks of the affection. We term the syndrome typical if the sleep disorder

appears in the form of hypersomnia, food disorder in the form of megaphagia, and if psychic abnormalities are clearly expressed. On the other hand, we term the syndrome atypical if one of the main symptoms is opposite. The incomplete syndrome consists of only two main symptoms. The attacks of the affection set on mostly suddenly, lasting from several days to several weeks, ending suddenly again. The interparoxysmal periods last from several days to several months, sometimes even to several years. The etiopathogenesis of the affection is still unknown. A number of reports indicate a disorder of the diencephalon, perhaps only of the hypothalamus. The pathological-anatomical findings following the death of persons suffering from the disorders of sleep and food intake and from psychic abnormalities mostly reveal lesions in the region of the third brain ventricle. The development of the typical syndrome is benign, however, and morphological studies are not available. The typical Kleine-Levin syndrome can hardly escape the attention of clinicians owing to the richness and clarity of symptoms. The atypical or discretely expressed forms, however, often remain unrecognized even after a detailed medical examination and may lead to diagnostic uncertainty.(ABSTRACT TRUNCATED AT 400 WORDS)
AN: 90144323

TI: Les hypersomnies recurrentes

_AU: Billiard, M., Cadilhac, J.

SO: Rev. Neurol (Paris) 144:249-258 1988

(1987)

TI: Lithium carbonate: effects on sleep patterns of normal and depressed subjects and its use in sleep-wake pathology.

_AU: Billiard-M

SO: Pharmacopsychiatry. 1987 Sep; 20(5): 195-6

LA: ENGLISH

AB: The effects of lithium carbonate on sleep patterns have been investigated both acutely in normal and depressed subjects and chronically in depressed subjects. In normal subjects receiving lithium for two weeks total sleep time did not vary, REM sleep decreased and REM sleep latency increased. In depressed subjects, either on short term therapy or on long term therapy stages 3 and 4 increased, REM sleep decreased, REM latency increased and REM activity/time spent asleep (an index of REM intensity per minute of sleep) decreased. Plasma lithium levels were negatively correlated with REM sleep percentage and positively correlated with REM sleep latency. Besides, it has been shown in one paper that short term therapy with lithium caused small but significant delays in the sleep-wake circadian rhythm. These effects are of interest in view of polygraphic sleep abnormalities found in affective disorders and possible circadian disturbances accounting for these abnormalities. Indeed lithium might act in correcting special sleep abnormalities and/or circadian disturbances. In addition to its predominant use for the prophylaxis of recurrent mania and depression, lithium carbonate has been proposed and tried in the prophylactic treatment of abnormally prolonged sleep episodes featuring the Kleine-Levin syndrome.

AN: 88041467

TI: Kleine-Levin syndrome after multiple cerebral infarctions.

✓AU: Drake-ME Jr

SO: Psychosomatics. 1987 Jun; 28(6): 329-30

LA: ENGLISH

AN: 88125731

TI: Kleine-Levin syndrome: unrecognized diagnosis in adolescent psychiatry.

✓AU: Gillberg-C

SO: J-Am-Acad-Child-Adolesc-Psychiatry. 1987 Sep; 26(5): 793-4

LA: ENGLISH

AN: 88032790

TI: Episodic hormone secretion during sleep in Kleine-Levin syndrome: evidence for hypothalamic dysfunction.

✓AU: Gadoth-N; Dickerman-Z; Bechar-M; Laron-Z; Lavie-P

SO: Brain-Dev. 1987; 9(3): 309-15

LA: ENGLISH

AB: "Acute" hypothalamic-pituitary function tests including insulin tolerance test, LRH, ACTH and TRH stimulation tests and nocturnal secretory pattern of human growth hormone, 11-OHCS, prolactin, FSH, LH and TSH were studied in a 23-year-old male with Kleine-Levin syndrome during the course of a typical hypersomnic attack. The "acute" tests revealed paradoxical growth-hormone response to TRH stimulation, borderline high basal plasma prolactin levels with normal response to TRH. The hormonal secretory pattern during sleep revealed abnormalities in LH, 11-OHCS and prolactin secretion. These together with the results of the "acute" tests are indicative of an abnormality in the hypothalamic regulation of various pituitary hormones. This observation may indeed be the first laboratory demonstration confirming a long-standing hypothesis that Kleine-Levin syndrome is related to hypothalamic dysfunction.

AN: 88021647

TI: Kleine-Levin syndrome: a review.

✓AU: Khan-MH; Johnson-FC

SO: South Dakota Journal Med. 1987 Jul; 40(7): 7-10

LA: ENGLISH

AN: 87319628

TI: A case of multiple sclerosis with an onset mimicking the Kleine-Levin syndrome.

_AU: Testa-S; Opportuno-A; Gallo-P; Tavolato-B

SO: Ital-J-Neurol-Sci. 1987 Apr; 8(2): 151-5

LA: ENGLISH

AB: A 20 years old patient is described, who presented two episodes of day-time hypersomnia, orthostatic hypotension and psychotic behavior. Compulsive masturbation and abnormalities of the eating habits were also present. Both episodes cleared up spontaneously in about one week. From the clinical point of view of Kleine-Levin

syndrome, was suspected. However a CSF examination showed IgG oligoclonal bands and an increased IgG index. A NMR showed multiple area of increased signal intensity, suggestive of a demyelinating disease. On these elements a final diagnosis of MS was made, in spite of a normal neurological examination. This case was compared to other cases of MS in which the disease begins with acute remitting psychiatric symptoms. However cases of MS with complex episodes as observed in our patient were not found in the Literature.

AN: 87249712

(1986)

TI: **[A case of Kleine-Levin syndrome stabilized for over 1 year with carbamazepine (letter)]**

✓AU: Savet-JF; Robert-H; Angeli-C

SO: Presse-Med. 1986 Jul 5-12; 15(27): 1281

LA: FRENCH

AN: 87016781

TI: **An unusual case of Kleine-Levin syndrome associated with sleep terrors.**

✓AU: Striano-S; Bilo-L; Meo-R

SO: Electroencephalogr-Clin-Neurophysiol. 1986 Dec; 64(6): 517-20

LA: ENGLISH

AB: The authors describe a case of Kleine-Levin syndrome (KLS) which in the hypersomniac period presents, together with the classical symptoms, frequent arousals accompanied by sleep terrors. Polygraphic study shows the absence of any nyctohemeral cycle and very frequent parasomnias occurring during arousals from NREM sleep. On the basis of data from the literature, the authors suggest that a disturbance of maintenance of sleep might not be exceptional in KLS, even if none of the previous reports describes such frequent parasomnias.

AN: 87053505

TI: **Kleine-Levin syndrome following acute viral encephalitis.**

✓AU: Merriam-AE

SO: Biol-Psychiatry. 1986 Nov; 21(13): 1301-4

LA: ENGLISH

AN: 87000833

TI: **[Diagnosis, etiopathogenesis and treatment of the Kleine-Levin syndrome]**

_AU: Smolik-P; Roth-B

SO: Cesk-Psychiatr. 1986 Apr; 82(2): 127-30

LA: CZECH

AN: 86272195

TI: **Kleine-Levin syndrome: a case report.**

✓AU: Ferguson-BG

SO: J-Child-Psychol-Psychiatry. 1986 Mar; 27(2): 275-8

LA: ENGLISH

AB: The case of a 10 yr old boy suffering from Kleine-Levin syndrome is described. The diagnosis was originally obscured by symptoms which suggested a primary depressive disorder. Unusual features included bizarre crying behaviour and unfounded allegations of sexual assault. Although the syndrome is rare, the need to be aware of the diagnosis is stressed, particularly in pre-pubertal children suffering from significant episodic depressive symptoms.

AN: 86168653

TI: [**A case of hypersomnia resembling Kleine-Levin syndrome**]

_AU: Domzal-Stryga-A; Emeryk-Szajewska-B; Kowalski-J

SO: Neurol-Neurochir-Pol. 1986 Mar-Apr; 20(2): 158-60

LA: POLISH

AB: A case report is presented of episodic hypersomnia with bouts of several days duration. The authors discuss various clinical syndromes with sleep disorders with particular consideration of the Kleine-Levin syndrome.

AN: 87039608

TI: [**Obesity and hypersomnia**]

_AU: Vantov-M; Damianova-G; Vurbanova-M

SO: Vutr-Boles. 1986; 25(1): 82-5

LA: BULGARIAN

AB: Seventy one patients with hypersomniac paroxysms--Pickwickian and Kleine-Levin syndromes, were studied, the incidence of combination of hypersomnia and obesity--followed up. They are supposed to have common pathogenetic mechanisms, giving weight to lesions of hypothalamo-reticulo-limbic structures in viral infections, craniocerebral traumas, intoxications and tumours. The pathological processes advanced in the reticulo-hypothalamo-hypophyseal region lead to hormonal regulatory disorders and disturbances in adaptation and compensatory mechanisms of metabolism, of lipid metabolism including, of sleep and vigilance. A schematic central explanation of the inter-relations of obesity and hypersomnias is presented.

AN: 86237353

(1985)

TI: **Sleep apnea and the Kleine-Levin syndrome.**

✓AU: Cuetter-AC

SO: Mil-Med. 1985 May; 150(5): 286-8

LA: ENGLISH

AN: 85214493

TI: **Neuroendocrine rhythms in a patient with the Kleine-Levin syndrome.**

✓AU: Thompson-C; Obrecht-R; Franey-C; Arendt-J; Checkley-SA

SO: Br-J-Psychiatry. 1985 Oct; 147: 440-3

LA: ENGLISH

AN: 86078338

TI: [The Kleine-Levin syndrome. Periodic hypersomnia and hyperphagia with abnormal behavior]

_AU: Jensen-J

SO: Ugeskr-Laeger. 1985 Feb 18; 147(8): 709-10

LA: DANISH

AN: 85169418

TI: Kleine-Levin Syndrome

_AU: Iijima, S.

SO: Jpn J. Clin Psychiatry 14: 591-594, 1985

LA: Japanese

TI: Kleine-Levin syndrome: normal CSF monoamines and response to lithium therapy [letter]

✓AU: Hart-EJ

SO: Neurology. 1985 Sep; 35(9): 1395-6

LA: ENGLISH

AN: 85268587

(1984)

TI: Increased cerebrospinal fluid 5-hydroxytryptamine and 5-hydroxyindoleacetic acid in Kleine-Levin syndrome.

✓AU: Koerber-RK; Torkelson-R; Haven-G; Donaldson-J; Cohen-SM; Case-M

SO: Neurology. 1984 Dec; 34(12): 1597-600

LA: ENGLISH

AB: A 17-year-old man with the Kleine-Levin syndrome died unexpectedly of cardiopulmonary arrest during a period of autonomic instability that followed an episode of megaphagia. At autopsy, the only pertinent finding was mild depigmentation of the locus ceruleus and substantia nigra. Premortem CSF levels of 5-hydroxytryptamine (5-HT) and 5-hydroxyindoleacetic acid (5-HIAA) levels were elevated. These findings indicate that many symptoms of the Kleine-Levin syndrome are a result of a neurotransmitter imbalance in the serotonergic pathway of the brainstem.

AN: 85061943

TI: [Fibrin fibrinogen degradation products in the cerebrospinal fluid of neurological patients]

_AU: Nowak-S; Ziolo-H; Błaszczyk-B

SO: Neurol-Neurochir-Pol. 1984 Nov-Dec; 18(6): 541-5

LA: POLISH

AB: The authors determined FDP levels in the cerebrospinal fluid by the method of Merskey in 214 neurological patients and found raised levels in 58.6% of cases (from 0.1 to 8.0 ug/ml, with normal value range 0-0.5 ug/ml). In the control group the FDP levels in the CSF were normal. No correlation was noted between the FDP levels in the CSF and in blood. Raised CSF FDP level was observed in exacerbations of multiple

sclerosis, strokes especially of embolic origin, syringomyelia, bulbar form of amyotrophic lateral sclerosis, epilepsy, migraine, lumbar disc lesions, polyneuropathy, parkinsonism, brain atrophy, after craniocerebral trauma, in Kleine-Levin syndrome. The authors are studying now the course of FDP changes in the CSF in various cases in the aspect of clinical-laboratory correlations.

AN: 85268302

TI: Recognizing and managing the adolescent with Kleine-Levin syndrome.

✓AU: Waller-DA; Jarriel-S; Erman-M; Emslie-G

SO: J-Adolesc-Health-Care. 1984 Apr; 5(2): 139-41

LA: ENGLISH

AB: This report calls attention to the Kleine-Levin Syndrome, a sleep disorder which predominantly occurs in adolescent males. It can easily be mistaken for other psychiatric or neurologic illness. Differential diagnosis and treatment are discussed.

AN: 84161563

TI: Multiple Sleep Latency Test findings in Kleine-Levin syndrome.

✓AU: Reynolds-CF; Kupfer-DJ; Christiansen-CL; Auchenbach-RC; Brenner-RP;

Sewitch-DE; Taska-LS; Coble-PA

SO: J-Nerv-Ment-Dis. 1984 Jan; 172(1): 41-4

LA: ENGLISH

AB: Multiple Sleep Latency Test (MSLT) findings in a case of Kleine-Levin syndrome are reported for the first time. MSLT data indicate sleepiness as severe as in narcolepsy or obstructive sleep apnea and the occurrence of four sleep onset rapid eye movement (REM) periods, with a greater REM propensity at 2:00 p.m. and 4:00 p.m. than at 10:00 a.m. and 12:00 noon. The replication of such findings might suggest that Kleine-Levin syndrome could be considered a form of periodic REM sleep disinhibition. Therefore, the traditional hypothesis of diencephalic dysfunction may require modification to include the role of more caudal brain stem structures specifically activated during REM sleep.

AN: 84089616

TI: [Non-epileptic seizures]

_AU: Krause-KH

SO: Nervenarzt. 1984 Oct; 55(10): 507-16

LA: GERMAN

AB: Following definition of the non-epileptic seizures terminological problems are discussed and the symptomatology and classification of these seizures are described. Four main groups are differentiated: anoxic cerebral seizures, hypnic cerebral seizures, metabolic cerebral seizures and psychic seizures. Within the first group we have to distinguish between seizures caused by ischaemia, asphyxia and anoxia. The generalized ischaemic anoxic seizures--the syncopes--are differentiated into vegetative seizures (vagotonic, asympathicotonic, sympathicotonic) and into syncopes caused by cardiac disturbances. Disturbances of sleep rhythm are to be seen in narcolepsy, periodic hypersomnia, Kleine-Levin syndrome and Pickwickian syndrome. The group of

metabolic seizures includes hypoglycaemic seizures, seizures in pheochromocytoma, in Addison's disease and Conn's syndrome and the tetanic seizures.

AN: 85061762

(1983)

TI: **The treatment of Kleine-Levin syndrome with lithium.**

✓AU: Goldberg-MA

SO: Can-J-Psychiatry. 1983 Oct; 28(6): 491-3

LA: ENGLISH

AB: A case of Kleine-Levin Syndrome without megaphagia is described in an adolescent male who responded well to lithium treatment. Seventy-two hours of sleep monitoring during a typical attack revealed decreased stages three and four but no sleep-onset REM period. Recent reports, including the present one, strongly suggest that Lithium Carbonate may be the treatment of choice for the Kleine-Levin Syndrome.

AN: 84054961

(1982)

TI: **[The periodic hypersomnia and megaphagia syndrome in a young female, correlated with menstrual cycle]**

_AU: Papy-JJ; Conte-Devolx-B; Sormani-J; Porto-R; Guillaume-V

SO: Rev-Electroencephalogr-Neurophysiol-Clin. 1982 Apr; 12(1): 54-61

LA: FRENCH

AB: A 21-year-old female was first seen in 1974 for periodic hypersomnia. It had started at 13 years of age and was characterized by 4-6 days of drowsiness with megaphagia and some light psychic disorders. The attacks usually occurred at approximately 5-6 days after menstruation. Later, after an interval of oral contraception, they appeared during ovulation and increased in duration. During the periods of contraception, these attacks disappeared completely. At present (1980), the episodes are characterized by periodic hypersomnia, seen in a nocturnal polygraphic study; megaphagia has decreased, but the patient appears uncommunicative, sub-active and inexpressive when awaking in the course of the periods of somnolence. Several EEG recordings were made between 1974 and 1980, which showed few or no abnormalities during the attacks and which were normal with in the inter-ictal periods. Hormonal investigations failed to show any striking abnormality or correlation with continuous polygraphic studies. Similarity of this syndrome with the 'periodic hypersomnia and megaphagia in adolescent males' syndrome (Kleine-Levin) is discussed.

AN: 82249006

TI: **[Kleine-Levin syndrome]**

_AU: Plasse-M; Joannard-A; Maynard-R; Jouk-PS; Gilly-R; Beaudoin-A

SO: Pediatrie. 1982 Dec; 37(8): 601-5

LA: FRENCH

AN: 83220665

TI: Kleine-levin syndrome: a case report from India [letter]

✓AU: Shukla-GD; Bajpai-HS; Mishra-DN

SO: Br-J-Psychiatry. 1982 Jul; 141: 97-8

LA: ENGLISH

AN: 83001061

TI: A pathologic basis for Kleine-Levin syndrome.

✓AU: Carpenter-S; Yassa-R; Ochs-R

SO: Arch-Neurol. 1982 Jan; 39(1): 25-8

LA: ENGLISH

AB: A patient with Kleine-Levin syndrome, typical except that onset was at 39 years of age, died during a symptomatic period. Autopsy disclosed recent and old lesions in the medial thalamus involving intralaminar, medial, and some dorsal nuclei as well as the pulvinar. Despite massive microglial infiltration, there was minimal neuronal loss. The hypothalamus was not involved. The findings suggest a viral cause for Kleine-Levin syndrome.

AN: 82112671

TI: [Differential diagnostic problems and polygraphic studies in a case of Pickwickian syndrome]

_AU: Zeitlhofer-J; Mamoli-B; Wolf-C

SO: Wien-Klin-Wochenschr. 1982 Sep 3; 94(16): 434-8

LA: GERMAN

AB: The case report of a male patient with the Pickwickian syndrome is presented. The results of the relevant neurophysiological examinations (polygraphic registration of EEG, electrocardiogram, respiration, thorax excursions, submental and intercostal electromyogram, electrooculogram) give an insight into the pathomechanisms and enable the differential diagnosis to be made between this condition and narcolepsy and also the Kleine-Levin syndrome; respiratory investigations (spirometry, hypercapnic ventilatory response: mouth occlusion pressure) make it possible to differentiate between the Pickwickian and Ondine's curse syndrome. The therapeutic management is discussed.

AN: 83069732

TI: The Kleine-Levin syndrome: a review.

✓AU: Orlosky-MJ

SO: Psychosomatics. 1982 Jun; 23(6): 609-21

LA: ENGLISH

AN: 83015699

TI: Case 9-7: Periodic hypersomnia and derealization in an 11-year-old girl

✓AU: Herskowitz, J. and Rosman, P.

SO: cited in **Pediatrics, Neurology and Psychiatry - Common Ground** (p. 216, 1982) [WS350 H572p}

TI: Neurotransmitter abnormalities in Kleine-Levin syndrome

_AU: Erman M., Stewart RM, Beckman B., Roffwarg H.

SO: Presented at the Annual Meeting of the Association of Sleep Disorder Centers, St. Louis 1982

AB: [Reported trial of medications to correct neurotransmitter metabolism]

(1981)

TI: **[Kleine-Levin syndrome--a case contribution]**

_AU: Greger-J; Lemke-S

SO: Z-Arztl-Fortbild-Jena. 1981 Nov 1; 75(21): 1003-5

LA: GERMAN

AN: 82177966

TI: **[Periodic hypersomnia--The Kleine-Levin syndrome and its problems. A review of recent cases]**

_AU: Messimy-R

SO: Int-J-Neurol. 1981; 15(1-2): 119-31

LA: FRENCH

AN: 92283582

AN: 86032720

TI: **Further observations on sleep abnormalities in Kleine-Levin syndrome: abnormal breathing pattern during sleep.**

✓AU: Lavie-P; Klein-E; Gadoth-N; Bental-E; Zomer-J; Bechar-M; Wajsbort-J

SO: Electroencephalogr-Clin-Neurophysiol. 1981 Jul; 52(1): 98-101

LA: ENGLISH

AB: In two adolescent and two adult patients with Kleine-Levin syndrome, polygraphic sleep recording performed during somnolent and non-somnolent periods revealed various forms of abnormal breathing patterns during sleep. These included periodic breathing and hypopnoeic episodes associated with brief arousals and, in one adult patient, a full blown sleep apnoea syndrome. It is suggested that abnormal breathing in sleep in this syndrome may result from central hypoexcitability.

AN: 81236354

(1980)

Fortschr Neurol Psychiatr Grenzgeb 1980 May;48(5):267-78

[The Kleine-Levin-Critchley-syndrome. A contribution to the solution of differential diagnosis].

LA: GERMAN

___ Hagel K, Freytag H, Kindt H

AB: The typical symptoms of the so-called Kleine-Levin-Critchley-Syndrome are described according to our own observation of four selected patients (one woman, three men) on the background of the literature on the subject. In contrast to the first descriptions we characterise this syndrome by the Trias: periodic hypersomnia, vegetative disturbances (especially of food intake) and psychopathologic symptoms.

Especially young men in their second decade of life suffer from this syndrome. As far as women are concerned the disease is found more seldom but the authors are sure that it exists. A spontaneous remission of the periodically proceeding disease often occurs in the third decade of life. In analogy to the respective literature a retardation of the EEG during the course of the disease going along with otherwise inconspicuous neurology was observed by the authors. Polygraphic-EEG studies are seldom. The cause of the syndrome is still open to question. Our observation show that a primarily disposition-linked and a secondarily acquisition-linked form can be assumed. Good effects are put down to amphetamines used for therapy. As far as differential diagnosis is concerned infections and abusos must be excluded.

Wien Med Wochenschr. 1980 Jun 15;130(11):373-5.

[Kleine-Levin-syndrome (author's transl)]

[Article in German]

Lenz H.

A report is given on a 18 year old girl with a Kleine-Levin-syndrome, which was studied 4 years after beginning in the menarche. At first there were only attacks of nausea and vertigo. One year later the attacks were combined with hypersomnia for several hours. Remarkable was a minimal overirritability of the right vestibular system and a slight hypersensitivity to touch and pain on the whole right side. Worth mentioning is also a disturbed water balance (retention). A disturbance of the hypothalamic regulation is supposed and methylphenidate-hydrochloride (Rilatin) is commanded for treatment.

TI: [Kleine-Levin syndrome]

_AU: Argentino-C; Sideri-G

SO: Riv-Neurol. 1980 Jan-Feb; 50(1): 26-31

LA: ITALIAN

AB: A case of a 15 years old boy with a Kleine-Levin syndrome is presented. Neurological examination and X-ray studies, including pneumoencephalography, were normal. Laboratory findings were also normal except for a flattened response to glucose load. The EEG was mildly abnormal, showing bifrontal discharges at 3 c/s, a finding already presented in other cases of Kleine-Levin syndrome. The differential diagnoses among the group of periodic hypersomnias is discussed.

AN: 81125939

TI: [Kleine-Levin syndrome]

_AU: Iakhno-NN

SO: Sov-Med. 1980(5): 109-11

LA: RUSSIAN

AN: 80213994

AB: [a case report]

TI: Similarities in EEG sleep findings for Kleine-Levin syndrome and unipolar depression.

✓AU: Reynolds-CF 3d; Black-RS; Coble-P; Holzer-B; Kupfer-DJ

SO: Am-J-Psychiatry. 1980 Jan; 137(1): 116-8

LA: ENGLISH
AN: 80107001

TI: **[Kleine-Levin syndrome -- lithium prophylaxis]**

_AU: Roth-B; Smolik-P; Soucek-K

SO: Ceskoslovenska-Psychiatrie. 1980 Jun; 76(3): 156-62

LA: CZECH

AN: 81042445

TI: **Familial hibernation (Kleine-Levin) syndrome**

✓AU: Popper, J. S., Hsia, Y. E., Rogers, T. and Yuen, J.

SO: (Abstract) Am. J. Hum. Genet. 32: 123A 1980

AB: A Hawaii-Caucasian family with nine affected members (5 male, 4 female).

[Note: partial retraction, personal communication]

(1979)

TI: **Sleep patterns in Kleine-Levin syndrome.**

✓AU: Lavie-P; Gadoth-N; Gordon-CR; Goldhammer-G; Bechar-M

SO: Electroencephalogr-Clin-Neurophysiol. 1979 Sep; 47(3): 369-71

LA: ENGLISH

AB: Diurnal and nocturnal sleep records were obtained from a male and a female with Kleine-Levin syndrome, during excessive daytime sleep attacks and while they were asymptomatic. A common pattern of abnormal sleep was seen in both patients even during the asymptomatic period. The female, afflicted with a severe form of periodic hypersomnia, showed nocturnal and diurnal sleep onset REM periods. The different pattern of sleep abnormality in the female could be an expression of the severity of her symptoms or might indicate a variant of sleep abnormality present only in females with Kleine-Levin syndrome.

AN: 80024062

TI: **Kleine-Levin syndrome: a case report**

_AU: Agarwal, A. K. & Agarwal, A. K.

SO: Indian Journal of Psychiatry 21:286-7 1979

(1978)

TI: **Kleine-Levin syndrome with periodic apnea during hypersomnic stages--
E.E.G. study.**

_AU: Vardi-J; Flechter-S; Tupilsky-M; Rabey-JM; Carasso-R; Streifler-M

SO: J-Neural-Transm. 1978; 43(2): 121-32

LA: ENGLISH

AB: A 33 year old male, suffering from Kleine-Levine syndrome associated with periods of apnea during the hypersomnic attacks, is reported. Ventilatory studies negate the Pickwickian syndrome. The E.E.G.'s recorded during the hypersomnic attacks and the apneic periods showed a direct correlation between high-voltage delta waves paroxysmal E.E.G. activity, and apneic period. Medications known to improve Kleine-

Levin syndrome, in our case, had no effect upon the clinical hypersomnic and apnea periods, nor on the correlatives E.E.G.'s pattern and spirometric studies. Theoretical considerations let us assume that these paroxysmal E.E.G. patterns associated with apnea are NREM-sleep serotonin dependent, and have an inhibitory influence on the respiratory centers, by alternating the equilibrium between the catecholamines and acetylcholine activities.

AN: 79090178

TI: Kleine-Levin syndrome associated with fire setting.

✓AU: Powers-PS; Gunderman-R

SO: Am-J-Dis-Child. 1978 Aug; 132(8): 786-9

LA: ENGLISH

AB: Kleine-Levin syndrome is characterized by periodic hypersomnia associated with megaphagia and striking behavioral and psychiatric symptoms; it occurs primarily in adolescent boys. We treated a 17-year-old boy who had typical recurring somnolent episodes. His bizarre behavior included fire setting and stealing, both of which may have represented compulsions. His EEG during a sleepy episode was diffusely abnormal with generalized slowing of background activity; as he awoke and improved clinically, his EEG returned to normal. Between episodes, his EEG was normal. The relationship between Kleine-Levin syndrome and other sleep disorders is discussed. The neurochemistry and neurocircuitry that may provide the requisite substrate for this complex and fascinating neuropsychiatric disorder are briefly reviewed.

AN: 78254079

TI: [New contribution to the clinical aspects and the pathophysiology of the Kleine-Levin syndrome]

_AU: Bucking-PH; Palmer-WR

SO: MMW-Munch-Med-Wochenschr. 1978 Nov 24; 120(47): 1571-2

LA: GERMAN

AN: 79072965

TI: [Kleine-Levin syndrome. A propos of a case]

_AU: Billard-C; Ponsot-G; Lyon-G; Arfel-G

SO: Arch-Fr-Pediatr. 1978 Apr; 35(4): 424-31

LA: FRENCH

AN: 78255994

TI: The Kleine-Levine syndrome--a variant?

✓AU: Yassa-R; Nair-NP

SO: J-Clin-Psychiatry. 1978 Mar; 39(3): 254-9

LA: ENGLISH

AB: A case of Kleine-Levin syndrome in an older man is described, with a review of the literature. An important point is emphasized in our case in which the patient reacted unfavorably to Ritalin, becoming sexually aroused, although his hypersomnia improved. A possibly different mechanism in hypersomnia and hypersexuality is speculated.

AN: 78129967

(1977)

TI: **Abnormal central monoamine metabolism in humans with "true hypersomnia" and "sub-wakefulness"**.

✓AU: Livrea-P; Puca-FM; Barnaba-A; Di-Reda-L

SO: Eur-Neurol. 1977; 15(2): 71-6

LA: ENGLISH

AB: A case of Kleine-Levin syndrome with true hypersomnia and a case of sub-wakefulness are described. In both patients lumbar cerebrospinal fluid homovanillic acid, 5-hydroxyindoleacetic acid, 3-methoxy-4-hydroxyphenylethylene glycol levels have been assayed during episodes of hypersomnia and normal sleep-waking cycles. Besides an increased 5-hydroxytryptamine turnover, mainly an increased dopamine turnover has been detected in both kinds of hypersomnia, and this finding was more remarkable in the case with sub-wakefulness. The probable role of dopamine in abnormalities in the sleep-waking cycle is discussed on the basis of results in experimental animal hypersomnias.

AN: 77162098

TI: **[Kleine-Levin syndrome]**

_AU: Iizuka-R; Nakamura-M

SO: Nippon-Rinsho. 1977 Spring; 35 Suppl 1: 688-9

LA: JAPANESE

AN: 78174632

TI: **[Kleine-Levin syndrome]**

✓AU: Turgman-J; Braham-J

SO: Harefuah. 1977 Jun 15; 92(12): 552-4

LA: HEBREW

AN: 77226223

TI: **Lithium prophylaxis of periodic hypersomnia [letter]**

✓AU: Abe K

SO: Br. J. Psychiatry 130:312-36 1977

LA: ENGLISH

AB: Li use

(1976)

TI: **Behavioral manifestations of the Kleine-Levin syndrome.**

_AU: Chiles-JA; Wilkus-RJ

SO: Dis-Nerv-Syst. 1976 Nov; 37(11): 646-8

LA: ENGLISH

AB: The authors present a case of the Kleine-Levin syndrome, and review the literature to assess the various ways this illness can appear. Psychiatric manifestations not only obscure the diagnosis, but may lead to inappropriate treatment.

AN: 77048140

TI: [**Kleine-Levin syndrome. Female cases and catamneses**]

_AU: Wenzel-U

SO: Fortschr-Neurol-Psychiatr-Grenzgeb. 1976 Apr; 44(4): 137-50

LA: GERMAN

AB: The Kleine-Levin syndrome (periodic hypersomnia and megaphagia in adolescence) was considered till 1962 to be a disorder limited to the male adolescents. Since 1924, however, a few female cases have been reported about. These, being approximately 20 patients, are registered by their essential symptoms together with 2 additional cases observed by the author. Moreover, prospective catamneses, covering periods of more than 12 years, were described for the first time. Probably, the Kleine-Levin-syndrome is the product of disturbed functions involving structures of hypothalamus and the reticular system. The syndrome seems to belong to the same groups of periodical diseases as migraine: in particular it exhibits similar relations to the genital cycle. Therapeutical success was attained by administration of amphetamines. In course of time, the episodes become spontaneously rarer and mostly cease.

AN: 76166433

TI: **Kleine-Levin syndrome in a female patient.**

✓AU: Takrani-LB; Cronin-D

SO: Can-Psychiatr-Assoc-J. 1976 Aug; 21(5): 315-8

LA: ENGLISH

AN: 77046782

TI: **Treatment of Periodic Somnence with Lithium Carbonate**

✓AU: Ogura, C., Okuma, T., Nakazawa, K., Kishimoto, A.

SO: Archives in Neurology 33: 143 1976

(1975)

TI: **Electrophysiological changes during episodes of the Kleine-Levin syndrome.**

✓AU: Wilkus-RJ; Chiles-JA

SO: J-Neurol-Neurosurg-Psychiatry. 1975 Dec; 38(12): 1225-31

LA: ENGLISH

AB: Diurnal EEGs of a 17 year old male with the Kleine-Levin syndrome revealed moderate diffuse abnormalities and stage REM at sleep onset during attacks. Overnight, stages 3, 4, and REM of sleep were decreased, but sleep onset REM stage was not seen. These records returned to normal between attacks.

AN: 76145398

TI: **A menstruation-linked periodic hypersomnia. Kleine-Levin syndrome or new clinical entity?**

✓AU: Billiard-M; Guilleminault-C; Dement-WC

SO: Neurology. 1975 May; 25(5): 436-43

LA: ENGLISH

AB: A 13-year-old girl showed periodic episodes of somnolence without megaphagia recurring in association with each menstruation. During somnolent episodes total sleep time averaged 14 hours and 19 minutes per 24 hours. The level of performance evaluated by means of the Wilkinson Addition Test was significantly impaired. There was an abnormal increase of 5-hydroxyindolacetic acid in the cerebrospinal fluid after probenecid test, suggesting an increase of the turnover of 5-hydroxytryptamine during periodic hypersomnia. Investigation of the menstrual cycle failed to document any striking hormonal abnormality. Nevertheless, the close relationship between the episodes of hypersomnia and the end of the menstrual cycle led us to hypothesize a role of progesterone and to try a hormonal type of treatment that is thus far successful.
AN: 75175396

TI: **[Report on a case of periodic hypersomnia (Kleine-Levin syndrome)]**

_AU: Schlierf-C

SO: Nervenarzt. 1975 Jun; 46(6): 317-24

LA: GERMAN

AN: 76075876

TI: **A possible neuroendocrine basis of two clinical syndromes: Anorexia nervosa and the Kleine-Levin syndrome**

_AU: Young, JK

SO: *Physiol Psychol* 3:322-330 1975

(1974)

TI: **The Kleine-Levin syndrome. Case report and review of the literature.**

✓AU: Frank-Y; Braham-J; Cohen-BE

SO: *Am-J-Dis-Child*. 1974 Mar; 127(3): 412-3

LA: ENGLISH

AN: 74110089

(1973)

TI: **[New observations in a case of Kleine-Levin syndrome]**

_AU: Gran-D; Begemann-H

SO: *Munch-Med-Wochenschr*. 1973 Jun 8; 115(23): 1098-102

LA: GERMAN

AN: 74261906

TI: **[Pathophysiologic mechanisms of periodic sleep and the Kleine-Levin syndrome]**

_AU: Lobzin-VS; Shamrei-RK; Churilov-IuK

SO: *Zh-Nevropatol-Psikhiatr*. 1973; 73(11): 1719-24

LA: RUSSIAN

AN: 74099561

TI: A case of Kleine-Levin syndrome.

_AU: McLaughlin-F

SO: J-Ir-Med-Assoc. 1973 Jun 23; 66(12): 324

LA: ENGLISH

AN: 73226439

TI: Periodic megaphagia and hypersomnia--an example of the Kleine-Levin syndrome in an adolescent girl.

_AU: Gilligan-BS

SO: Proc-aust-Assoc-Neurol. 1973; 9: 67-72

LA: ENGLISH

AN: 73221624

TI: Depression and mania associated with Kleine-Levin-Critchley syndrome

✓AU: Jeffries J., Lefebvre A.

SO: Can Psychiatr Assoc J 18:439-444 1973

(1972)

TI: [Clinical and polygraphic study of a case of Kleine-Levin Critchley syndrome during a 24-hour period]

_AU: Popoviciu-L; Corfariu-O

SO: Rev-Roum-Neurol. 1972; 9(4): 221-8

LA: FRENCH

AN: 73180691

TI: [Kleine-Levin syndrome]

_AU: Ledic-P; Milohanovic-S; Willheim-K

SO: Neuropsihijatrija. 1972; 20(3): 335-40

LA: SERBO-CROATIAN-ROMAN

AN: 74112392

TI: A case of atypical Kleine-Levin syndrome: 30 years' observation.

✓AU: Wilder-J

SO: J-Nerv-Ment-Dis. 1972 Jan; 154(1): 69-72

LA: ENGLISH

AN: 72081129

TI: A case of Kleine-Levin syndrome

_AU: Narayanan, H.S., Narayana Reddy, G.N. & Rama Rao, B.S.S.

SO: Indian Journal of Psychiatry 14:356-9 1972

(1971)

TI: [Kleine-Levin syndrome (recurrent hypersomnia of male adolescents)]

_AU: Fresco-R; Giudicelli-S; Poinso-Y; Tatossian-A; Mouren-P
SO: Ann-Med-Psychol-Paris. 1971 May; 1(5): 625-68
LA: FRENCH
AN: 71253889

TI: [**Lethargy and gluttony as an organic syndrome (Kleine-Levin syndrome)**]

_AU: Overweg-J
SO: Ned-Tijdschr-Geneskd. 1971 Mar 27; 115(13): 556-8
LA: DUTCH
AN: 71155729

(1970)

TI: [**2 cases of Kleine-Levin syndrome**]

_AU: Fresco-R; Blumen-G; Tatossian-A; Sutter-JM
SO: Rev-Neuropsychiatr-Infant. 1970: Suppl:55-9
LA: FRENCH
AN: 72256611

TI: [**EEG study in a case of Kleine-Levin syndrome**]

_AU: Smirne-S; Castellotti-V; Passerini-D; Lizzi-F
SO: Riv-Neurol. 1970 Sep-Oct; 40(5): 357-65
LA: ITALIAN
AN: 71135033

TI: **Kleine-Levin syndrome. A case with EEG evidence of periodic brain dysfunction.**

✓AU: Green-LN; Cracco-RQ
SO: Arch-Neurol. 1970 Feb; 22(2): 166-75
LA: ENGLISH
AN: 70065771

TI: **EEG in Kleine-Levin syndrome.**

✓AU: Green-LN; Cracco-RQ
SO: Electroencephalogr-Clin-Neurophysiol. 1970 Sep; 29(3): 325
LA: ENGLISH
AN: 70287375

TI: [**Kleine-Levin syndrome**]

_AU: Kandic-B; Lesic-Z; Dordevic-D; Zdravkovic-P
SO: Med-Pregl. 1970; 23(1): 49-50
LA: SERBO-CROATIAN-CYRILLIC
AN: 71052149

TI: [**Kleine-Levin syndrome: observations during a course of hypothymic episodes**]

_AU: Cante-C; Marocchino-R

SO: Osp-Psichiatri. 1970 Oct-Dec; 38(4): 603-13
LA: ITALIAN
AN: 72105877

TI: **Kleine-Levin syndrome: an atypical case?**
_AU: Miller-DL
SO: Psychiatr-Q. 1970; 44(1): 26-35
LA: ENGLISH
AN: 72052669

TI: **A case of the Kleine-Levin syndrome of long duration.**
✓AU: George-HR
SO: Br-J-Psychiatry. 1970 Nov; 117(540): 521-3
LA: ENGLISH
AN: 71035601

TI: **A case of Kleine-Levin syndrome in India.**
✓AU: Prabhakaran-N; Murthy-GK; Mallya-UL
SO: Br-J-Psychiatry. 1970 Nov; 117(540): 517-9
LA: ENGLISH
AN: 71035600

TI: **The Kleine-Levin syndrome. Report of a case and discussion.**
✓AU: Persson-T; Olsson-L; Ortman-E
SO: Acta-Psychiatr-Scand. 1970; 46(2): 106-10
LA: ENGLISH
AN: 71030935

(1969)

TI: **The Kleine-Levin syndrome: report of a case and discussion.**
✓AU: Persson-T; Olsson-L; Ortman-E
SO: Behav-Neuropsychiatry. 1969 Sep; 1(6): 4-6
LA: ENGLISH [essentially same paper as above]
AN: 70138092

TI: **[Kleine-Levin syndrome (periodic hypersomnia in adolescent males): 2 personal cases]**
_AU: Blumen-G; Fresco-R; Giudicelli-S; Tatossian-A; Mouren-P; Sutter-JM
SO: Mars-Med. 1969; 106(4): 321-6
LA: FRENCH
AN: 70186798

TI: **The EEG in a case of periodic hypersomnia.**
_AU: Thacore VR, Ahmed M, Oswald I
SO: Electroencephalogr Clin Neurophysiol 1969 Dec;27(6):605-6

(1968)

TI: **The Kleine-Levin syndrome. A case study with a psychopathologic approach.**

✓AU: Haberland-C; Weissman-S

SO: Acta-Psychiatr-Scand. 1968; 44(1): 1-10

LA: ENGLISH

AN: 69169271

TI: **Periodic hypersomnia.**

_AU: Elian M

SO: Electroencephalogr Clin Neurophysiol 1968 Feb;24(2):193

TI: **A female case of the Kleine-Levin Syndrome.**

✓AU: Duffy-JP; Davison-K

SO: Br-J-Psychiatry. 1968 Jan; 114(506): 77-84

LA: ENGLISH

AN: 68124222

(1967)

TI: **The Kleine-Levin syndrome.**

_AU: Gallinek-A

SO: Dis-Nerv-Syst. 1967 Jul; 28(7 Pt 1): 448-51

LA: ENGLISH

AN: 90126199

TI: **[Contribution to the study of the Kleine-Levin syndrome in early childhood]**

_AU: Lamote-de-Grignon-C; Fernandez-Alvarez-E

SO: Rev-Neuropsychiatr-Infant. 1967 Apr-May; 15(4): 365-72

LA: FRENCH

AN: 69040593

TI: **[Polygraphic and clinical findings in a case of periodic hypersomnia with megaphagia (Kleine-Levin syndrome)]**

_AU: Barontini-F; Zappoli-R

SO: Riv-Neurol. 1967 Sep-Oct; 37(5): 477-86

LA: ITALIAN

AN: 68233629

TI: **Psychopathological investigation of a case of periodic hypersomnia and Bulimia (Kleine-Levin syndrome).**

✓AU: Jaffe-R

SO: Isr-Ann-Psychiatr-Relat-Discip. 1967; 5(1): 43-52

LA: ENGLISH

AN: 68099401

TI: **[The syndrome of hypersomnia and periodical megapagia in the adult male (Kleine-Levin): what is its natural course?]**

_AU: Critchley-M

SO: Revue Neurologique (Paris). 1967 Jun; 116(6): 647-50

LA: FRENCH

AN: 70007386

TI: **[Periodic hypersomnia with megaphagia in adolescent males: Kleine-Levin syndrome. A case with an electroencephalographic study and comments]**

_AU: Insua-JA; Turner-M; Ague-C

SO: Prensa-Med-Argent. 1967 Mar 10; 54(2): 49-54

LA: SPANISH

AN: 69118329

TI: **[Syndrome of recurring somnolence of Kleine-Levin]**

_AU: Kozik-M

SO: Neurol-Neurochir-Pol. 1967 May-Jun; 1(3): 405-7

LA: POLISH

AN: 68017099

TI: **[Contribution to the clinical study of sleep abnormalities. 3 cases of "periodic somnolence" (Kleine-Levin)]**

_AU: Haynal-A; Regli-F

SO: Encephale. 1967 Jan-Feb; 56(1): 33-44

LA: FRENCH

AN: 67202993

TI: **Kleine-Levin syndrome: report of a case.**

✓AU: Markman-RA

SO: Am-J-Psychiatry. 1967 Feb; 123(8): 1025-6

LA: ENGLISH

AN: 67090082

(1965)

TI: **The Kleine-Levin syndrome. Some further observations.**

✓AU: Garland-H; Sumner-D; Fourman-P

SO: Neurology. 1965 Dec; 15(12): 1161-7

LA: ENGLISH

AN: 66106651

TI: **[A case of Kleine-Levin syndrome]**

_AU: Azuma-H; Murakami-M; Mori-J

SO: Nippon-Naika-Gakkai-Zasshi. 1965 Jul 10; 54(4): 342-8

LA: JAPANESE

AN: 66009588

Psychiatr Q. 1965 Jan;39:79-83

Periodic Hypersomnia and Megaphagia (The Kleine-Levin Syndrome).

_ Earle BV.

Neurology. 1964 Sep;14:844-50.

Periodic Hypersomnia and Bulimia. (The Kleine-Levin Syndrome).

_ Gilbert GJ.

TI: **Periodic hypersomnia and megaphagia in adolescent males**

✓AU: Critchley, M.

SO: Brain. 85: 627-656 (1962)

Rev Clin Esp. 1951 Dec 15;43(5):341-5

[Periodic hypersomnia and morbid hunger (Levin's syndrome) as manifestations of genuine epilepsy.]

[Article in Undetermined Language]

_ Castilla Del Pino C.

Br J Med Psychol. 1951;24(4):296-300.

A case of periodic hypersomnia.

_ O'Connor WA.

TI:

_AU: Critchley, M. and Hoffman, H.

SO: Br. Med. J. 1:137 (1942)

TI: **Periodic somnolence and morbid hunger: a new syndrome**

✓AU: Levin, M.

SO: Brain 59: 494-504 (1936)

TI: **Narcolepsy (Gelineau's syndrome) and other varieties of morbid somnolence**

_AU: Levin, M.

SO: Arch Neurol. Psychiat. 22: 1172-1200 (1929)

TI: **Periodische Sclafsucht**

_AU: Kleine, W.

SO: Monatssch. Psychiatr. Neurol. 57: 285-298 (1925)

Supplemental Bibliography for “periodic hypersomnia”

Arch Neurol. 2002 Oct;59(10):1553-62.

The role of cerebrospinal fluid hypocretin measurement in the diagnosis of narcolepsy and other hypersomnias.

_ Mignot E, Lammers GJ, Ripley B, Okun M, Nevsimalova S, Overeem S, Vankova J, Black J, Harsh J, Bassetti C, Schrader H, Nishino S.

Center for Narcolepsy, Stanford University, 701B Lower Welch Rd, Palo Alto, CA 94304-5742, USA. mignot@leland.stanford.edu

CONTEXT: Narcolepsy, a neurological disorder affecting 1 in 2000 individuals, is associated with HLA-DQB1*0602 and low cerebrospinal fluid (CSF) hypocretin (orexin) levels. OBJECTIVES: To delineate the spectrum of the hypocretin deficiency syndrome and to establish CSF hypocretin-1 measurements as a diagnostic tool for narcolepsy. DESIGN: Diagnosis, HLA-DQ, clinical data, the multiple sleep latency test (MSLT), and CSF hypocretin-1 were studied in a case series of patients with sleep disorders from 1999 to 2002. Signal detection analysis was used to determine the CSF hypocretin-1 levels best predictive for International Classification of Sleep Disorders (ICSD)-defined narcolepsy (blinded criterion standard). Clinical and demographic features were compared in narcoleptic subjects with and without low CSF hypocretin-1 levels. SETTING: Sleep disorder and neurology clinics in the United States and Europe, with biological testing performed at Stanford University, Stanford, Calif. PARTICIPANTS: There were 274 patients with narcolepsy; hypersomnia; obstructive sleep apnea; restless legs syndrome; insomnia; and atypical hypersomnia cases such as familial cases, narcolepsy without cataplexy or without HLA-DQB1*0602, recurrent hypersomnias, and symptomatic cases (eg, Parkinson disease, depression, Prader-Willi syndrome, Niemann-Pick disease type C). The subject group also included 296 controls (healthy and with neurological disorders). INTERVENTION: Venopuncture for HLA typing, lumbar puncture for CSF analysis, primary diagnosis using the International Classification of Sleep Disorders, Stanford Sleep Inventory for evaluation of narcolepsy, and sleep recording studies. MAIN OUTCOME MEASURES: Diagnostic threshold for CSF hypocretin-1, HLA-DQB1*0602 positivity, and clinical and polysomnographic features. RESULTS: HLA-DQB1*0602 frequency was increased in narcolepsy with typical cataplexy (93% vs 17% in controls), narcolepsy without cataplexy (56%), and in essential hypersomnia (52%). Hypocretin-1 levels below 110 pg/mL were diagnostic for narcolepsy. Values above 200 pg/mL were considered normal. Most subjects with low levels were HLA-DQB1*0602-positive narcolepsy-cataplexy patients. These patients did not always have abnormal MSLT. Rare subjects without cataplexy, DQB1*0602, and/or with secondary narcolepsy had low levels. Ten subjects with hypersomnia had intermediate levels, 7 with narcolepsy (often HLA negative, of secondary nature, and/or with atypical cataplexy or no cataplexy), and 1 with periodic hypersomnia. Healthy controls and subjects with other sleep disorders all had normal levels. Neurological subjects had generally normal levels (n = 194). Intermediate (n = 30) and low (n = 3) levels were observed in various acute neuropathologic conditions. CONCLUSIONS: Narcolepsy-cataplexy

with hypocretin deficiency is a genuine disease entity. Measuring CSF hypocretin-1 is a definitive diagnostic test, provided that it is interpreted within the clinical context. It may be most useful in cases with cataplexy and when the MSLT is difficult to interpret (ie, in subjects already treated with psychoactive drugs or with other concurrent sleep disorders).

Neurophysiol Clin. 2001 Dec;31(6):356-75.

Sleep and brain lesions: a critical review of the literature and additional new cases.

_ Autret A, Lucas B, Mondon K, Hommet C, Corcia P, Saudeau D, de Toffol B.
Service de neurologie CHU Bretonneau, 37044 Tours, France. alain.autret@med.univ-tours.fr

We present a comprehensive review of sleep studies performed in patients with brain lesions complemented by 16 additional personal selected cases and by discussion of the corresponding animal data. The reader is cautioned about the risk of establishing an erroneous correlation between abnormal sleep and a given disorder due to the important inter and intra variability of sleep parameters among individuals. Salient points are stressed: the high frequency of post-stroke sleep breathing disorders is becoming increasingly recognised and may, in the near future, change the way this condition is managed. Meso-diencephalic bilateral infarcts induce a variable degree of damage to both waking and non-REM sleep networks producing an abnormal waking and sometimes a stage 1 hypersomnia reduced by modafinil or bromocriptine, which can be considered as a syndrome of catecholaminergic deficiency. Central pontine lesions induce REM and non-REM sleep insomnia with bilateral lateral gaze paralysis. Bulbar stroke leads to frequent sleep breathing disorders. Polysomnography can help define the extent of involvement of various degenerative diseases. Fragmented sleep in Parkinson's disease may be preceded by REM sleep behavioural disorders. Multiple system atrophies are characterised by important sleep disorganization. Sleep waking disorganization and a specific ocular REM pattern are often seen in supra-nuclear ophthalmoplegia. In Alzheimer patients, sleep perturbations parallel the mental deterioration and are possibly related to cholinergic deficiency. Fronto-temporal dementia may be associated with an important decrease in REM sleep. Few narcoleptic syndromes are reported to be associated with a tumour of the third ventricle or a multiple sclerosis or to follow a brain trauma; all these cases raise the question whether this is a simple coincidence, a revelation of a latent narcolepsy or, as in non-DR16/DQ5 patients, a genuine symptomatic narcolepsy. Trypanosomiasis and the abnormal prion protein precociously alter sleep patterns. Polysomnography is a precious tool for evaluating brain function provided it is realised under optimal conditions in stable patients and interpreted with caution. Several unpublished cases are presented: one case of pseudohypersomnia due to a bilateral thalamic infarct and corrected by modafinil, four probable late-onset autosomal recessive cerebellar ataxias without sleep pattern anomalies, six cases of fronto-temporal dementia with strong reduction in total sleep time and REMS percentage on the first polysomnographic night, one case of periodic hypersomnia associated with a Rathke's cleft cyst and four cases of suspected symptomatic narcolepsy with a DR16-DQ5 haplotype, three of which were post-traumatic without MRI anomalies, and one associated with multiple sclerosis exhibiting

pontine hyper signals on MRI.
Type: Review

J Assoc Physicians India. 1998 Apr;46(4):399.

Periodic hypersomnia.

_ Desai N, Diwan P, Kelkar PN.

Dept of Psychiatry, LTMMC and LTMG Hospital, Sion, Mumbai-400 022.

Type: Case Report

J Neurol Neurosurg Psychiatry. 1998 Jan;64(1):113-6.

Novel chromosomal aberration in a patient with a unique sleep disorder.

_ Hasegawa Y, Morishita M, Suzumura A.

Department of Neurology, Prefectural Tajimi Hospital, Maehata, Japan.

A 45 year old woman presenting with periodic hypersomnia for 17 years is reported on. She would sleep for three weeks followed by the same period awake. Polysomnography in the somnolent period disclosed an excess of total sleeping time with remarkably increased stage 1, 3/4, and REM sleep, without cataplexy or sleep paralysis. HLA typing was incompatible with narcolepsy or REM sleep behavioural disorder. Her chromosomes showed premature centromere division with chromatid puffing in areas of constitutive heterochromatin, which is exclusively found in the syndrome of infants termed Roberts' syndrome/SC phocomelia. Other laboratory findings were not normal. It is suggested that the present case is a novel sleep disorder related to a unique chromosomal aberration.

Type: Case Report

Jpn J Psychiatry Neurol 1993 Jun;47(2):457-9

Polysomnography and chronobiologic study on periodic hypersomnia.

__ Uchimura N, Sakurai S, Sakamoto T, Kotorii T, Nakamura J, Maeda Y, Tsuchiyama Y, Nakazawa Y

Department of Neuropsychiatry, Kurume University School of Medicine.

Jpn J Psychiatry Neurol 1992 Jun;46(2):489-91

Periodic hypersomnia: a case with very early onset, age 7.

__ Hattori H, Maihara T, Hirao T

Department of Pediatrics, Kitano Hospital, Tazuke Kofukai Medical Research Institute, Osaka.

Zeitschrift fur Arztliche Fortbildung 1992 Oct;86(20):1025-30

[Rapid cycling syndromes in Adolescence]

LA: GERMAN [have English translation]

✓_ Hassler F, Gierow B

Abteilung fur Psychiatrie und Neurologie des Kindes- und Jugendalters, Universitatsnervenlinik Rostock

Sleep 1991 Oct;14(5):460-3

Periodic hypersomnia: case report with biochemical and EEG findings.

___ Froscher W, Maier V, Fritschi T

Department of Neurology, University of Ulm, Germany.

AB: We report on a 23-year-old patient with periodic hypersomnia.

Electroencephalographic (EEG) background activity for this individual was slightly slowed in the EEG during an episode of hypersomnia, and intermittent slow activity was found in addition. Usual laboratory parameters were normal; however, leucine-enkephalin was markedly elevated in the plasma at that time, whereas free cysteine could not be demonstrated. Clinical findings were normal in the following years, and the EEG background activity returned to normal; leucine-enkephalin and cysteine also returned to normal values.

No To Hattatsu 1991 May;23(3):303-5

[A case of periodic hypersomnia: the effect of Tokishakuyakusan].

LA: JAPANESE

___ Sugimoto T, Ota T, Suzukawa Y, Nishida N, Yasuhara A

Ugeskr Laeger 1988 Mar 7;150(10):608-9

[Periodic hypersomnia. A case of possibly traumatic origin].

LA: DANISH

___ Bisgard C

Semin Neurol 1987 Sep;7(3):250-8

Disorders of excessive sleepiness: narcolepsy and hypersomnia.

___ Manfredi RL, Brennan RW, Cadieux RJ

Sleep Research and Treatment Center, Pennsylvania State University, College of Medicine, Hershey.

REVIEW: Besides sleep apnea, the main disorders of excessive daytime sleepiness include narcolepsy and hypersomnia. Narcolepsy is characterized by periods of irresistible sleepiness and sleep attacks of brief duration and, most often, by one or more of the auxiliary symptoms: cataplexy, sleep paralysis, and hypnagogic hallucinations. Generally, sleepiness and sleep attacks in hypersomnia are of longer duration and are more resistible than in narcolepsy; also, the auxiliary symptoms are absent. There are three types of hypersomnia: idiopathic, secondary, and periodic. Nocturnal sleep is typically disrupted in narcolepsy, whereas in idiopathic hypersomnia it is prolonged and in secondary hypersomnia it is variable. The exact causes of narcolepsy and idiopathic hypersomnia are unknown; however, there is evidence for genetic predisposition for either disorder. In secondary hypersomnia causative factors include: neurologic, such as head injuries, cerebrovascular insufficiency, and brain tumors; general medical, such as metabolic disorders, various intoxications, and conditions leading to brain hypoxia; and psychiatric, most notably depression. Although the cause of periodic hypersomnia is unclear, most research supports the notion of underlying organic disease. Often, the evaluation of patients with excessive daytime sleepiness can be completed in the office setting, based on the sleep history and a thorough neurologic, general medical, and psychiatric assessment. Whenever indicated, ancillary laboratory studies, such as computed tomography and magnetic resonance scans, should be performed. Sleep laboratory recordings generally are not necessary

unless there is suspicion of sleep apnea or narcolepsy in the absence of auxiliary symptoms.

Dtsch Med Wochenschr 1985 Apr 19;110(16):656-7

[Periodic hypersomnia and intermittent juvenile icterus. Coincidence or pathogenetic connection]?

LA: GERMAN

__ Berlitz P, Krause KH, Marx A

Type: Letter

Neurology 1982 Dec;32(12):1376-9

Menstruation-related periodic hypersomnia: a case study with successful treatment.

__ Sachs C, Persson HE, Hagenfeldt K

AB: A 16-year-old girl suffered from 1 to 2-week periods of hypersomnia associated with each menstruation. Serum hormone levels were normal. CSF concentrations of homovanillic acid and 5-hydroxyindolacetic acid were lower in hypersomniac than in symptom-free phases. 3-methoxy-4-hydroxyphenylethylene glycol was not affected. The sleep periods occurred only in connection with ovulatory menstrual cycles. When ovulation was inhibited by a combination of ethinylestradiol and lynestrenol, an oral contraceptive pill, the hypersomnia ceased. Thus, the hypersomnia seemed to be linked to the occurrence of ovulatory menstruations.

PIP: A 16-year old girl who had undergone normal pubertal development at 13 years 8 months began to menstruate with moderate or severe dysmenorrhea and 1 to 2 weeks of hypersomnia at 16 years 2 months. She was without symptoms between hypersomniac phases. The patient was followed for 3 years, in the hospital for 31 days and as an outpatient thereafter. Examination, including neurologic and gynecologic status, was normal. Serum levels of follicle stimulating hormone, luteinizing hormone, estradiol, and progesterone were normal. CSF concentrations of homovanillic acid and 5-hydroxyindolacetic acid were lower in her hypersomniac than in symptom-free phases. 3-methoxy-4-hydroxyphenylethylene glycol was not affected. The sleep periods occurred only in connection with ovulatory menstrual cycles. Inhibition of ovulation with the oral contraceptive pill Lyndiol, which contains a combination of 50 mcg ethinyl estradiol and 2.5 mg lynestrenol, led to a cessation of the hypersomnia. When treatment was discontinued, the patient had 2 cycles without ovulation and no sleep periods, but ovulation and periodic hypersomnia occurred regularly thereafter. Reinstitution of the contraceptive pill controlled the symptoms. Discontinuation of the treatment was tested 2 more times for 3 months each, and the ovulatory cycles were again accompanied by sleep periods.

Publication Type: Case Reports

J Neurol Sci 1977 Jan-Feb;31(1):13-27

235 cases of excessive daytime sleepiness. Diagnosis and tentative classification.

_ Guilleminault C, Dement WC

REVIEW: A series of 235 consecutive patients referred to the Stanford University Sleep Disorders Clinic with the complaint of excessive daytime sleepiness (EDS) were investigated extensively. A satisfactory final diagnosis involving a consistent syndrome or pathogenic process was made in all but 7 patients. In the course of this work a variety of tests, including prolonged polygraphic monitoring of multiple variables and CSF measurements before and after probenecid ingestion, were utilized. Different syndromes were confirmed (harmonious hypersomnia, subwakefulness syndrome); the definitions of others were clarified and extended (narcolepsy, drug dependency, periodic hypersomnia associated with menstruation, upper airway sleep apnea in children). Two new entities were tentatively identified (narcolepsy with sleep apnea, the neutral state syndrome). Narcolepsy and upper airway sleep apnea accounted for the majority of the cases (199). A strategic schema utilizing specific categories and frequency of occurrence in the case series is presented to improve the diagnosis of the complaint of excessive daytime sleepiness by the practicing physician. This case series was analysed in order to develop tentatively a meaningful nosology.

Electroencephalogr Clin Neurophysiol. 1969 Dec;27(6):605-6.

The EEG in a case of periodic hypersomnia.

_ Thacore VR, Ahmed M, Oswald I.

Electroencephalogr Clin Neurophysiol. 1968 Feb;24(2):193.

Periodic hypersomnia.

_ Elian M.

[end]