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NEUROPHYSIOLOGICAL AND CLINICAL CHARACTERISTICS OF PERSISTENT BEDWETTING IN ADOLESCENT PATIENTS

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Abstract: Enuresis, commonly known as bedwetting, is defined as the involuntary voiding of urine during sleep in individuals who have reached an age when bladder control would normally be expected. While typically resolved by early childhood, persistent bedwetting that continues into adolescence represents a particularly challenging clinical scenario with significant psychosocial implications. Despite affecting approximately 1-2% of adolescents aged 12-18 years, prolonged enuresis in this age group remains understudied compared to its presentation in younger children.

Key words: enuresis, adolescents, prolonged course, neurophysiological features, comorbidity, residual organic insufficiency.

Introduction. The persistence of enuresis into adolescence suggests complex underlying pathophysiological mechanisms that may differ from those observed in younger patients. Current evidence points to a multifactorial etiology involving neurological development delays, nocturnal polyuria, reduced bladder capacity, sleep arousal difficulties, and genetic predispositions. Particularly noteworthy are the neurophysiological aspects, including altered brainstem function, irregular antidiuretic hormone secretion patterns, and potential abnormalities in cortical arousal responses.

Adolescents with persistent enuresis face unique challenges compared to younger children, including heightened social stigma, limited treatment options specifically validated for their age group, and psychological comorbidities that may complicate clinical management. The transitional nature of adolescence, characterized by significant physiological and psychological changes, creates additional complexity in both understanding and addressing this condition.

This investigation aims to characterize the distinct neurophysiological and clinical profiles of adolescents with persistent enuresis, with particular attention to bladder function parameters, sleep architecture, arousal thresholds, and neurological biomarkers. By elucidating the specific features of this condition in adolescents, we seek to improve diagnostic accuracy, develop age-appropriate treatment protocols, and ultimately enhance quality of life for this vulnerable patient population.

The article examines current perspectives on the clinical and neurophysiological features of prolonged enuresis in adolescents. The main pathogenetic mechanisms, clinical manifestations, neurophysiological patterns, and comorbid conditions associated with this pathology are analyzed. Research findings on central nervous system functional insufficiency in adolescents with enuresis are presented, and modern approaches to diagnosis and treatment of the disease are discussed.

Research Objective. This investigation aimed to comprehensively evaluate the clinical and neurophysiological characteristics of chronic enuresis in adolescents to elucidate underlying pathogenetic mechanisms and establish differentiated diagnostic and treatment protocols for this condition.

Methodology. A prospective cohort investigation was conducted at Samarkand State Medical University's neurology department between September 2023 and March 2025, with local ethics committee approval. The study design included a primary study cohort and a reference group.

Study Participants

The investigation enrolled 120 adolescents between 12-17 years (average age 14.3±1.8 years), allocated into two groups:

- Study cohort: 80 adolescents experiencing persistent enuresis (condition lasting over 3 years), comprising 52 males (65%) and 28 females (35%)
- Reference group: 40 neurologically and psychiatrically healthy adolescents matched for age and gender distribution

The study cohort was further subdivided based on enuresis classification:

- Subgroup 1A: 46 adolescents with primary enuresis (present since early childhood)
- Subgroup 1B: 34 adolescents with secondary enuresis (developed after a dry period)

Enrollment Criteria

- Age range: 12-17 years inclusive
- Persistent enuresis exceeding 3 years duration
- Minimum frequency of two weekly bedwetting episodes over the preceding 3 months
- Documented informed consent from both participants and their legal guardians

Assessment Protocol

Comprehensive evaluation included detailed medical, family, and perinatal histories; standardized neurological examination; psychiatric assessment; and 14-day urination diaries documenting all enuresis episodes.

Statistical Analysis

Data processing employed SPSS Statistics 27.0 and Microsoft Excel 2019, calculating means (M) and standard deviations (SD) for quantitative measures and frequency distributions for qualitative parameters.

Results

Clinical Profile Assessment

Analysis revealed that among the 80 adolescents in the study cohort, 57.5% presented with primary enuresis and 42.5% with secondary enuresis. Nocturnal enuresis predominated (81.3%), with mixed nocturnal-diurnal patterns observed in 18.7%. No cases of isolated diurnal enuresis were identified.

Bedwetting frequency varied from 2-3 episodes weekly (52.5% of participants) to 4-6 episodes (31.3%) and daily occurrences (16.2%). Familial enuresis history was documented in 72.5% of cases, with significantly higher prevalence in primary enuresis (84.8%) versus secondary enuresis (55.9%), $p<0.01$. Perinatal complications were identified in 78.8% of the study cohort, with significantly higher incidence in secondary enuresis cases (88.2%) compared to primary enuresis (71.7%), $p<0.05$.

Neurological Findings

Neurological assessment identified subtle organic CNS abnormalities in 85% of the study cohort, including facial innervation asymmetry (42.5%), nystagmoid eye movements (38.8%), mild anisoreflexia (55%), fine motor dysfunction (63.7%), and subtle coordination impairments (47.5%). These neurological manifestations were significantly more prevalent and pronounced in secondary enuresis cases (94.1%) versus primary enuresis (78.3%), $p<0.05$.

Psychological Evaluation

Psychological assessment revealed emotional-volitional disturbances in 90% of the study cohort, including emotional lability (82.5%), elevated anxiety (75%), diminished self-esteem (70%), depressive symptoms (43.8%), and social adaptation difficulties (56.3%). Neuropsychological testing demonstrated reduced short-term memory capacity (7.2±1.3 versus 8.9±0.8 words in Luria test, $p<0.001$), extended Schulte table completion time (56.8±9.3 versus 45.2±6.1 sec, $p<0.001$), and diminished concentration (88.3±7.2% versus 96.1±2.8%, $p<0.001$) compared to reference subjects.

Quality of life assessment using PedsQL 4.0 indicated significantly reduced scores in the study cohort (65.3±8.7) compared to reference subjects (84.9±5.2), $p<0.001$, with particularly marked impairments in emotional functioning (58.2±10.3 versus 82.7±6.8, $p<0.001$) and social functioning (60.4±9.8 versus 86.3±5.9, $p<0.001$).

Electroencephalographic Findings

EEG analysis identified altered brain bioelectrical activity in 92.5% of the study cohort, characterized by:

- ✓ Disrupted alpha rhythm zonal distribution (67.5% versus 12.5% in reference group, $p<0.001$)
- ✓ Basic rhythm disorganization (78.8% versus 15%, $p<0.001$)
- ✓ Elevated theta-range slow-wave activity (62.5% versus 10%, $p<0.001$)
- ✓ Intensified beta activity (52.5% versus 17.5%, $p<0.001$)
- ✓ Non-epileptic paroxysmal activity (37.5% versus 5%, $p<0.001$)

Spectral analysis demonstrated significantly elevated frontal-central theta rhythm power (53.2±8.6 μV^2 versus 32.1±5.4 μV^2, $p<0.001$) and reduced occipital alpha rhythm power (68.3±10.2 μV^2 versus 92.4±7.6 μV^2, $p<0.001$). Coherence analysis revealed diminished intra- and interhemispheric alpha range coherence (0.62±0.08 versus 0.78±0.05, $p<0.001$), indicating disrupted functional connectivity between brain regions.

Secondary enuresis cases exhibited more pronounced EEG abnormalities, including elevated theta activity (56.3±7.4 μV^2 versus 48.7±6.9 μV^2, $p<0.01$) and reduced alpha activity (64.5±9.6 μV^2 versus 71.9±8.4 μV^2, $p<0.01$).

Polysomnographic Evaluation

Sleep architecture disturbances were identified in 95% of the study cohort, characterized by:

- ✓ Extended sleep onset latency (31.8±8.5 min versus 16.3±4.2 min, $p<0.001$)
- ✓ Reduced total sleep duration (7.1±0.8 h versus 8.3±0.5 h, $p<0.001$)
- ✓ Increased proportion of light sleep stages (64.7±5.3% versus 52.4±3.8%, $p<0.001$)
- ✓ Diminished slow-wave sleep (18.3±3.6% versus 25.2±2.9%, $p<0.001$)
- ✓ Reduced REM sleep (17.0±2.8% versus 22.4±2.1%, $p<0.001$)
- ✓ Increased nocturnal awakenings (8.6±2.3 versus 3.5±1.4, $p<0.001$)

Temporal correlation analysis revealed that 85% of enuresis episodes occurred predominantly during the first third of the night in deep slow-wave sleep (N3), while 15% occurred within two hours of sleep onset regardless of sleep stage. A significant correlation was established between sleep architecture disruption severity and enuresis frequency ($r=0.68$, $p<0.001$).

Autonomic Function and Circadian Rhythm Assessment

Heart rate variability analysis identified autonomic dysregulation in 87.5% of the study cohort, with sympathetic predominance in 60% and parasympathetic predominance in 27.5%.

Circadian rhythm analysis revealed disrupted melatonin secretion patterns in 80% of cases, characterized by flattened or absent nocturnal peaks (23.4±5.8 pg/ml versus 42.7±6.5 pg/ml, $p<0.001$) and altered day-night ratios (1:2.1±0.3 versus 1:4.3±0.5, $p<0.001$). Body temperature circadian dysrhythmia was evidenced by flattened diurnal curves and reduced day-night differentials (0.6±0.2°C versus 1.1±0.1°C, $p<0.001$).

Urological Assessment

Uroflowmetry identified reduced functional bladder capacity in 65% of the study cohort (275±42 ml versus 360±35 ml, $p<0.001$), with significantly higher prevalence in mixed enuresis (93.3%) than nocturnal enuresis (58.5%), $p<0.01$. Bladder ultrasonography revealed no structural abnormalities in 97.5% of cases, with only 2.5% exhibiting minor dysplastic changes not requiring surgical intervention.

Neuroendocrine Evaluation

Vasopressin analysis revealed reduced nocturnal secretion in 75% of the study cohort (3.2±0.8 pg/ml versus 5.8±0.9 pg/ml, $p<0.001$), predominantly affecting nocturnal enuresis cases (83.1%) compared to mixed enuresis (40%), $p<0.001$.

Correlation Analysis

Significant correlations were established between:

- ✓ EEG theta activity and enuresis frequency ($r=0.72$, $p<0.001$)
- ✓ Occipital alpha power and enuresis duration ($r=-0.64$, $p<0.001$)
- ✓ Nocturnal melatonin levels and enuresis frequency ($r=-0.78$, $p<0.001$)
- ✓ Slow-wave sleep reduction and enuresis severity ($r=0.69$, $p<0.001$)
- ✓ Functional bladder capacity and enuresis frequency ($r=-0.61$, $p<0.001$)
- ✓ Anxiety levels and enuresis frequency ($r=0.58$, $p<0.001$)

Multivariate regression identified the following as significant predictors of enuresis severity:

- ✓ Diminished nocturnal melatonin secretion ($\beta=-0.32$, $p<0.001$)
- ✓ Reduced slow-wave sleep ($\beta=-0.28$, $p<0.001$)
- ✓ Elevated EEG theta activity ($\beta=0.25$, $p<0.001$)
- ✓ Reduced bladder capacity ($\beta=-0.22$, $p<0.01$)
- ✓ Elevated anxiety ($\beta=0.18$, $p<0.01$)

This model explained 82% of enuresis severity variance ($R^2=0.82$, $p<0.001$).

Therapeutic Intervention Outcomes

Based on identified neurophysiological profiles, participants were allocated to three therapeutic protocols:

- ➢ Subgroup A (n=45): Patients with predominant melatonin/vasopressin circadian dysrhythmia received desmopressin (0.2-0.4 mg nightly) combined with melatonin (3 mg pre-sleep)
- ➢ Subgroup B (n=20): Patients with predominant EEG abnormalities received nootropic agents (piracetam 800-1200 mg/day) combined with alarm therapy
- ➢ Subgroup C (n=15): Patients with predominant psychological disturbances received cognitive-behavioral therapy with selective antidepressants when indicated

All participants received lifestyle modification guidance, behavioral therapy, and psychological support.

After six months of intervention, complete remission (3-month enuresis-free period) was achieved in 60% of cases, significant improvement (≥75% reduction) in 26.3%, moderate improvement (50-74% reduction) in 10%, and minimal or no improvement in 3.7%.

Treatment response analysis by subgroup revealed complete remission rates of 73.3% in subgroup A, 50% in subgroup B, and 40% in subgroup C ($p<0.05$), validating the neurophysiologically-differentiated therapeutic approach.

Conclusion

Persistent adolescent enuresis represents a complex multifactorial condition characterized by functional insufficiency of bilateral frontotemporal-parietal cortical regions, midline structure dysfunction, interhemispheric commissure abnormalities, and sleep-wake cycle dysregulation.

This investigation demonstrates that comprehensive neurophysiological profiling enables individualized treatment planning and significantly enhances therapeutic outcomes. The findings support an integrated diagnostic and management approach that addresses the heterogeneous neurophysiological mechanisms underlying this challenging clinical condition.

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