www.ijarp.com ISSN 2456-9992 Page: 01-23

International Journal Advanced Research Publications

Review Article

Volume: 02
Issue: 09

KLÜVER-BUCY SYNDROME AND ITS NEUROREHABILITATION: A NARRATIVE REVIEW WITH A PROPOSED PHYSIOTHERAPY ASSESSMENT AND MANAGEMENT PROTOCOL

[1]Smrutiranjan Sahu, [2]Priyabrata Dash, [3*]Nihar Ranjan Mohanty

[1]Assistant Professor, KIMS School of Physiotherapy, KIMS, KIIT-DU, Bhubaneswar, Odisha.

[2]Professor, KIMS School of Physiotherapy, KIMS, KIIT-DU, Bhubaneswar, Odisha.

[3]Associate Professor, KIMS School of Physiotherapy, KIMS, KIIT-DU, Bhubaneswar, India.

Article Received: 25 July 2026, Article Revised: 15 August 2026, Published on: 05 September 2026

***Corresponding Author: Nihar Ranjan Mohanty**

Associate Professor, KIMS School of Physiotherapy, KIMS, KIIT-DU, Bhubaneswar, India.

Doi: https://doi-doi.org/101555/ijarp.3602

ABSTRACT

Background: Klüver–Bucy syndrome (KBS) is a rare neurobehavioral syndrome associated predominantly with bilateral dysfunction or structural damage of the medial temporal-limbic system. The classical syndrome is characterized by hyperorality, hypersexuality, hyperphagia, hypermetamorphosis, visual agnosia, and altered emotional behavior, particularly placidity and reduced fear. Cognitive abnormalities, especially attention and memory impairment, may also occur. Although KBS is primarily described within neurology, neuropsychiatry, and behavioral medicine, affected individuals may have substantial physical and functional limitations requiring multidisciplinary neurorehabilitation.

Objective: To review the current understanding of the clinical and neuroanatomical characteristics of KBS and to develop an evidence-informed physiotherapy assessment and management framework for individuals with KBS.

Methods: A narrative review was undertaken using published literature on KBS, including historical descriptions, case reports, case series, systematic reviews, and clinical reviews. Because direct physiotherapy research in KBS is extremely limited, evidence from acquired brain injury, traumatic brain injury (TBI), stroke, and neurological rehabilitation was used cautiously to inform recommendations for physical assessment, mobility, balance, gait, strengthening, endurance, behavioral safety, and caregiver education.

Results: KBS is most strongly associated with bilateral medial temporal-lobe dysfunction involving the amygdala, hippocampal formation, and interconnected limbic networks. The etiological spectrum includes central nervous system infections, particularly herpes simplex encephalitis, traumatic brain injury, stroke, epilepsy and temporal-lobe surgery, autoimmune encephalitis, hypoxic injury, neoplasia, and neurodegenerative disorders. A 2026 systematic review of 157 reported cases found hypersexuality and hyperorality in 84% of cases and hyperphagia in 73%; bilateral temporal-lobe lesions were reported in 73% of cases. Physiotherapy-specific evidence remains absent. Nevertheless, acquired-brain-injury rehabilitation guidance supports individualized, goal-oriented, task-specific training, gait and balance rehabilitation, exercise, postural management, and physical activity. In KBS, these interventions require additional behavioral and environmental modifications because hyperorality, hypermetamorphosis, disinhibition, impaired attention, memory dysfunction, and poor safety awareness may interfere with conventional rehabilitation.

Conclusion: Physiotherapy has a potentially important supportive role in the multidisciplinary management of KBS, particularly for mobility, balance, gait, physical conditioning, prevention of secondary complications, and functional independence. However, recommendations should currently be regarded as extrapolated neurorehabilitation principles rather than KBS-specific evidence. Prospective case series, standardized outcome measurement, and collaborative rehabilitation research are required to establish a disease-specific physiotherapy protocol.

KEYWORDS: Klüver-Bucy syndrome; neurorehabilitation; physiotherapy; temporal lobe; amygdala; traumatic brain injury; herpes simplex encephalitis; gait; balance; hyperorality; hypersexuality.

INTRODUCTION

Klüver-Bucy syndrome (KBS) is an uncommon but clinically distinctive neurobehavioral syndrome that develops following dysfunction or bilateral structural injury of the medial temporal-limbic system. The syndrome is traditionally associated with bilateral temporal-lobe lesions, particularly involving the amygdala and hippocampal regions [1-4]. Its classical manifestations include hyperorality, hypersexuality, hyperphagia or altered dietary behavior, hypermetamorphosis, visual agnosia, and placidity or reduced emotional responsiveness [1,3,4].

The syndrome has considerable historical importance in understanding the relationship between the temporal lobes and human behavior. The experimental observations of Klüver and Bucy demonstrated that bilateral temporal-lobe removal could produce profound alterations in emotional, sexual, feeding, and exploratory behavior [1]. Human cases subsequently demonstrated that comparable behavioral abnormalities could arise from bilateral temporal-lobe pathology [2].

KBS is not a single-disease entity. Rather, it represents a clinical syndrome that can result from several pathological processes affecting interconnected temporal-limbic structures. Herpes simplex encephalitis (HSE), traumatic brain injury, stroke, epilepsy, autoimmune encephalitis, hypoxic injury, tumors, and neurodegenerative disorders have all been reported as causes [3-8].

The rarity of KBS means that most published evidence consists of case reports and case series. Consequently, the syndrome has historically been defined by variable diagnostic criteria and inconsistent reporting. A recent systematic review of 141 articles involving 157 cases demonstrated substantial heterogeneity but confirmed hypersexuality, hyperorality, and hyperphagia as the most frequently reported manifestations [9]. Bilateral temporal-lobe lesions were demonstrated on neuroimaging in 73% of cases, while central nervous system infections, traumatic brain injury, and autoimmune disorders represented important etiological groups [9].

From a rehabilitation perspective, KBS presents a unique challenge. Behavioral symptoms may coexist with hemiparesis, ataxia, balance impairment, deconditioning, impaired endurance, altered motor control, visual-perceptual deficits, and cognitive dysfunction arising from the underlying neurological disease. The physiotherapist therefore needs to address not only physical impairments but also the behavioral and cognitive factors that determine safe participation.

Importantly, there is currently no established KBS-specific physiotherapy treatment protocol. The rehabilitation approach proposed in this review is therefore based on principles established for acquired brain injury, TBI, stroke, and neurological rehabilitation and adapted to the behavioral phenotype of KBS [10-18].

HISTORICAL BACKGROUND

The behavioral consequences of bilateral temporal-lobe damage were described experimentally before the syndrome was formally named. Klüver and Bucy subsequently

provided the classic description of the behavioral syndrome following bilateral temporal-lobe ablation in rhesus monkeys in 1939 [1].

The first well-documented human reproduction of the syndrome was subsequently described by Terzian and Dalle Ore in 1955 in a patient who had undergone bilateral temporal-lobe removal for epilepsy [2]. Later, Marlowe and colleagues described complete KBS following viral meningoencephalitis associated with bilateral temporal-lobe injury [3].

These observations established an important relationship between the temporal-limbic system and behavioral regulation. Contemporary neuroimaging and neuropsychiatric studies have subsequently expanded this model, suggesting that KBS may arise from disruption of interconnected mesial temporal and limbic networks rather than from destruction of the amygdala alone [6,19].

NEUROANATOMICAL BASIS

The medial temporal region contains several structures involved in memory, emotional processing, motivation, learning, social behavior, and behavioral regulation. The amygdala is particularly important for processing emotionally salient stimuli, fear and threat responses, motivation, and social-emotional behavior, whereas the hippocampal formation has a central role in learning and memory [4,6].

The classical KBS phenotype has therefore been interpreted as the consequence of disruption of a network involving:

- Amygdala
- Hippocampus
- Parahippocampal cortex
- Uncus
- Medial temporal cortex
- Inferotemporal cortex
- Limbic connections
- Associated frontal and subcortical networks

The relationship between anatomical damage and clinical phenotype is not absolute. Some patients with partial KBS may have relatively restricted temporal lesions, while others may have more extensive abnormalities involving frontotemporal and limbic networks [6,19].

Caro and Jimenez proposed that mesiotemporal disconnection and reduced activity within interconnected neural systems may contribute to KBS rather than a simple model of isolated

amygdala destruction [19]. This network perspective is important for rehabilitation because physical function, attention, emotional regulation, visuospatial processing, and motor behavior may all be influenced by the wider neural injury.

ETIOLOGY

Table 1. Major reported causes of Klüver-Bucy syndrome.

Etiological category	Examples	Rehabilitation relevance
Central nervous system infection	Herpes simplex encephalitis, other encephalitides	Weakness, fatigue, cognitive dysfunction, seizures and deconditioning may coexist
Traumatic brain injury	Moderate/severe TBI, bilateral temporal injury	Balance, gait, executive dysfunction, fatigue and behavioral problems
Cerebrovascular disease	Temporal-lobe infarction or hemorrhage	Hemiparesis, balance, gait and visual-perceptual deficits
Epilepsy/surgery	Bilateral temporal epilepsy or temporal lobectomy	Seizure precautions, memory deficits and behavioral changes
Autoimmune encephalitis	Limbic encephalitis and related disorders	Cognitive, motor and autonomic complications
Hypoxic brain injury	Global cerebral hypoxia	Severe motor and cognitive impairment
Neoplasia	Temporal-lobe tumors	Focal neurological deficits and postoperative impairments
Neurodegenerative disease	Selected dementing disorders	Progressive cognitive and functional decline
Other structural lesions	Subdural collections and other bilateral temporal lesions	Variable physical and cognitive disability

HSE is particularly important because of its predilection for the temporal lobes. Several reports have documented KBS after HSE [5,20]. Traumatic brain injury is another important cause; a systematic review identified 24 reported cases of KBS following TBI and found sexual hyperactivity, altered dietary behavior, and hyperorality among the most frequent symptoms [7].

A 2026 systematic review identified central nervous system infections as the largest etiological category, followed by traumatic brain injury and autoimmune conditions [9].

CLINICAL MANIFESTATIONS

KBS is characterized by a variable combination of behavioral, emotional, cognitive, perceptual, and feeding abnormalities.

Table 2. Major clinical manifestations and their functional implications.

Manifestation	Clinical description	Potential rehabilitation consequence
Hyperorality	Oral exploration or inappropriate placement of objects in mouth	Choking, ingestion of equipment, unsafe treatment
Hypersexuality	Inappropriate or excessive sexual behavior	Boundary and safeguarding issues
Hyperphagia	Increased eating or abnormal food preference	Dietary supervision and behavioral management
Hypermetamorphosis	Excessive response to visual stimuli	Distractibility and unsafe environmental exploration
Visual agnosia	Difficulty recognizing objects despite preserved vision	Difficulty identifying equipment and obstacles
Placidity	Reduced fear, anxiety or aggression	Reduced protective responses and safety awareness
Memory impairment	Deficits in learning and recall	Poor retention of instructions and exercise routines
Attention impairment	Reduced sustained or selective attention	Difficulty participating in treatment
Emotional dysregulation	Altered emotional response	Inconsistent engagement
Behavioral disinhibition	Reduced social inhibition	Difficulty maintaining therapeutic boundaries

The six classical behavioral components are hyperorality, hypersexuality, hyperphagia, placidity, visual agnosia, and hypermetamorphosis [3,4]. Contemporary reports additionally emphasize memory, attention, executive and social-cognitive abnormalities [6,9].

The syndrome may be complete or partial. Historically, patients presenting with at least three characteristic features have often been categorized as having partial KBS [4]. The recent systematic review emphasizes that the traditional criteria remain incompletely validated and proposes a more structured diagnostic framework [9].

FUNCTIONAL CONSEQUENCES RELEVANT TO PHYSIOTHERAPY

Although KBS itself is predominantly a behavioral syndrome, the underlying neurological lesion may cause significant physical disability.

The patient's functional profile may include:

1. Reduced muscle strength.
2. Abnormal muscle tone.

3. Impaired postural control.
4. Ataxia.
5. Hemiparesis.
6. Reduced gait speed.
7. Impaired dynamic balance.
8. Reduced endurance.
9. Deconditioning.
10. Poor motor learning.
11. Visual-perceptual problems.
12. Reduced attention.
13. Memory impairment.
14. Poor safety awareness.
15. Impulsive or compulsive movement.
16. Falls.
17. Reduced participation in activities of daily living.

In TBI, balance impairment is a common rehabilitation problem, and systematic reviews indicate that physical therapy interventions may improve balance, although the evidence base is heterogeneous [21]. Similarly, gait and balance rehabilitation has been examined in ambulatory TBI populations, although the evidence remains less robust than that available for stroke [22].

Therefore, the physiotherapist should avoid assuming that a patient with KBS is physically normal simply because the presenting syndrome is behavioral.

PHYSIOTHERAPY ASSESSMENT

There is no validated physiotherapy assessment battery specifically designed for KBS. Assessment should therefore combine neurological, musculoskeletal, functional, behavioral, and environmental evaluation.

1. Subjective assessment

Where possible, information should be obtained from the patient and caregiver.

Important domains include:

- Etiology of neurological injury
- Date of onset
- Previous level of function

- Current mobility
- Falls
- Seizure history
- Medication
- Fatigue
- Pain
- Sleep
- Behavioral triggers
- Feeding behavior
- Cognitive changes
- Communication difficulties
- Social behavior
- Caregiver concerns
- Home environment

2. Physical assessment

The following should be examined:

Motor function

- Muscle strength
- Muscle tone
- Selective motor control
- Coordination
- Motor planning
- Range of motion

Posture and balance

- Sitting balance
- Standing balance
- Static postural control
- Dynamic postural control
- Reactive balance
- Weight transfer

Mobility

- Bed mobility
- Transfers

- Sit-to-stand
- Standing tolerance
- Gait
- Stair negotiation
- Community mobility

Cardiopulmonary status

- Heart rate
- Blood pressure
- Oxygen saturation where indicated
- Exercise tolerance
- Respiratory status
- Fatigue

RECOMMENDED FUNCTIONAL OUTCOME MEASURES

Table 3. Potential physiotherapy outcome measures.

Domain	Outcome measure
Balance	Berg Balance Scale
Functional mobility	Timed Up and Go
Gait speed	10-Metre Walk Test
Walking endurance	6-Minute Walk Test
Lower-limb function	Five Times Sit-to-Stand
Ambulation	Functional Ambulation Category
Functional independence	Functional Independence Measure or equivalent
Falls	Falls diary/frequency
Participation	Goal Attainment Scaling
Stroke-related disability	Modified Rankin Scale, where appropriate
Exercise capacity	6-Minute Walk Test or individualized exercise testing

These measures should not be applied indiscriminately. Severe cognitive or behavioral impairment may invalidate some tests. The therapist should select measures that can be performed safely and reliably.

BEHAVIORAL ASSESSMENT BEFORE PHYSIOTHERAPY

Behavioral assessment is essential because conventional physiotherapy may become unsafe in patients with severe KBS.

The therapist should identify:

- Triggers for hyperorality
- Environmental stimuli producing excessive exploration
- Episodes of sexual disinhibition

- Aggressive or non-aggressive behavioral responses
- Attention span
- Ability to follow one-step commands
- Ability to maintain task engagement
- Safety awareness
- Risk of wandering
- Risk of ingesting objects
- Appropriate caregiver presence

The rehabilitation environment should be modified accordingly.

PHYSIOTHERAPY MANAGEMENT

1. Fundamental principles

The proposed physiotherapy model is based on six principles:

Safety → Simplicity → Repetition → Function → Progression → Participation

Because KBS-specific trials are lacking, these principles are extrapolated from acquired-brain-injury rehabilitation and should be individualized [10-18].

The 2025 NICE guideline for chronic neurological disorders and acquired brain injury recommends individualized exercise programs that consider executive function, fatigue, pain, risks, supervision needs, and clinically relevant exercise dose [10]. For stability, mobility, and limb function, NICE recommends targeted interventions such as task-based training, gait training, balance exercises, sensorimotor training, and postural management when clinically indicated [11].

PROPOSED PHYSIOTHERAPY ASSESSMENT AND MANAGEMENT PROTOCOL

Table 4. KBS-specific physiotherapy assessment and management protocol.

Stage	Assessment	Intervention	Primary goal
1. Safety	Behavior, cognition, fall risk, hyperorality	Environmental modification, supervision	Prevent injury
2. Baseline function	Strength, ROM, tone, balance, gait	Appropriate standardized measures	Establish baseline
3. Bed mobility	Rolling, bridging, sitting	Repetitive functional practice	Independence
4. Transfers	Sit-to-stand, bed-chair	Task-specific training	Safe transfers
5. Balance	Static/dynamic balance	Weight shifting, reaching, stepping	Fall reduction
6. Gait	Speed, endurance, gait pattern	Overground/treadmill gait training	Mobility
7. Conditioning	HR, fatigue, endurance	Aerobic and strengthening exercise	Physical capacity
8. Cognitive-motor integration	Attention, memory, following commands	Simple cues, repetition, external cues	Motor learning
9. Community mobility	Obstacles, turning, stairs	Functional/community training	Participation
10. Home program	Caregiver ability and environment	Individualized home exercise	Long-term maintenance

ENVIRONMENTAL SAFETY MODIFICATION

Environmental management is arguably the first physiotherapy intervention in severe KBS.

The treatment area should be:

- Predictable
- Uncluttered
- Quiet when possible
- Free from unnecessary small objects
- Organized consistently
- Adequately supervised

Because hyperorality may lead to oral exploration, small detachable objects, elastic components, loose weights, straps, and other potentially ingestible materials should not be left unattended.

Hypermetamorphosis may cause the patient to approach or touch environmental objects indiscriminately. Therefore, unnecessary visual stimuli should be reduced.

Placidity may paradoxically increase risk because reduced fear can diminish normal avoidance of dangerous situations.

POSITIONING AND PREVENTION OF SECONDARY COMPLICATIONS

Patients with severe neurological injury may experience prolonged immobility. Physiotherapy should therefore include:

- Regular repositioning
- Joint range maintenance
- Contracture prevention
- Pressure injury prevention
- Seating assessment
- Upright tolerance
- Respiratory positioning
- Progressive mobilization

The 2025 NICE guideline recommends consideration of 24-hour postural management in patients with limited independent sitting, standing, or movement who are at risk of skin breakdown, pain, respiratory dysfunction, or contracture [11].

BED MOBILITY TRAINING

Bed mobility is an appropriate early target.

Interventions include:

Rolling

Practice

- Head and trunk rotation
- Lower-limb positioning
- Reaching
- Segmental rolling

Bridging

Useful for:

- Pelvic control
- Hip extensor activation
- Bed mobility
- Preparation for transfers

Supine-to-sitting

Training should use consistent verbal cues and physical guidance.

For example:

"Turn → legs over → push → sit."

Short commands are preferable to long explanations in patients with attention or memory deficits.

TRANSFER TRAINING

Transfer training should emphasize:

- Sit-to-stand
- Stand-to-sit
- Bed-to-chair
- Chair-to-bed
- Toilet transfer
- Car transfer when appropriate

The therapist should use consistent movement sequences. Repetition can support procedural learning even when explicit memory is impaired.

The 2025 ESO motor rehabilitation guideline emphasizes repetitive practice and task-specific rehabilitation in stroke populations, while the NICE guideline similarly supports targeted functional training [11,18]. These recommendations are not specific to KBS but are relevant when KBS occurs secondary to stroke or another acquired brain injury.

BALANCE REHABILITATION

Balance assessment should identify whether the primary impairment is related to:

- Weakness
- Hemiparesis
- Ataxia
- Sensory loss
- Vestibular dysfunction
- Visual-perceptual dysfunction
- Truncal control
- Attention
- Fear or, conversely, reduced protective behavior

Interventions may include:

1. Static sitting balance.
2. Dynamic sitting balance.
3. Supported standing.
4. Weight shifting.
5. Reaching.
6. Sit-to-stand.
7. Stepping.
8. Dynamic balance.
9. Perturbation training where appropriate.
10. Functional obstacle negotiation.

NICE recommends targeted balance interventions, including core stability and perturbation-based training, when appropriate [11]. Stroke guidelines similarly support balance assessment and training for individuals at risk of falls [12].

GAIT REHABILITATION

Gait rehabilitation should be individualized according to the patient's motor and behavioral status.

Potential interventions

- Parallel-bar walking
- Overground walking
- Treadmill training
- Body-weight-supported treadmill training
- Step training
- Turning practice
- Obstacle negotiation
- Stair training
- Assistive-device training
- Community walking

For individuals with significant gait impairment following stroke, intensive and repetitive mobility-task training is recommended [12]. The 2025 ESO guideline also addresses gait-therapy dose and repetitive walking practice [18].

However, KBS presents a special challenge: a patient may suddenly deviate from the walking path because of hypermetamorphosis or environmental exploration. Consequently, gait training should initially occur in a highly controlled environment.

STRENGTHENING

Weakness may result from the underlying neurological lesion, immobilization, systemic illness, or deconditioning.

Strengthening may include:

- Active range of motion
- Active-assisted exercises
- Functional strengthening
- Sit-to-stand repetitions
- Step-ups
- Resistance exercises
- Closed-chain activities

- Progressive resistance training

Exercise should be prescribed according to neurological status, cardiovascular tolerance, fatigue, pain, and behavioral safety.

The NICE guideline recommends individualized exercise programs designed to optimize muscle strength, exercise capacity, and physical function while considering executive function, fatigue, pain, risks, supervision, and exercise dose [10].

AEROBIC CONDITIONING

Long periods of hospitalization and neurological disability can result in severe deconditioning.

Potential aerobic activities include:

- Walking
- Treadmill exercise
- Stationary cycling
- Upper or lower limb ergometry
- Circuit-based activity

The exercise prescription should follow:

Frequency → Intensity → Time → Type → Progression

Heart rate, perceived exertion, blood pressure, oxygen saturation, fatigue, and neurological symptoms should be monitored when clinically appropriate.

Because executive dysfunction and poor safety awareness may impair independent exercise, supervised activity is preferable in moderate to severe KBS.

COGNITIVE-MOTOR REHABILITATION

Cognitive dysfunction is highly relevant to physical rehabilitation. Attention and memory problems are frequently reported in KBS [9].

INCOG 2.0 guidelines for TBI emphasize structured assessment and individualized cognitive rehabilitation, including management of attention, memory, executive functions, and cognitive-communication disorders [13-17].

For physiotherapy, practical adaptations include:

- One instruction at a time.
- Demonstration before performance.
- Consistent terminology.
- Repeated practice.
- External cues.
- Visual or tactile cues where appropriate.
- Reduced background distractions.
- Predictable treatment sequence.
- Immediate feedback.
- Functional task practice.

For example, rather than saying:

“Please stand up, walk across the room, turn around and sit on the chair.”

the therapist may use:

“Stand.”

“Walk.”

“Turn.”

“Sit.”

This approach reduces cognitive load.

HYPERORALITY AND PHYSIOTHERAPY SAFETY

Hyperorality is one of the most important safety issues.

The patient may attempt to:

- Bite exercise equipment
- Put bands in the mouth
- Chew foam
- Bite furniture
- Place fingers in the mouth
- Pick up environmental objects

A published case following HSE described severe oral exploration and biting behavior [20].

Therefore:

- Avoid small objects.
- Use robust equipment.

- Maintain direct supervision.
- Do not place loose equipment within reach.
- Inspect the treatment environment.
- Educate caregivers regarding choking and ingestion risks.

The physiotherapist should also coordinate with occupational therapy, nursing, speech-language therapy, and medical staff if swallowing or ingestion concerns arise.

HYPERSEXUALITY AND PROFESSIONAL BOUNDARIES

Hypersexuality is a classical KBS feature and was present in 84% of cases in the recent systematic review [9].

This may create difficult situations during physiotherapy because treatment frequently involves:

- Physical contact
- Close proximity
- Transfers
- Manual facilitation
- Assistance with dressing or positioning

A professional and structured approach should include:

1. Clearly established therapist-patient boundaries.
2. Appropriate draping.
3. Chaperone support where required.
4. Avoidance of unnecessary physical contact.
5. Documentation of significant incidents.
6. Consistent behavioral responses among staff.
7. Multidisciplinary behavioral management.
8. Staff education.

The behavior should be understood within the neurological syndrome while still maintaining appropriate safeguarding procedures.

HYPERMETAMORPHOSIS AND VISUAL-PERCEPTUAL PROBLEMS

Hypermetamorphosis refers to excessive attention and behavioral response to visual stimuli [4]. During physiotherapy, this may manifest as:

- Touching every object

- Walking toward environmental stimuli
- Repeatedly picking up equipment
- Difficulty maintaining task focus

Treatment should therefore begin in a minimally distracting environment.

As functional control improves, controlled environmental challenges can gradually be introduced.

If visual agnosia is present, the patient may have difficulty recognizing exercise equipment or obstacles despite preserved visual acuity. Occupational therapy and neuropsychology assessment are therefore important.

MEMORY AND ATTENTION MANAGEMENT

Memory and attention impairment may prevent the patient from retaining exercise instructions.

The physiotherapist should:

- Use consistent routines.
- Repeat the same task.
- Use external cues.
- Avoid unnecessary verbal information.
- Provide immediate feedback.
- Use meaningful functional goals.
- Train caregivers to use identical instructions.

INCOG 2.0 emphasizes individualized cognitive rehabilitation and the use of evidence-informed strategies for attention, memory and executive dysfunction after TBI [13-17]. These principles can reasonably be adapted to KBS when cognitive dysfunction is secondary to acquired brain injury.

CAREGIVER EDUCATION

Caregivers play a major role in long-term rehabilitation.

Education should cover:

Mobility

- Safe transfers
- Walking assistance
- Use of walking aids

- Fall prevention

Behavioral safety

- Hyperorality
- Hypersexuality
- Wandering
- Environmental exploration

Exercise

- Home exercise
- Walking program
- Strengthening
- Balance activities

Environment

- Removal of dangerous objects
- Bathroom safety
- Stair safety
- Bedroom arrangement
- Community supervision

The rehabilitation plan should be coordinated with caregivers and regularly reviewed. NICE recommends personalized rehabilitation planning involving the individual's goals and coordinated multidisciplinary care [23].

MULTIDISCIPLINARY REHABILITATION

KBS cannot be effectively managed by physiotherapy alone.

Table 5. Multidisciplinary rehabilitation team.

Professional	Primary contribution
Neurologist	Diagnosis, neurological management, seizure management
Physiatrist	Overall rehabilitation coordination
Physiotherapist	Mobility, gait, balance, strength, endurance
Occupational therapist	ADL, environmental modification, cognitive-functional training
Neuropsychologist	Cognitive and behavioral assessment
Psychiatrist	Behavioral and psychiatric management
Speech-language therapist	Communication and swallowing/cognitive-communication
Dietitian	Hyperphagia and nutritional management
Nursing team	Supervision and behavioral safety
Social worker	Family and community support
Caregiver	Home supervision and rehabilitation continuity

The need for coordinated multidisciplinary rehabilitation is consistent with modern neurological rehabilitation practice [10,12,23].

CONCLUSION

Klüver-Bucy syndrome is a rare and complex neurobehavioral syndrome associated predominantly with bilateral medial temporal-limbic dysfunction. Its classical manifestations include hyperorality, hypersexuality, hyperphagia, hypermetamorphosis, visual agnosia and placidity, while cognitive abnormalities involving attention and memory are also common. The syndrome may occur after herpes simplex encephalitis, traumatic brain injury, stroke, epilepsy, autoimmune encephalitis, hypoxic injury, tumors, and neurodegenerative conditions.

The recent systematic review of 157 cases provides the strongest contemporary description of the clinical phenotype and confirms considerable heterogeneity in both presentation and etiology [9]. This heterogeneity is particularly important for rehabilitation because physical disability is determined not only by KBS itself but also by the underlying neurological injury.

At present, there is insufficient direct evidence to establish a KBS-specific physiotherapy protocol. Nevertheless, physiotherapy can play an important role in multidisciplinary rehabilitation by addressing mobility, balance, gait, strength, endurance, postural control, prevention of secondary complications, and community participation.

The physiotherapist should incorporate behavioral and cognitive adaptations into every stage of treatment. Hyperorality requires environmental safety precautions; hypermetamorphosis requires control of visual distractions; hypersexuality requires professional boundaries and appropriate safeguarding; and memory or attention deficits require simplified instructions, repetition, and structured routines.

Evidence from acquired brain injury, TBI, and stroke rehabilitation supports task-specific training, gait rehabilitation, balance training, physical activity, strengthening, and individualized exercise programs [10-18,21,22]. These interventions should be interpreted as evidence-informed extrapolations rather than interventions proven specifically for KBS.

A multidisciplinary, individualized, goal-oriented rehabilitation model is therefore recommended. Future research should systematically document physiotherapy interventions and functional outcomes in KBS to establish an evidence base capable of supporting disease-specific rehabilitation guidelines.

REFERENCES

1. Klüver H, Bucy PC. Preliminary analysis of functions of the temporal lobes in monkeys. *Arch Neurol Psychiatry*. 1939;42:979-1000.
2. Terzian H, Dalle Ore G. Syndrome of Klüver and Bucy; reproduced in man by bilateral removal of the temporal lobes. *Neurology*. 1955;5(6):373-380. doi:10.1212/WNL.5.6.373.
3. Marlowe WB, Mancall EL, Thomas JJ. Complete Klüver-Bucy syndrome in man. *Cortex*. 1975;11(1):53-59. doi:10.1016/S0010-9452(75)80020-7.
4. Das JM, Siddiqui W. Klüver-Bucy Syndrome. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2026.
5. Poduval RG, Mukherji JD, Kumaravelu S. Klüver-Bucy syndrome following herpes simplex encephalitis. *Med J Armed Forces India*. 2005;61(4):389-390. doi:10.1016/S0377-1237(05)80078-4.
6. Caro MA, Jimenez XF. Mesiotemporal disconnection and hypoactivity in Klüver-Bucy syndrome: case series and literature review. *J Clin Psychiatry*. 2016;77(8):e982-e988. doi:10.4088/JCP.14r09497.
7. Clay FJ, Kuriakose A, Lesche D, Hicks AJ, Zaman H, Azizi E, et al. Klüver-Bucy syndrome following traumatic brain injury: a systematic synthesis and review of pharmacological treatment from cases in adolescents and adults. *J Neuropsychiatry Clin Neurosci*. 2019;31(1):6-16. doi:10.1176/appi.neuropsych.18050112.
8. Al-Attas AA, Aldayel AY, Aloufi TH, et al. Klüver-Bucy syndrome secondary to a nondominant middle cerebral artery ischemic stroke: a case report and review of the literature. *J Med Case Rep*. 2021;15:346.
9. Muñoz JF, Ramírez-Bermúdez J, Peña-Casanova J, Restrepo D, Gómez-Henk C, Muñoz JR, et al. The neuropsychiatric phenotype of Klüver-Bucy syndrome: a systematic review of reported cases. *J Neuropsychiatry Clin Neurosci*. 2026. doi:10.1176/appi.neuropsych.20250144.
10. National Institute for Health and Care Excellence. *Rehabilitation for chronic neurological disorders including acquired brain injury*. NICE guideline NG252. London: NICE; 2025.
11. National Institute for Health and Care Excellence. Rehabilitation to maintain, improve or support function. In: *Rehabilitation for chronic neurological disorders including acquired brain injury*. NICE guideline NG252. London: NICE; 2025.

12. Winstein CJ, Stein J, Arena R, Bates B, Cherney LR, Cramer SC, et al. Guidelines for adult stroke rehabilitation and recovery: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2016;47(6):e98-e169. doi:10.1161/STR.0000000000000098.
13. Bayley MT, Janzen S, Harnett A, Bragge P, Togher L, Kua A, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury: methods, overview, and principles. *J Head Trauma Rehabil*. 2023;38(1):7-23. doi:10.1097/HTR.0000000000000838.
14. Ponsford J, Velikonja D, Janzen S, Harnett A, McIntyre A, Wiseman-Hakes C, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, Part II: attention and information processing speed. *J Head Trauma Rehabil*. 2023;38(1):38-51. doi:10.1097/HTR.0000000000000839.
15. Jeffay E, Ponsford J, Harnett A, Janzen S, Patsakos E, Douglas J, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, Part III: executive functions. *J Head Trauma Rehabil*. 2023;38(1):52-64. doi:10.1097/HTR.0000000000000834.
16. Togher L, Douglas J, Turkstra LS, Welch-West P, Janzen S, Harnett A, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, Part IV: cognitive-communication and social cognition disorders. *J Head Trauma Rehabil*. 2023;38(1):65-82. doi:10.1097/HTR.0000000000000835.
17. Velikonja D, Ponsford J, Janzen S, Harnett A, Patsakos E, Kennedy M, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, Part V: memory. *J Head Trauma Rehabil*. 2023;38(1):83-102. doi:10.1097/HTR.0000000000000837.
18. Alt Murphy M, Munoz-Novoa M, Heremans C, Branscheidt M, Cabanas-Valdés R, Engelter ST, et al. European Stroke Organisation guideline on motor rehabilitation. *Eur Stroke J*. 2025;10(4):1160-1188. doi:10.1177/23969873251338142.
19. Caro MA, Jimenez XF. Mesiotemporal disconnection and hypoactivity in Klüver-Bucy syndrome: case series and literature review. *J Clin Psychiatry*. 2016;77(8):e982-e988. doi:10.4088/JCP.14r09497.
20. Begum H, Nayek K, Khuntdar BK. Kluver-Bucy syndrome—a rare complication of herpes simplex encephalitis. *J Indian Med Assoc*. 2006;104(11):637-638.
21. Alashram AR, Annino G, Reddy RS, et al. Effects of physical therapy interventions on balance ability in people with traumatic brain injury: a systematic review. *NeuroRehabilitation*. 2020.

22. Bland DC, Zampieri C, Damiano DL. Effectiveness of physical therapy for improving gait and balance in ambulatory individuals with traumatic brain injury: a systematic review of the literature. *Brain Inj*. 2011;25(7-8):664-679. doi:10.3109/02699052.2011.576306.
23. National Institute for Health and Care Excellence. Rehabilitation planning and delivery. In: *Rehabilitation for chronic neurological disorders including acquired brain injury*. NICE guideline NG252. London: NICE; 2025.
24. Duggal HS, Khess CRJ, Nizamie SH. Subdural hygroma presenting as dementia with Klüver-Bucy symptoms. *Indian J Psychiatry*. 1999;41(4):371-373.
25. Muñoz JF, Ramírez-Bermúdez J, Peña-Casanova J, et al. The neuropsychiatric phenotype of Klüver-Bucy syndrome: a systematic review of reported cases. *J Neuropsychiatry Clin Neurosci*. 2026. doi:10.1176/appi.neuropsych.20250144.
26. Terzian H, Dalle Ore G. Syndrome of Klüver and Bucy; reproduced in man by bilateral removal of the temporal lobes. *Neurology*. 1955;5:373-380.
27. Clay FJ, Kuriakose A, Lesche D, et al. Klüver-Bucy syndrome following traumatic brain injury: systematic synthesis and pharmacological treatment review. *J Neuropsychiatry Clin Neurosci*. 2019;31:6-16.
28. National Institute for Health and Care Excellence. Rehabilitation for chronic neurological disorders including acquired brain injury: evidence reviews. London: NICE; 2025.